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patients. *Practitioner* 184: 70-79,

ey, H. M. American Drinking
Studies, New Brunswick, N.J.

ana and other drug use by grad-
er. *J. Psychiat.* 129: 415-420.

s' Use of Drugs and Alcohol.
gh, Scotland, Sept. 18, 1971.
ck Physician: Impairment by
holism and Drug Dependence.

ilities are beautifully illustrated
R. D. Attitudes in Medicine
ally at the Region VI meeting
Alcoholism, April 19, 1974, at
Center, 42nd and Dewey Ave.,

. Physician care of alcoholics:
. *C. Med. J.* 33: 859-861, 1972.
Problems in *Proceedings of the*
hol and Alcoholism. Hillhouse

e, B. A. and Cochrane, C. M.
for Prefreshman Medical Stu-
163, 1971.

The Association of Alcoholism Mortality with Legal Availability of Alcoholic Beverages

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FOREWORD: This article is a revised version of a paper presented at the 31st International Congress on Alcoholism and Drug Dependence which was held in Bangkok, Thailand on February 23-28, 1975 and is published with the permission of the Director of the International Council on Alcohol and Addictions.

The subject of legal controls for the use and availability of alcoholic beverages has frequently been of interest to legislators, crusaders, the liquor industry, and social drinkers, and there are many opinions and very few facts to back up any one point of view. The study of legal controls for alcoholic beverages is important because if in fact laws are associated with alcoholism, they can be much more easily regulated by society than can other factors such as unhealthy family styles or urbanization. Most frequently, however, legal controls are put into practice with very little (if any) data to show potential effectiveness and no provision to study the new measures before and after their institution. Canadian studies (Popham *et al.*, 1971) have shown that various controls such as government-controlled monopoly systems versus private enterprise, diversification of licensed drinking places (i.e. introduction of cocktail and dining lounges), hours of sale, and the number of outlets do not explain the variation in reported liver cirrhosis mortality in different geographical areas.

There have been some studies, though, where changes in availability have measurably influenced drinking behavior. A Finnish work (Kuusi, 1957) set out to study the change in drinking behavior in rural communities after the opening of state beer and wine package stores. The results were inconclusive because while beer and wine consumption increased, illicit liquor consumption decreased. The amount of absolute alcohol consumed probably did not change as much as its form.

Whitehead (1974) cites evidence to support the stance that removal or liberalization of alcohol control measures tends to increase per capita consumption. This was true in Finland when in 1969 medium strength beer (which had previously only been available in the State Monopoly System) was released for sale on the unrestricted retail market. The other legal control investigated by Whitehead was the lowering of drinking ages in several areas in North America. Both consumption and alcohol-related

traffic collisions increased for the group that were added to the legal drinking age group.

Most changes in legal availability controls have been in the direction of increasing the availability of alcohol. There are, however, a few documented instances of decreased availability, but this is a rarity. The American experience of prohibition is probably the best example of this. Cirrhosis rates dropped (Terris, 1967), and people drank more secretly and preferred distilled spirits because the alcohol was more concentrated (Room, 1971). The strike in the stores of the Finnish Alcohol Monopoly is also an example of an abrupt decrease in availability of alcoholic beverages (Makela, 1974). During this five week strike, there was a marked decrease in arrests for public drunkenness, driving under the influence arrests, crimes of violence, alcohol-related accidents, and hangover patients in outpatient clinics. Additionally in interviews with a representative sample of the population, over three-quarters regarded the strike as a matter of indifference to themselves and their families. Due to the fact that the duration of the strike was believed to be short, smuggling never really got organized, and it is impossible to know the extent to which this would have entered the picture given time.

The present study is an investigation of the association of the legal availability of alcoholic beverages with alcoholism death rates in the state of North Carolina. In North Carolina there is a very unusual system for the distribution of alcoholic beverages. For one thing, there is no "liquor-by-the-drink" in the state, which means that it is illegal to sell cocktails other than beer or wine. Distilled spirits may only be bought in state controlled A.B.C. (Alcoholic Beverage Control) stores, and a pint is the smallest amount available. Beer and wine may be bought in grocery stores and in restaurants which have special permits. The decision as to whether or not beer, wine, or distilled spirits will be legally available in a county is determined by local referendums. Each of these three types of alcoholic beverages is voted upon separately in the referendums, so there could be a number of combinations of types of beverages available in the various counties. In fact, there are five different availability classifications which will be described in the next section. This type of alcohol availability system provides an excellent natural setting with a relatively homogeneous population for a first analysis of the effect of legal control of alcoholic beverages on alcohol-related problems.

It would be unfair, though to study this problem without considering other environmental factors which are known to be associated with high rates of alcohol problems. Urbanization has frequently been found to be correlated with alcoholism and is also known to influence mortality rates in general. In their study *American Drinking Practices*, Cahalan *et al.* (1969) found that people with high rates of alcohol-related problems were more likely to be residents of cities and people who had moved from rural

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areas or from small towns to large cities. Sundby (1967) has also shown
a remarkable urban/rural difference in Norway and estimates that from
25.0% to 38.3% of Oslo's excess male mortality may be accounted for by
the higher prevalence of alcoholism in Oslo.

Popham *et al.* (1971) found that the ratio of average cost of a gallon of
alcohol to average disposable income is closely associated with alcohol
consumption rates and liver cirrhosis mortality. The smaller the ratio is,
the higher alcohol consumption and liver cirrhosis mortality are. We must
consider the possibility then that the more affluent counties have more
disposable income and might also tend to be the wet counties. Figures on
the average disposable income for each North Carolina county are not
available, so average per capita income can be used as a somewhat crude
substitute to check for the effect of economy on alcohol death rate. The
cost of distilled spirits is state controlled so it is the same in all counties.
While prices of beer and wine vary slightly from store to store, the overall
variation from county to county is negligible.

If we look at social stress theory, we would predict that in areas where
there is rapid social change, there will be more stress and a higher risk
of pathology (Dohrenwend, 1961). With this in mind, Smith (1973) has
constructed an index of change for North Carolina counties which is
"suggestive of the amount of change occurring in a social setting." No
distinction was made between negative change and positive change; only
the absolute amount of change was considered. The index is rather crude
in that respect; nevertheless, it is the best measure of social change avail-
able on a county basis. The variables included in this index are:

1. Percent change in population, 1960-1970
2. Change in percent nonwhite, 1960-1970
3. Change in rate of unemployment, 1965-1970
4. Change in per capita income, 1962-1970
5. Change in percent high school graduates joining the work force,
1965-1970
6. Change in percent females ever married, now separated, widowed
or divorced, 1960-1970

Lastly, the population of each county was considered as an independent
variable on the theory that in less populous counties there might be more of
a tendency to report alcohol deaths inaccurately. There is less anonymity
in small counties, and there would be more of a tendency to protect fam-
ilies from the stigma of an alcoholic death.

METHOD: All 1971 North Carolina death certificates were examined,
and those with primary cause of death due to alcoholic cirrhosis, chronic
alcoholism, and acute alcoholism (ICDA classifications 571.0, 303.2, and
303.9) were counted as alcoholic deaths (N = 436). Age, race, and
county of residence were also noted.

All 100 counties in North Carolina were classified according to legal

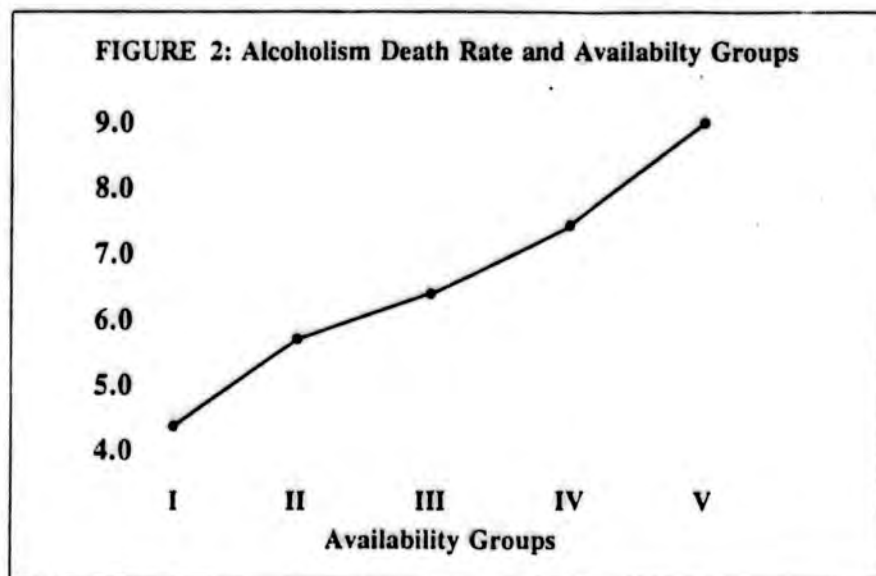
availability of alcoholic beverages in any part of that county for the year 1971. The five county groupings which resulted from the legal availability classification are:

- I. No beer, wine, or distilled spirits legally available (15 counties)
- II. Only beer and wine available (2 counties)
- III. Only distilled spirits available (5 counties)
- IV. Only beer and distilled spirits available (7 counties)
- V. Beer, wine, and distilled spirits all available (71 counties)

Death certificates were classified according to county of residence of the deceased, and then crude alcoholic death rates were calculated based on the populations at risk for each of the five county groupings listed above. Death rates were then adjusted for age and race using the direct method. Adjusted death rates were also calculated separately for each county for correlations with other variables. Figure 1 shows the distribution of adjusted death rates for all 100 counties. The majority of the death rates are less than 6.9 per 100,000, with 21% reporting no deaths. Only 5% had 19 or more deaths per 100,000.

In order to describe more extensively the relationship between alcohol death rate and other variables which might additionally be important in accounting for variations in the alcohol death rates, multiple regressions were performed with the following independent variables: percent of total county population living in urban areas, average per capita income, total population, and index of social change.

RESULTS: Table 1 shows alcoholic death rates that were obtained for the five groups. The adjusted death rates increase from 4.5 per 100,000 to 9.0 per 100,000 in an almost linear fashion. (See also Figure 2).



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The results of the multiple regression analyses help to refine the relationship between alcohol death rate, legal classification, and the other variables of interest (social change, per capita income, population, and percent urban). For purposes of this analysis, the middle three legal groupings were combined (Groups II, III, IV—the only beer and wine, only distilled spirits, and only beer and distilled spirits). Three separate stepwise regressions were run on the data: one for the dry counties, one for the wet counties, and one for the middle groups.

For the dry counties, there were no significant predictors. Social change did, however, account for 19.9% of the variance and was inversely related to alcohol death rate. When all other variables are entered in, the multiple r only improves from 0.4466 to 0.5140.

For the wet counties, percent of the population living in urban areas was significant at the .05 level; however, it only accounted for 5.9% of the variance. Prediction only slightly improves with the addition of the other variables (multiple r goes from 0.2430 to 0.3294).

For the middle counties, the social change index is the only significant predictor ($p < .05$) and accounts for 33.5% of the variance. Even though none of the other variables are significant, multiple r improves from 0.5785 to 0.7311, thus ultimately all the variables can account for 53.4% of the variance.

As a check to see if legal availability and the other factors contribute independently to alcohol death rate, a stepwise regression was performed for all 100 counties. The legal availability classifications were weighted as follows based roughly on the rank order of the percent alcohol of the types of beverages available:

Dry	= 1
Beer, wine	= 2
Distilled	= 3
Beer, distilled	= 4
Wet	= 5

Percent urban was the first factor to be entered and is significant at the .01 level ($F = 8.43$). Legal Availability entered next; the overall regression remained significant ($p < .01$), and the partial F for the contribution of legal availability is significant at the .05 level. These are the only significant predictors.

DISCUSSION: The present study does not enable us to presume a causal effect between alcoholism mortality and the legal availability of alcoholic beverages. We cannot answer definitely whether legislative control affects drinking behavior; however, the association that is found implies some intriguing explanatory hypotheses and some competing hypotheses can be ruled out.

Bias in death certificate reporting has always been a problem in working with alcoholism mortality, and there is a possibility that such a bias could account for part of the observed variability in this study. If there were such a bias, we would expect under-reporting in the "dry" counties and the least populous counties where there would probably be more of a stigma attached to an alcoholic death. The regression analyses showed that there was no relationship between alcoholic death rate and county population. In North Carolina, there is a Medical Examiner System whereby all death certificates are reviewed in a central office to eliminate as much as possible bias and inconsistency in reporting. By January 1, 1971, 67 of the 100 counties were operating under this system, another 17 came on during 1971, and 16 came on after 1971. During 1971, there were 21 counties that reported no deaths. It is these counties where we might expect the reporting during 1971 to be biased; however, of these 21 counties, 17, or 81% were on the Medical Examiner System before 1971, leaving only 4 that came on during or after 1971. Of the 15 "dry" counties, 11 or 73% were on the System before 1971. From these data, it can be concluded that there is no systematic bias in reporting deaths in the dry counties or the counties with no alcoholism mortality since a greater proportion of the possible "high-bias" counties (81% and 73%) were having records reviewed than the 67% of all the counties.

We must consider the possibility that distilled spirits are more toxic than beer and wine, thus explaining the fact that death rates are higher where distilled spirits are available. In other words, if two areas have the same number of drinkers, the area with the largest proportion of distilled spirits drinkers would have a higher alcoholism death rate. Gabriel (1935) did find that the overall death rate of distilled spirits drinkers was higher than the death rate among wine and beer drinkers, but it is likely that the more advanced alcoholics had switched to distilled spirits as their alcoholism progressed, thus inflating the death rate for distilled spirits drinkers. On the other hand, Wallgren and Barry (1970) concluded that type of beverage is not the crucial factor in the development of organic pathology. At this point, it can only be said that the question of differential death rates by type of beverage consumed remains open-ended.

The present study shows that alcohol deaths are not evenly distributed in North Carolina. Based on the regression for all 100 counties, alcohol deaths are more apt to occur in "wet" counties and urban counties. Even though these two factors are correlated ($r = 0.3312$, $p < .01$), the overall regression shows that they are independently significant predictors of alcohol death rate.

A possible explanation for the predominance of deaths in urban areas is that abusive drinkers tend to migrate to urban counties. In fact, Sundby (1967) summarizes a Norwegian study (Bratholm, 1964) which shows that when place of birth and place of residence were

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checked for all incarcerated vagrants on one particular day, both urban-
 borns and urban-immigrants were over represented. In the present
 study, this leads us to question whether urbanization itself as a stres-
 sor is important, or the fact that alcoholics migrate to urban and/or
 wet areas.

The regression analyses that were performed on each of the three
 legal groupings show that there are different groups. Availability is
 in itself an important factor, but it is also an indicator of social senti-
 ment and attitudes toward the use of alcoholic beverages. The voters
 in a county who would vote not to allow alcohol in that county would
 also exert strong pressure not to drink. For the dry counties in this
 study, no factors were found to be significant. Thus, availability, so-
 cial sentiment, and the interaction of these two (as reflected in the
 laws) appear to be the most important factors contributing to alcohol
 death rates.

In the middle counties, (Groups II, III, IV), the fact that social
 change is a significant predictor reinforces the fact that these counties
 have transitional laws and transitional attitudes. Where there is more
 social change the alcohol death rate is higher. Some weight is added
 to this theory when we look at changes in county availability laws from
 1971 to 1973. Four counties made changes during this time: three of
 the middle counties became wet, and one of the dry counties moved
 to the middle category.

In the wet counties where the laws and social sentiment permit and
 encourage drinking, we have the traditional finding that urbanization
 is the most important predictor of alcohol death rate.

Popham *et al.* (1971) show that the amount of excessive drinking in a
 population is directly proportional to the per capita consumption
 in that population. In other words, to lower or raise the per capita con-
 sumption would result in increasing or decreasing the number of ex-
 cessive drinkers. The data in this study could be taken as evidence that
 the legal availability and social sentiment concerning the use of alco-
 holic beverages in fact influences per capita consumption, and correspond-
 ingly influences the number of excessive drinkers and the number of
 alcoholic deaths.

CONCLUSION: The most important contribution of this paper is
 the identification of a new variable, legal availability of alcoholic bev-
 erages, which is associated with alcoholism mortality independently
 of urbanization. More variables need to be explored which will help
 in the development of a more extensive model to explain the variation
 of alcoholism mortality.

Whether legal availability has a direct causal relationship to per cap-
 ita consumption, attitudes toward use of alcoholic beverages, and al-

coholism death rates or vice versa is intriguing and leads into a fruitful area for research. More work needs to be done to separate the influences of law as a reflection or indicator of societal attitudes toward drinking versus availability. This will help to refine the cause-effect relationship. There is probably an interaction effect between attitudes (practice) and availability which will be important to examine closely because of the implications for alcoholism prevention. If attitude is the most important aspect, then education programs designed to raise the level of public awareness and change attitudes will be the best prevention strategies. If, on the other hand, availability is the crucial factor, then laws and other limitations such as taxation will be the best strategies.

References

- Bratholm, A., En sosial-statistisk analyse av klientelet pa Opstad arbeidshus. Innstilling fra Straffelovradet om endringer i de strafferettslige regler om drukkenskapsforseelser og alkoholdmisbruk. Oslo, Justis-og politidepartementet 1964, pp. 61-108.
- Cahalan, D., I. H. Cisin, and H. N. Crossley, American Drinking Practices. A national study of drinking behavior and attitudes. Rutgers Center of Alcoholic Studies, New Brunswick, 1969.
- Dohrenwend, Bruce P. The social psychological nature of stress. *Journal of Abnormal and Social Psychology*, 62:294-302, 1961.
- Gabriel, E., Uber die Todesursachen bei Alkoholikern, *Z. ges Neurol. Psychiat.*, 1935, 153, pp. 385-406.
- Kuusi, Pekka, *Alcohol Sales Experiment in Rural Finland*. Helsinki: Finnish Foundation for Alcohol Studies., Vol 3a, 1957.
- Makela, Klaus, Types of alcohol restrictions, types of drinkers and types of alcohol damages: the case of the personnel strike in the Stores of the Finnish Alcohol Monopoly. Paper presented at the 20th International Institute on the Prevention & Treatment of Alcoholism, Manchester, England, June 23-28, 1974.
- North Carolina Vital Statistics 1971, V. 1. North Carolina State Board of Health.
- Popham, Robert E., Wolfgang Schmidt, and Jan deLint, Epidemiological research bearing on legislative attempts to control alcohol consumption and alcohol problems, in *Law & Drinking Behavior*, Center for Alcohol Studies, University of North Carolina, Chapel Hill, 1971.
- Room, Robin, "Drinking laws and drinking behavior: some past experience," in *Law and Drinking Behavior*, Center for Alcohol Studies, University of North Carolina, Chapel Hill, 1971.

uing and led into a fruitful
 lone to separate the influen-
 cietal attitudes toward drink-
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v klientelet pa Opstad arbeid-
 endringer i de strafferettslige
 kohdmisbruk. Oslo, Justis-og

ey, American Drinking Prac-
 or and attitudes. Rutgers Cen-
 k, 1969.

gical nature of stress. *Journal*
 294-302, 1961.

Alkoholikern, *Z. ges Neurol.*

in *Rural Finland*. Helsinki:
 , Vol 3a, 1957.

is, types of drinkers and types
 nnel strike in the Stores of the
 nted at the 20th International
 t of Alcoholism, Manchester,

. North Carolina State Board

d Jan deLint, Epidemiological
 control alcohol consumption
Behavior, Center for Alcohol
 Chapel Hill, 1971.

g behavior: some past experi-
 enter for Alcohol Studies, Uni-
 1971.

Smith, Harvey L., *Mental Health Centers in North Carolina: Their Com-
 munities Staffs, and Patients*. Division of Health Affairs, University of
 North Carolina at Chapel Hill, July, 1973.

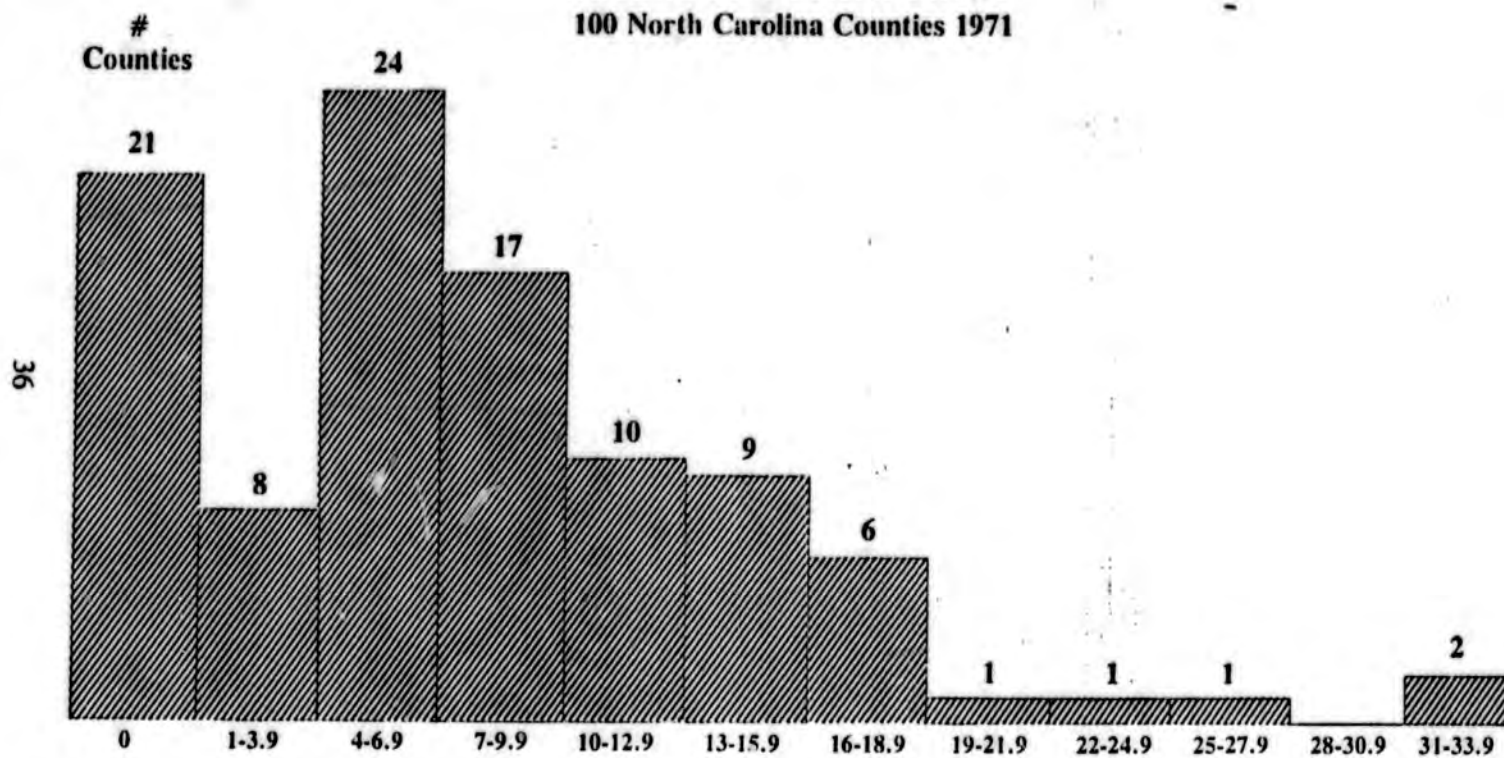
Sundy, Per, *Alcoholism & Mortality*. Universitetsforlaget, Norway, 1967.

Terrism Milton, Epidemiology of cirrhosis of the liver: national mortality
 data. *American Journal of Public Health*, V. 57, No. 12, Dec., 1967.

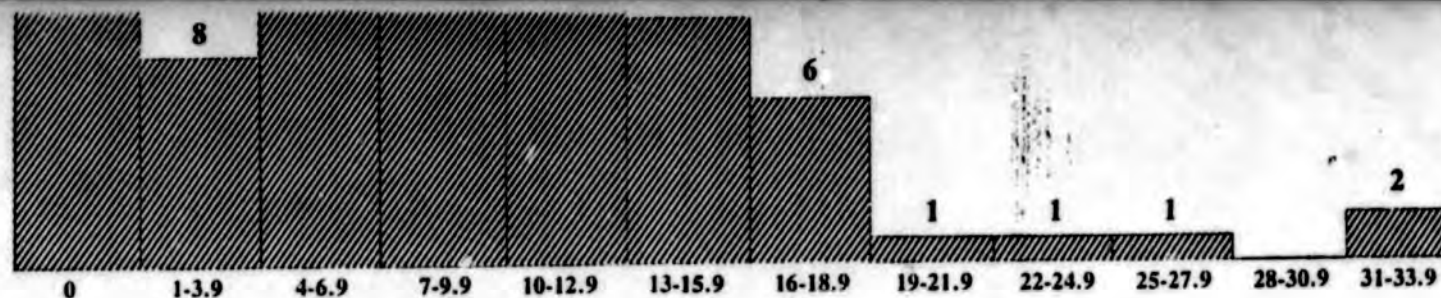
Walgren, H., and Barry, H., *Actions of Alcohol*, V. II, Elsevier Publishing
 Co., Amsterdam, 1970.

Whitehead, Paul C., Effects of changing alcohol control measures. Re-
 vised version of a paper presented to the International Session of the
 ' North American Congress on Alcohol & Drug Problems. San Fran-
 cisco, Dec., 1974.

FIGURE 1
ALCOHOLISM DEATH RATES*
100 North Carolina Counties 1971



* per 100,000 and standardized for age & race.



* per 100,000 and standardized for age & race.

TABLE I: ALCOHOLISM DEATH RATES BY COUNTY LIQUOR LAW

Group	Counties	Beer	Wine	Distilled	Population Total	Alcoholic Deaths	Crude Alc. D. R. [(Alc. Deaths/ Pop. tot.) x 100,000]	Adjusted (for Age and Race) Alc. D.R. per 100,000
I	15	0	0	0	413,437	13	3.1	4.5
II	2	Yes	Yes	0	57,363	4	7.0	5.7
III	5	0	0	Yes	183,126	11	6.0	6.4
IV	7	Yes	0	Yes	310,996	22	7.1	7.5
V	71	Yes	Yes	Yes	4,182,875	375	9.0	9.0
Total	100				5,147,797	*425	8.3	

*All out-of-state alcohol deaths are excluded.

The Relationship of Availability of Alcoholic Beverages to Per Capita Consumption and Alcoholism Rates

Reginald G. Smart¹

SUMMARY. A study of the relationship between availability of beverage alcohol and per capita consumption and alcoholism rates in the U.S.A. indicates that income and urbanism are more closely related to consumption and alcoholism rates than is availability.

NUMEROUS STUDIES have examined the effect of changes in the availability of alcoholic beverages on consumption (1, 2). Some changes appear to have little effect on consumption, e.g., selling alcohol by the drink (3) and increasing the number of outlets in areas where they already exist (1). On the other hand, large changes in numbers of outlets and lowering the legal alcohol-purchasing age do seem to increase consumption. Bruun et al. (2), in an important review of the field, concluded (p. 83) that "the availability of alcohol is an important factor in the general level of consumption." However, studies examining the total range of variables which may affect availability are difficult to find. Bacon (4) studied nine communities in five states and concluded that variations in availability had little effect on consumption. There appear to be no studies which have examined general availability in a wide variety of jurisdictions for effects on consumption.

The purpose of the present study was to examine the relationship between over-all availability of beverage alcohol and per capita consumption and alcoholism rates in the 50 U.S. states and the District of Columbia. The availability of alcoholic beverages was assessed by giving each state a score on 8 items concerning legal beverage control. These scores had been developed for a study

¹ Addiction Research Foundation, 33 Russell St., Toronto, Ont., Canada M5S 2S1. Received for publication: 28 April 1976. Revision: 2 December 1976.

of alcohol control laws.² Efforts were made to differentiate the effects of availability on consumption from those related to income and urban-rural differences in the various states.

METHOD

The availability scores were developed to take account of all legal and control factors which might affect the availability of alcoholic beverages. Initially a 22-item list was developed; 8 items were then selected as more important than the others in influencing availability. The selection was made using expert judgments and a "Delphi" panel approach. The final 8 items were (1) minimal legal age for purchase; (2) limitations on availability for off-premise sales; (3) limitations on availability for on-premise sales; (4) density of outlets for on-premise purchase; (5) Sunday retail sales on premise; (6) weekday closing hours for on-premise sale; (7) Sunday retail sales off-premise; and (8) weekday closing hours for off-premise sale.

On each item each state was defined as falling in the high, medium or low category; values of 6, 4 and 2, respectively, were given to each category. Total scores could vary between 16 and 48, the latter indicating the greatest availability. No effort was made to weight any of the 8 factors more than the others.

The data on per capita consumption and alcoholism were taken from Efron et al. (5). Data on average income and percentage of urban population in each state were taken from the Statistical Abstract of the United States (6).

RESULTS

Table 1 shows all of the data by state, while Table 2 illustrates the mean values of these data when the total sample of states is

TABLE 1.—*Availability Scores, Per Capita Consumption, Income, Urbanism and Alcoholism Rates in the 51 Jurisdictions*

<i>Jurisdiction</i>	<i>Alcohol Availability Score, 1974</i>	<i>Per Capita Consumption, 1972*</i>	<i>Per Capita Personal Income (Dollars) 1972</i>	<i>% Population in Urban Areas, 1970</i>	<i>Rates of Alco- holism, 1970^b</i>
Alaska	44	3.86	5162	48.4	3110
Wyoming	42	2.85	4345	60.5	230
Nevada	40	6.19	5215	80.9	6770
Arizona	38	3.04	4300	79.6	3110
Hawaii	38	2.48	4995	83.1	1780
Louisiana	38	2.46	3528	66.1	3980
Nebraska	38	2.50	4341	61.5	3087

[continued on following page—

² MEDICINE IN THE PUBLIC INTEREST. A study in the actual effects of alcoholic beverage control laws. [Contract No. ADM 281-75-0002.] Washington, DC; 1976.

TABLE 1.—continued

Jurisdiction	Alcohol Availability Score, 1974	Per Capita Consumption, 1972 ^a	Per Capita Personal Income (Dollars) 1972	% Population in Urban Areas, 1970	Rates of Alco- holism, 1970 ^b
New Jersey	38	2.91	5126	88.9	4930
New Mexico	38	2.81	3656	69.8	3160
Rhode Island	38	3.00	4399	87.1	6070
California	36	3.24	5002	90.9	6610
Delaware	36	3.17	4983	72.2	4040
Michigan	36	2.77	4817	73.8	4460
New Hampshire	36	5.42	4092	56.4	3660
New York	36	2.86	5319	85.6	5500
West Virginia	36	1.75	3574	39.0	3130
Colorado	34	2.98	4449	78.5	4240
Illinois	34	2.91	5126	83.0	5140
Maryland	34	3.04	4897	76.6	4100
Massachusetts	34	2.93	4870	84.6	5850
Minnesota	34	2.50	4332	66.4	3400
South Dakota	34	2.16	3716	44.6	2640
Wisconsin	34	3.37	4207	65.9	5110
District of Columbia	32	6.54	6383	100.0	5430
Indiana	32	1.89	4391	64.9	3940
Iowa	32	1.86	4318	57.2	2800
Maine	32	2.60	3571	50.8	4290
Missouri	32	2.28	4206	70.1	5090
Montana	32	2.81	3897	53.4	3730
Tennessee	32	1.74	3640	58.8	2920
Texas	32	2.41	4045	79.7	2830
Vermont	32	3.92	3865	32.2	4170
Connecticut	30	2.73	5342	77.4	4940
Kentucky	30	1.94	3601	52.3	3290
North Dakota	30	2.69	3718	44.3	3570
Oregon	30	2.67	4296	67.1	2610
Alabama	28	1.65	3333	58.4	1830
Arkansas	28	1.52	3357	50.0	2890
Georgia	28	2.25	3846	60.3	2550
Idaho	28	2.43	3635	54.1	1990
Ohio	28	2.71	4512	75.3	4340
Pennsylvania	28	2.32	4447	71.5	4360
Virginia	28	2.23	4258	63.1	2370
Florida	26	3.23	4188	80.5	3590
Mississippi	26	1.88	3063	44.5	2320
Utah	26	1.56	3745	80.4	2070
Washington	26	2.71	4476	72.6	2910
Kansas	18	1.72	4593	66.1	2490
Oklahoma	18	1.80	3802	68.0	2140
South Carolina	16	2.44	3448	47.6	2760
North Carolina	16	1.93	3721	45.0	2050

^a U.S. gallons of absolute alcohol consumed by population aged 15 and over (5, Table 2).

^b Per 100,000 adults aged 20 years and over (5, Table 13).

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Population in Urban Areas, 1970	Rates of Alco- holism, 1970 ^b
48.4	3110
60.5	2300
80.9	6770
79.6	3110
83.1	1780
66.1	3980
61.5	3087

continued on following page—

ual effects of alcoholic
Washington, DC; 1976.

divided into three subgroups (high, medium and low) according to alcohol availability scores.

To determine whether there were significant intergroup differences, mean scores on each variable were compared and *t*-ratios were calculated (Table 2). The low-availability states had a significantly lower average alcoholism rate and per capita consumption than the high- or middle-availability states. Furthermore, although the three groups did not differ significantly in their average percentage of urban population, the high-availability states did have a significantly higher per capita income than the low-availability states.

Partial correlation analyses were made in which the correlation between availability scores of states was correlated with per capita consumption and alcoholism rates, alone and with the correlation with average income and degree of urbanism controlled or partialled out. Tests for significance of the partial correlations were made with the *t* tests described by McNemar (7).

As shown in Table 3, the correlation between per capita consumption and availability is positive and significant. However, the correlation between per capita consumption and income is even greater, and that with urbanism is also positive.

The partial correlations show that much of the correlation between per capita consumption and availability is accounted for by income and urbanism. When income is controlled, the correlation is nonsignificant; however, with urbanism controlled availability and per capita consumption are still correlated ($r = .362$, $p < .01$).

TABLE 2.—Average Alcohol Availability Score, Per Capita Consumption and Income, Percentage of Population in Urban Areas and Rates of Alcoholism in States with High, Medium and Low Availability

Availability	Availability Score	Per Capita Consumption	Per Capita Income	% of Population in Urban Areas	Rates of Alcoholism
1. High (N = 16)	38.00	3.21	4553.38	71.49	4106.06
2. Medium (N = 20)	32.30	2.80	4343.50	65.38	4004.50
3. Low (N = 15)	24.53	2.16	3894.93	62.49	2710.67
<i>t ratios:</i>					
Groups 1 and 2	8.96*	1.14	0.94	1.14	0.24
Groups 1 and 3	9.83*	3.42*	3.30*	1.80	3.26*
Groups 3 and 2	-6.04*	-2.43*	-2.12	-0.06	-4.10*

* $P < .05$.

TABLE 3.—Correlations Between Availability Scores, Per Capita Consumption, Income, Urbanism and Alcoholism Rates

	1	2	3	4
	Availability	Consumption	Income	Urbanism
Availability score		.41†	.39†	.25
Per capita consumption			.60†	.34*
Income				.72†

$$r_{12.3} = .248, t = 1.78.$$

$$r_{12.4} = .362, t = 2.70.†$$

$$r_{12.34} = .244, t = 1.74.$$

	1	2	3	4
	Availability	Alcoholism	Income	Urbanism
Availability score		.42†	.39†	.25
Alcoholism rate			.59†	.53†
Income				.72†

$$r_{12.3} = .251, t = 1.80.$$

$$r_{12.4} = .345, t = 2.57.†$$

$$r_{12.34} = .265, t = 1.93.$$

$$* P < .05. \quad † P < .01.$$

When the correlation is controlled for both urbanism and income, availability and consumption are unrelated.

Similar results are obtained for alcoholism rates, which are also positively related to availability, but more strongly to income and urbanism. When the effects of income, and income and urbanism together, are controlled, availability scores and alcoholism rates are not significantly correlated.

DISCUSSION

It appears that over-all availability of beverage alcohol, as indicated by control laws, has, at best, a spurious correlation with both per capita consumption and alcoholism rates. The positive correlation disappears when the effects of average income and degree of urbanism are partialled out. This suggests that slight changes in availability may have relatively little effect on either consumption or alcohol problems, provided neither income nor urbanism are changed.

Certain cautionary notes are in order. The availability scores are constructed to give each element (e.g., opening hours, legal age) the same score and this may not be the most realistic weighting. Unfortunately, price has not been included as an item in the availability scores since all of the elements relate to laws. Also, the range of per capita consumption is not very great in the 51 jurisdictions studied. Cross-national comparisons of a variety of countries with widely differing availabilities and levels of per capita consumption might give different results.

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Consumption and Rates of Alcoholism Availability

Population Urban Areas	Rates of Alcoholism
71.49	4106.06
55.38	4004.50
32.49	2710.67

1.14	0.24
1.80	3.26*
0.06	-4.10*

REFERENCES

1. POPHAM, R. E., SCHMIDT, W. and DE LINT, J. The effects of legal restraint on drinking. Pp. 579-625. In: KISSIN, B. and BEGLEITER, H., eds. *The biology of alcoholism. Vol. 4. Social aspects of alcoholism.* New York; Plenum; 1976.
2. BRUUN, K., EDWARDS, C., LUMIO, M., MÄKELÄ, K., PAN, L., POPHAM, R. E., ROOM, R., SCHMIDT, W., SKOG, O.-J., SULKUNEN, P. and ÖSTERBERG, E. Alcohol control policies in public health perspective. (Finnish Foundation for Alcohol Studies, Vol. 25.) Helsinki; 1975.
3. BRYANT, C. W. Effects of sale of liquor by the drink in the State of Washington. *Quart. J. Stud. Alc.* 15: 320-324, 1954.
4. BACON, S. D. The role of law in meeting problems of alcohol and drug use and abuse. *Int. Congr. Alcm Drug Depend., Proc.*, 29th, pp. 162-172, 1971.
5. EFRON, V., KELLER, M. and GURIOLI, C. Statistics on consumption of alcohol and on alcoholism. New Brunswick, NJ; Rutgers Center of Alcohol Studies; 1974.
6. U.S. DEPARTMENT OF COMMERCE. BUREAU OF THE CENSUS. Statistical abstract of the United States. Washington, DC; 1973.
7. MCNEMAR, Q. Psychological statistics. 3d ed. New York; Wiley; 1962.

M. M. Field evaluation
report. Prepared for the
in Arbor; University of

Post, D. V., MUDGE,
blem drinkers: report on
ghway Safety Research

Alcohol Availability, Alcoholic Beverage Sales and Alcohol-Related Problems¹

Jerome Rabow, Ph.D. and Ronald K. Watts, Ph.D.²

SUMMARY. *A theoretical integration and an empirical test of the sociocultural model and the distribution of consumption model of alcohol problems is made for 51 California counties. In the counties of California, the kinds and number of outlets of alcoholic beverages are differentially related to the social area characteristics and to arrest rates for public drunkenness, misdemeanor and felony drunken-driving arrest rates and cirrhosis mortality rates.*

Two sociological perspectives have dominated the theoretical and empirical literature on drinking behavior and alcohol-related problems in the past several decades. The older of the two, the sociocultural model, has provided the impetus for over four decades (1, 2). This model emphasizes the structure of social norms as manifested in different populations or subgroups. The second perspective, developed more recently, is the distribution of consumption model and, while being similar to the sociocultural model in the attempt to examine differences in consumption and problems across subgroups, it also focuses on the overall level and rate of consumption in a society: rates of alcohol problems or damage are seen here as being inexorably tied to rates of consumption (3-6). This model has been used by some researchers who have examined the physical availability of alcohol and its relationship to alcohol problems. Thus, rates of alcoholic beverage outlets have

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² Authorship is alphabetical. Our work the past 3 years has been a collaborative effort.

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been substituted or added to the original model of rates of consumption, and these have been examined in relation to a problem indicator; e.g., liver cirrhosis mortality, or to consumption itself (7-10).

The models have been treated as competing, when, in fact, a synthesis is possible in which the normative structure of subgroups may be seen to be reflected in differential consumption and consequent alcohol-related problems. Frankel and Whitehead (11), using path analysis, tested an integrated model with data from 68 societies and found relatively more support for the distribution of consumption model.

Although they made an important contribution to theoretical and empirical analysis of alcohol problems, Frankel and Whitehead (11) tested their model with data from whole societies, most of which were small and of greatest interest to anthropologists. We propose an alternative model that may be tested within one large-scale industrial society: the United States. Our model is based on two assumptions: (1) that different normative patterns with respect to drinking behavior and possible alcohol-related damage may be operationalized by referring to various subgroups which manifest those norms; and (2) that alcohol consumption may be operationalized by the differential distribution of various types of alcoholic beverage outlets.

The recent concern over the issue of availability and its effects on consumption and alcohol problems grew out of the work of Bruun et al. (12). They found that large changes in the number of alcoholic beverage outlets and lowering the legal age for purchase of alcoholic beverages appeared to increase consumption. They concluded that "the availability of alcohol is an important factor in the general level of consumption" (12, p. 83). More recently, several researchers tested hypotheses about availability and its effects on both consumption and liver cirrhosis mortality. Smart (7), using an 8-item scale of availability, found that income and urbanization were more closely related to consumption and alcoholism rates than was availability. Parker et al. (9), using essentially the same data but substituting a more narrowly defined measure of availability (rates of on-premise and off-premise sales outlets), found strong effects of beverage availability on both per capita consumption and alcoholism rates in states not having restrictions on the number of outlets per unit of population. Harford et al. (10), using only rates of on-premise outlets, found that when consumption and income

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were controlled, outlet rates and urbanization were significantly related to rates of alcoholism in 38 states and the District of Columbia.

These analyses used aggregate data of states and the District of Columbia to examine the relationships among alcohol problems while controlling for relevant sociodemographic variables. These research findings are important starts at investigating the relationships between alcohol availability and consumption; however, they have generated results that are at times conflicting. On at least one point, they can be considered deficient since they do not account for tourism: travel to the various states and the District of Columbia is differentially distributed and accounts for more variation in consumption of alcoholic beverages than does availability, urbanism or income (13).

Another problem with analyzing state aggregate data is that the considerable heterogeneity within states with respect to outlet rates, consumption and sociodemographic features is not tapped. Clearly what is needed are more "refined" indicators to allow more extensive testing of a number of hypotheses for which, at present, the evidence is contradictory and confusing.

Although both Parker et al. (9) and Harford et al. (10) were critical of Smart's (7) use of an 8-item index as a measure of availability, the internal properties of which they claimed had not been tested, and although they restricted definitions of availability to the presence of outlets, both on- and off-premise (9) and on-premise only (10), they proceeded to use the same unit of analysis as had Smart. They thereby continued to "mix" conceptions of availability, while other factors, such as hours of sale and whether beverage alcohol was sold on Sundays, may be influencing consumption and alcohol-related problems. Although the analyses are useful in pointing out the role of outlet availability in influencing consumption and problems, we also believe that it would be more useful to examine relationships within a state where there is no (or little) local control over the licensing of alcoholic beverage outlets. Our study presents data for 51 of the 58 counties in California and concentrates on the sociodemographic characteristics of counties in conjunction with the rates of several types of on-premise and off-premise outlets.³ By examining specific types of alcoholic beverage

³ Smart (8) has suggested that there are other aspects of availability that need to be investigated. Given the type of data we have employed in our analysis, we could not follow

outlets in conjunction with sociodemographic characteristics of the counties, we can sort out their relative effects on selected alcohol-related problems.

In particular, we were interested in comparing the relative merits of hypotheses derived from the two approaches to alcohol problems. By employing correlation and regression techniques we examined the effects of selected sociodemographic and outlet characteristics on the dependent variables of interest.

Hypotheses

Previous research (14-16) has shown that social rank is negatively related to alcohol problems, although there is some contrary evidence (17), while urbanization and minority race-ethnicity are positively related to alcohol problems (15, 18, 19). On the basis of this research, we tested the following hypotheses:

1. Social status is inversely related to alcohol-related problems.

That is, social status as measured by a number of indicators, such as income, education and occupation, is negatively associated with alcohol-related problems such as arrests for alcohol-related offenses and cirrhosis mortality.

2. Urbanization is positively associated with alcohol-related problems.

That is, the more urbanized an area, the greater the level of alcohol-related arrests and cirrhosis mortality. To the extent that some minorities (e.g., Spanish-Americans, Blacks and American Indians) may have norms that differ from those of the dominant group, the degree to which their relative populations vary across units of analysis is positively related to alcohol-related problems.⁴ Therefore, the following hypothesis was considered:

Smart's suggestions. We considered taxable retail sales of packaged liquor stores only in the bivariate correlations.

⁴ Of course one could plausibly argue that these groups are no more likely than others to experience alcohol-related damage, but are merely more subject to the scrutiny of the police (in the case of public drunkenness arrests or misdemeanor drunken driving), or whose deaths are more likely to be attributed to cirrhosis. However, this is not the premise upon which many Black leaders operate when they call for controls over certain types of alcoholic beverage outlets in their communities.

3. Minority status is positively related to alcohol-related problems of the types considered in the present analysis.

The degree to which people are integrated into a familial and community setting, it is theorized by the socioculturalists, should be inversely related to the extent to which alcohol-related damage occurs. Accordingly, the following hypothesis was investigated:

4. Household and family structure and familism are inversely related to alcohol-related problems.

Alcohol-related damage should be directly related to the extent to which alcohol is both available and consumed. The distribution of consumption advocates have argued that alcohol consumption is positively associated with a particular type of medical complication, liver cirrhosis, and that this relationship holds across societies and populations. Much attention has been addressed recently to the issue of alcohol consumption and policies directed at controlling consumption through the limitation of physical availability. The two methods in question are price manipulation (12, 20).⁵ and limitations on the number of outlets per given unit of population (21). Accordingly, on the basis of previous research, the following hypothesis was tested:

5. The physical availability of alcohol and its consumption are positively related to alcohol-related damage.

Both consumption of alcoholic beverages and their physical availability should not be randomly distributed across social areas. That is, both availability and consumption should be in part determined by the area's social characteristics. Therefore, the following hypothesis was tested:

6. Outlet types, or the physical availability (both on- and off-premise sales) of specific types, are related in specific ways to the population characteristics of the units of analysis.

That is, some types of outlets, such as those located in bona fide eating places, are likely to be relatively more common in areas of

⁵ LAU, H.-H. Consumption of alcoholic beverages in Canada—a reply to Lidman. Ottawa: 1977.

higher social rank, nonminority areas and relatively more urbanized areas.

METHOD

Recently Donnelly (17) demonstrated the usefulness of the Mental Health Demographic Profile System (MHDPS) (22, 23) in analyzing alcoholic beverage sales and alcohol-related problems in Pennsylvania (a monopoly state) within the framework of social area analysis (24). The present study replicates and extends Donnelly's approach to the counties of California. It replicates his use of selected county social profile characteristics in the analysis of alcoholic beverage sales and alcohol problems. It extends his analysis by considering specific types of arrests—public drunkenness, misdemeanor drunken-driving and felony drunken-driving—each of which is hypothesized to be differentially related to social area characteristics. Donnelly's use of total arrests may have obscured differential relationships between specific types of arrests and both social area characteristics and beverage sales. Furthermore, our analysis extends both Donnelly's work and the work of Parker and Harmon (6), Smart (7), Parker et al. (9) and Harford et al. (10) by examining types of on-premise and off-premise outlets and their relationships both to social area profile characteristics and to specific alcohol-related problems.

The Data

As a nonmonopoly state California regulates alcoholic beverage outlets through a licensing process performed by the state Alcoholic Beverage Control board (ABC). In 1976, there were 54,450 fiscal- and calendar-year licenses in force in California. The great majority of these licenses (90%) were of 2 off-premise and 5 on-premise outlet types.

To test whether sociodemographic variables in conjunction with availability and consumption variables influence selected indicators of alcohol problems, a data system was developed from several sources and is similar to the one used by Donnelly (17).

Availability. Information on the number of 7 specific types of outlets was obtained from the state ABC for the years 1976 and 1977 for all counties in the state. Of 25,563 fiscal-year licenses in force in 1976, 89% were retailers' type off-premise sales outlets: Type 20, beer and wine only, 11,929; and Type 21, general (all types of alcoholic beverages), 10,937. Of 28,887 calendar-year licenses in force in 1976, 91% were of 5 types of on-premise outlets: Type 40, beer only, 6069; Type 41, beer and wine for bona fide eating places, 3113; Type 42, beer and wine for public premises, 1751; Type 47, general for bona fide eating places, 6836; and Type 48, general for public premises, 4586.

Consumption. Donnelly obtained alcoholic beverage sales information for state-operated outlets in Pennsylvania and created a per capita sales variable as an indicator of consumption; however, it did not include beer sales since state-run outlets in Pennsylvania only sell distilled spirits and

wine. Our indicator of consumption is based on taxable retail sales information of packaged liquor stores (Type 21), licensed to carry distilled spirits, wine and beer. Unfortunately, in California other taxable items are marketed through packaged liquor stores. Bunce⁶ concluded that approximately 75% of taxable retail sales in packaged liquor stores consists of alcoholic beverages. Thus, taxable retail sales may be considered as a rough indicator of consumption at the county level. Taxable retail sales were gathered for years 1971-75 for 51 of the 58 counties in California.⁷

Alcohol-Related Problems. Selected indicators were gathered from the State Office of Alcoholism.⁸ They included adult arrests (aged 18 and over) for public drunkenness (California Criminal Code, section 647f), misdemeanor drunken driving (856m) and felony drunken driving (850f). The latter is drunken driving where an injury has occurred resulting from an accident. Cirrhosis mortality counts for the years 1970-74 were also obtained from the state Office of Alcoholism for the 51 counties.

Rates were created to remove the gross effects of differences in population size across counties, using as the denominator persons aged 15 and over on 1 April 1970. The basic rates for the counties are: (1) Seven types of retail beverage outlets for 1976 and 1977 per 1,000 persons; (2) per capita taxable retail sales for the years 1972-75; (3) several types of arrests for the years 1972-75 per 1,000 persons; and (4) cirrhosis mortality 1970-74 per 100,000 persons. These rates were averaged over the years available to reduce the effects of year-to-year fluctuations.⁹

Mental Health Demographic Profile System (MHDPS). The MHDPS was created by the National Institute of Mental Health (22, 23) from the second and fourth counts of the 1970 Census as a tool for the analysis of social areas in health planning. The initial efforts of the institute were directed toward identifying demographic and ecological dimensions that would be useful in differentiating among residential subareas of American cities and that could also be derived from census data. Of particular concern was the desire to identify areas "with high risk populations, that is, populations with those characteristics which past studies have shown to be associated with high rates of mental illness and/or high incidence of social disorganization or disruption" (22).

⁶ BUNCE, R. Meanings and interrelationships of alcohol-related problems and indicators. Berkeley; University of California, Social Research Group; n.d. [Mimeographed.]

⁷ Although the State Board of Equalization reports taxable retail sales of all kinds for most county (and city) jurisdictions, it does not release taxable sales figures for jurisdictions where there are only a few outlets. Doing so would reveal information about individual businesses. Casual inspection of their reports suggests that four is the number of reporting permits below which information on sales is withheld. Seven counties (Alpine, Calaveras, Colusa, Mariposa, Modoc, Sierra and Trinity) were eliminated from the analysis.

⁸ MAXFIELD, T. and KLAUS, A. An in-depth analysis; alcohol-related arrest data, 1971-75. Sacramento; California State Office of Alcoholism; n.d. [Mimeographed.]

⁹ Though these data were obtained for different years, we felt reasonably safe in assuming that the cross-sectional nature of the relationships in the data would be revealed with any similar set of data from the recent period.

The principal theoretical underpinning of this type of research lies in the concept of "social area analysis" first proposed by Shevky and Bell (24) and Greer (25). Greer suggested that social area analysis is based on the notion that "residence-related behavior can be understood and accounted for in terms of three types of society-wide population characteristics or dimensions: social rank, life-style or urbanization, and ethnicity." Greer provided definitions for these dimensions: (1) Social rank "indicates the 'objective' factors of social class: occupation, education and income"; (2) life-style "refers to the way of life chosen by the population, whether the 'family-committed life' in the single-dwelling-unit area, or the life of the working couple in the apartment-house areas"; (3) Ethnicity refers to "the differentiation of population by racial and cultural background; the proportion and kinds of minority groups to be found in the neighborhood."

Social area analysis grew out of the ecological analysis of area units and has had a venerable tradition in the social sciences, particularly sociology.¹⁰

RESULTS

Table 1 presents the basic data for the 51 counties analyzed in the state of California. Before presenting the results of the correlation and regression analyses, we briefly discuss the salient features of the system of alcohol-related variables.

The first point that should be made about Table 1 is that there are 17 counties with less than 50,000 persons aged 15+ in 1970. A perusal of the table suggests that it is often the smaller counties which have relatively higher outlet rates, with Mono County having the highest rate for 5 of the 7 outlets (Types 20, 21, 40, 41, 47) and the smallest population (2995).

With respect to per capita taxable retail sales of packaged liquor stores, two counties, Mono (\$182.00; 2995 persons aged 15+) and Inyo (\$132.00; 11,458 persons aged 15+) have the highest per capita sales. However, inspection of the sales of other small counties suggests that county size is not positively related to sales. This will be shown more clearly when we discuss the relationships between these variables and the MHDPS.

Two jurisdictions, Fresno and Yuba, had public drunkenness arrest rates (53.4 and 63.7 per 1000 persons, respectively) several standard deviations above the mean while Napa County had an arrest rate somewhat greater than one standard deviation below the

¹⁰ Durkheim (26) was one of the first to study area characteristics and relate them to suicide rates.

TABLE 1.—Rates of On- and Off-Premise Alcoholic Beverage Outlets, Per Capita Taxable Retail Sales of Packaged Liquor Stores, Alcohol-Related Arrests, and Cirrhosis Mortality, by 51 Counties in California*

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
Alameda	0.645	0.819	0.269	0.509	0.110	0.311	0.438	97.20	14.80	12.525	4.230	27.56	3.25	703.126
Amador	2.736	1.751	0.930	1.313	0.328	2.408	1.478	41.13	7.53	9.148	0.323	17.00	1.97	913.36
Butte	1.372	0.712	0.550	0.718	0.233	0.427	0.324	78.95	5.51	9.793	0.224	15.53	2.20	77.258
Contra Costa	0.798	0.605	0.226	0.437	0.055	0.388	0.316	87.37	5.20	11.649	0.214	17.08	2.85	391.525
Del Norte	3.046	1.376	0.442	0.737	0.098	0.688	0.737	84.18	5.13	22.402	0.530	28.12	2.55	101.78
El Dorado	2.288	0.747	0.794	1.478	0.187	0.731	0.140	58.54	10.94	16.821	0.273	28.03	2.68	321.30
Fresno	1.650	0.736	0.780	0.446	0.172	0.408	0.341	79.79	53.37	11.671	0.484	65.53	3.69	290.216
Glen	1.413	1.334	0.785	0.628	0.275	0.471	0.706	70.35	6.53	30.199	0.504	36.94	1.86	127.41
Humboldt	1.344	1.198	0.340	0.582	0.312	0.547	0.721	47.32	13.01	18.030	0.594	31.63	2.69	721.55
Imperial	2.594	1.005	1.733	0.574	0.185	0.592	0.400	75.59	26.88	41.701	0.594	69.15	3.96	87.604
Inyo	1.020	1.484	0.742	1.527	0.305	0.873	0.524	132.90	20.51	11.143	0.149	32.60	2.97	227.340
Kern	1.474	0.684	0.607	0.367	0.288	0.109	0.345	96.69	21.72	10.774	0.101	28.91	2.56	437.37
King	1.669	0.686	0.816	0.343	0.080	0.411	0.354	69.35	15.12	13.617	0.177	28.91	2.56	437.37
Lake	3.632	1.671	1.043	1.029	0.366	1.029	0.332	90.29	12.37	17.962	0.276	30.63	2.70	155.56
Lassen	1.744	1.581	0.459	0.642	0.184	1.239	0.688	18.58	13.86	7.139	0.329	21.33	1.84	108.97
Los Angeles	0.610	0.701	0.367	0.399	0.118	0.368	0.221	95.81	15.58	22.452	0.257	38.32	3.32	514.870
Madera	2.245	0.686	0.960	0.900	0.103	0.514	0.309	73.59	21.82	26.167	0.683	51.69	3.09	201.73
Martin	0.514	0.729	0.156	1.024	0.027	0.563	0.245	62.33	2.97	14.622	0.164	17.76	2.31	159.000
Mendocino	2.098	1.372	0.551	0.982	0.134	0.929	0.847	56.25	14.01	13.895	0.316	28.22	3.00	371.80
Merced	1.711	0.686	0.912	0.474	0.191	0.452	0.410	85.51	13.55	23.566	0.251	37.37	2.49	707.25
Mono	7.513	5.342	2.003	6.845	0.0	5.809	0.334	182.51	17.78	12.980	0.952	31.71	2.67	299.5
Monterey	0.982	0.609	0.488	0.971	0.145	0.493	0.252	80.15	17.60	13.210	0.185	31.04	1.93	182.005
Napa	1.048	0.659	0.423	0.761	0.135	0.634	0.482	54.85	0.47	15.256	0.201	15.93	3.35	501.38
Nevada	1.383	1.443	0.647	1.393	0.199	1.742	0.771	70.72	10.05	17.856	0.184	28.09	3.58	299.96
Orange	0.631	0.685	0.291	0.585	0.132	0.560	0.129	96.52	9.63	12.305	0.188	22.12	1.44	982.315
Placer	1.323	1.190	0.595	0.835	0.240	0.746	0.506	56.08	10.34	9.661	0.233	20.25	2.66	56.299
Plumas	4.162	4.394	1.272	1.503	0.0	2.639	1.676	53.56	13.87	12.872	0.201	27.24	4.02	86.49
Riverside	1.201	0.632	0.664	0.667	0.128	0.616	0.150	88.69	12.06	17.722	0.279	30.08	2.54	329.091
Sacramento	0.844	0.633	0.377	0.523	0.219	0.441	0.331	49.68	17.05	17.683	0.255	34.99	3.11	445.317
San Benito	1.749	0.795	0.636	0.795	0.040	0.705	0.954	79.07	17.95	17.864	0.203	36.02	2.70	12.460
S. Bernardino	1.101	0.593	0.632	0.409	0.143	0.242	0.242	79.22	6.69	19.363	0.272	26.33	2.85	478.709
San Diego	0.780	0.619	0.375	0.473	0.189	0.501	0.202	70.54	10.55	17.866	0.109	28.53	2.78	997.022
S. Francisco	0.619	1.814	0.253	0.904	0.040	0.950	1.002	90.70	26.63	6.014	0.074	32.72	7.71	583.107
S. Joaquin	1.313	0.751	0.457	0.462	0.251	0.421	0.505	77.35	29.15	10.719	0.180	50.05	3.68	296.022
S. Luis Obispo	1.556	0.711	0.631	1.030	0.055	0.509	0.366	90.45	7.53	12.259	0.324	20.21	2.97	81.585

continued —

TABLE 1.—Rates of On- and Off-Premise Alcoholic Beverage Outlets, Per Capita Taxable Retail Sales of Packaged Liquor Stores, Alcohol-Related Arrests, and Cirrhosis Mortality, by 51 Counties in California*

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
Alameda	0.645	0.819	0.260	0.509	0.110	0.311	0.438	97.20	14.80	12.525	6.230	27.56	3.25	793,128
Alameda	2.736	1.751	0.930	1.313	0.328	2.408	1.478	41.13	7.53	9.148	0.323	17.06	1.07	91,366
Butte	0.372	0.712	0.550	0.718	0.233	0.427	0.324	78.95	5.51	9.703	0.224	15.53	2.20	77,258
Contra Costa	0.798	0.605	0.259	0.437	0.055	0.368	0.316	87.37	5.20	11.649	0.214	17.06	2.85	391,525
Del Norte	3.046	1.378	0.442	0.737	0.098	0.688	0.737	84.18	5.13	22.402	0.530	28.12	2.55	101,778
El Dorado	2.288	0.747	0.794	1.478	0.187	0.731	0.140	58.54	10.94	16.821	0.273	28.03	2.68	321,300
Fresno	1.650	0.736	0.780	0.446	0.172	0.408	0.341	72.79	53.37	11.671	0.484	36.94	1.88	290,218
Glenn	1.413	1.334	0.785	0.628	0.275	0.471	0.706	79.35	6.53	30.190	0.296	30.94	1.88	127,411
Humboldt	1.344	1.199	0.340	0.582	0.312	0.547	0.721	47.32	13.01	18.030	0.594	31.63	2.69	72,155
Imperial	2.594	1.005	1.733	0.574	0.185	0.502	0.400	75.59	26.89	41.761	0.594	69.15	3.98	48,768
Inyo	1.020	1.481	0.742	1.527	0.305	0.873	0.524	112.90	20.51	11.443	0.149	31.80	5.93	11,458
Kern	1.474	0.684	0.697	0.367	0.288	0.499	0.335	96.69	21.72	10.774	0.101	32.60	2.97	227,300
Kings	1.669	0.686	0.846	0.343	0.080	0.411	0.354	69.35	15.12	13.617	0.177	28.91	2.56	43,737
Lake	3.632	1.671	1.033	1.029	0.386	1.029	0.932	99.29	12.37	17.982	0.276	30.63	2.70	155,586
Lassen	1.744	1.581	0.459	0.642	0.184	1.231	0.688	18.58	13.86	7.139	0.329	21.33	1.81	108,97
Los Angeles	0.610	0.701	0.367	0.399	0.118	0.368	0.221	95.81	15.58	22.452	0.257	38.32	3.32	514,570
Madera	2.245	0.686	0.960	0.900	0.103	0.514	0.309	73.59	21.82	26.187	0.683	51.69	3.08	201,73
Marin	0.514	0.729	0.156	1.024	0.027	0.563	0.245	62.33	2.97	14.622	0.164	17.76	2.31	159,809
Mendocino	2.098	1.372	0.551	0.982	0.134	0.928	0.847	56.25	14.01	13.895	0.316	28.22	3.00	37,180
Merced	1.711	0.686	0.912	0.474	0.191	0.452	0.410	85.51	13.55	23.566	0.251	37.37	2.49	70,725
Mono	7.513	5.342	2.003	6.845	0.0	5.509	0.334	162.51	17.78	12.980	0.952	31.71	2.07	29,95
Monterey	0.982	0.669	0.488	0.971	0.145	0.499	0.252	80.15	17.66	13.210	0.165	31.04	1.93	182,305
Napa	1.048	0.659	0.423	0.761	0.135	0.634	0.482	54.85	0.47	15.256	0.201	15.93	3.35	501,38
Nevada	1.393	1.443	0.647	1.393	0.199	1.712	0.771	70.72	10.05	17.856	0.184	28.09	3.58	209,96
Orange	0.631	0.685	0.201	0.585	0.132	0.560	0.129	58.52	9.63	12.305	0.188	22.12	1.44	982,315
Placer	1.323	1.190	0.595	0.835	0.240	0.746	0.506	56.08	10.34	9.681	0.233	20.25	2.60	56,299
Plumas	4.102	4.394	1.272	1.503	0.0	2.659	1.676	53.56	13.87	12.872	0.201	27.24	4.62	86,419
Riverside	1.201	0.632	0.664	0.667	0.128	0.618	0.150	88.69	12.08	17.722	0.279	30.08	2.54	320,981
Sacramento	0.844	0.633	0.377	0.523	0.219	0.441	0.331	48.68	17.05	17.683	0.255	34.99	3.11	445,337
San Benito	1.749	0.795	0.636	0.795	0.040	0.705	0.954	79.07	17.95	17.684	0.203	36.08	2.70	12,580
S. Bernardino	1.101	0.503	0.632	0.409	0.143	0.473	0.242	79.22	6.69	19.363	0.272	26.33	2.85	478,709
San Diego	0.780	0.619	0.375	0.473	0.189	0.591	0.202	70.51	10.55	17.868	0.109	28.53	2.78	997,922
S. Francisco	0.619	1.814	0.253	0.904	0.040	0.950	1.002	90.70	26.63	6.014	0.074	32.72	7.71	583,107
S. Joaquin	1.313	0.751	0.457	0.462	0.251	0.421	0.505	77.35	39.15	10.719	0.180	50.05	3.68	209,022
S. Luis Obispo	1.526	0.711	0.631	1.030	0.055	0.509	0.398	90.45	7.53	12.259	0.324	20.21	2.97	81,585

continued —

TABLE 1.—continued

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
San Mateo	0.620	0.502	0.232	0.609	0.048	0.455	0.272	86.52	4.73	13.885	0.217	18.81	2.59	409186
Santa Barbara	0.823	0.646	0.510	0.653	0.120	0.505	0.187	94.95	7.78	13.625	0.179	21.58	2.57	192058
Santa Clara	0.697	0.644	0.257	0.570	0.073	0.486	0.272	92.22	7.15	10.619	0.203	17.97	2.45	735967
Santa Cruz	1.205	0.812	0.446	1.035	0.154	0.605	0.504	99.92	9.32	12.914	0.355	22.50	3.35	94224
Shasta	2.077	0.811	0.957	0.856	0.282	0.456	0.547	69.13	13.25	17.062	0.269	30.58	3.79	54879
Siskiyou	1.759	2.598	0.593	0.839	0.205	1.084	0.941	74.62	10.97	11.637	0.177	22.78	3.03	24410
Solano	0.846	0.720	0.398	0.540	0.163	0.343	0.435	64.72	3.09	17.092	0.270	20.45	2.51	119412
Sonoma	1.050	0.881	0.313	0.791	0.279	0.632	0.565	58.05	8.02	8.354	0.221	16.60	3.28	148619
Stanislaus	1.555	0.627	0.475	0.471	0.127	0.370	0.388	83.47	17.06	16.826	0.423	34.31	2.19	137907
Sutter	1.552	0.597	0.648	0.273	0.085	0.222	0.546	36.40	6.53	9.040	0.186	15.76	1.64	29317
Tehama	1.837	1.002	0.692	0.453	0.429	0.575	0.239	71.88	9.94	11.561	0.155	20.66	2.00	20962
Tulare	1.963	0.644	0.679	0.357	0.077	0.418	0.288	66.57	17.63	17.717	0.231	35.58	2.32	130421
Tuolumne	2.192	1.599	1.007	1.244	0.178	1.333	1.125	59.97	11.56	11.174	0.407	23.14	4.03	16883
Ventura	0.712	0.698	0.395	0.419	0.126	0.474	0.245	82.17	8.20	12.125	0.195	20.52	2.03	150857
Yolo	1.039	0.643	0.359	0.718	0.120	0.426	0.336	69.81	14.62	20.208	0.192	35.02	2.21	66867
Yuba	1.500	1.134	0.884	0.333	0.150	0.567	0.784	36.01	63.72	21.120	0.179	85.02	3.00	29993
Mean	1.63	1.11	0.63	0.85	0.16	0.78	0.51	76.11	14.38	15.38	0.28	21.47	2.94	282186
SD	1.14	0.89	0.36	0.92	0.10	0.82	0.33	25.21	11.45	6.18	0.16	91.65	1.04	737741

(1) Type 20: Mean off-premise sales, beer and wine, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(2) Type 21: Mean off-premise sales, general outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(3) Type 40: Mean on-premise sales, beer-only outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(4) Type 41: Mean on-premise sales, beer and wine for bona fide eating place outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(5) Type 42: Mean on-premise sales, beer and wine for public premises outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(6) Type 47: Mean on-premise sales, general for bona fide public eating place outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(7) Type 48: Mean on-premise sales, general for public premises outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.

(8) Mean taxable retail sales of packaged liquor stores (Type 21), 1972-75, per capita of persons aged 15+ on 1 April 1970.

(9) Mean public drunkenness arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.

(10) Misdemeanor drunken-driving arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.

(11) Felony drunken-driving arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.

(12) Mean total alcohol related arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.

(13) Mean cirrhosis mortality, 1970-74, per 100,000 persons aged 15+ on 1 April 1970.

(14) Population aged 15+ on 1 April 1970.

* Alpine, Calaveras, Colusa, Mariposa, Modoc, Sierra and Trinity counties were eliminated from the analysis.

mean (0.47). Imperial County possessed the highest misdemeanor drunken-driving arrest rate of all counties (41.8) while San Francisco County had the lowest (6.0). That San Francisco had the lowest rates for both misdemeanor and felony drunken-driving arrests is not surprising; a relatively larger proportion of the population in San Francisco uses public transportation than in other counties. In cirrhosis mortality, however, San Francisco assumed the lead (7.7 per 100,000 persons) and Inyo was second (5.9).

To test our hypotheses about bivariate relationships between variables, we obtained the correlations and partial correlations (controlling for urbanization and income) for the arrest rates, cirrhosis mortality rates, outlet rates and the MHDPS variables.

Arrest Rates, Cirrhosis Mortality Rates and the MHDPS Variables. Table 2 reports the correlations and partial correlations (controlling for urbanization and income) between the alcohol-related problems and the social indicator variables of the MHDPS. We found confirmation of the first hypothesis that social rank varies inversely with alcohol-related problems. Most of the social-rank variables are correlated with the arrest rates in the expected directions. Median income, for example, is correlated most strongly with public drunkenness arrest rates ($r = -.30$) and is weakest with both felony drunken-driving arrest and cirrhosis mortality rates ($r = -.08$),¹¹ while only the percentage of families in poverty is consistently and significantly related to arrest rates after controls for urbanization and income were made. However, although hypothesized to be inversely associated with cirrhosis mortality, none of the social rank variables was significantly related to cirrhosis death rates after controlling for urbanization and income.

We found very little support for our hypothesis that urbanization is positively related to alcohol-related problems. The only moderate correlations obtained were between the percentage of persons residing in places with populations of less than 2500 outside urban areas¹² and felony drunken-driving arrest rates ($r = .40$), and between the percentage of single-family detached dwelling units

¹¹ Rather than use asterisks to flag coefficients significant at the .05 level in the correlation tables, we have chosen to indicate significance by showing the minimum correlation coefficients required to achieve significance (one-tailed test) at the .05 level, for both zero-order and partial correlations. Coefficients required are listed in the footnotes to the tables.

¹² Donnelly (17) employed the same measure of urbanization, which is actually a measure of "ruralization."

- (6) Type 47. Mean on-premise sales, general for bona fide public eating place outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.
- (7) Type 48. Mean on-premise sales, general for public premises outlets, 1976-77, per 1000 persons aged 15+ on 1 April 1970.
- (8) Mean taxable retail sales of packaged liquor stores (Type 21), 1972-75, per capita of persons aged 15+ on 1 April 1970.
- (9) Mean public drunkenness arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.
- (10) Misdemeanor drunken-driving arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.
- (11) Felony drunken-driving arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.
- (12) Mean total alcohol-related arrests, 1972-75, per 1000 persons aged 15+ on 1 April 1970.
- (13) Mean cirrhosis mortality, 1970-74, per 100,000 persons aged 15+ on 1 April 1970.
- (14) Population aged 15+ on 1 April 1970.

* Alpine, Calaveras, Colusa, Mariposa, Modoc, Sierra and Trinity counties were eliminated from the analysis.

TABLE 2.—Correlations (and Partial Correlations Controlling for Urbanization and Income) between Alcohol-Problem Indicators and Mental Health Demographic Profile System Variables (N = 51)*

		Public Drunkenness Arrest Rates	Misdemeanor Drunken-Driving Arrest Rates	Felony Drunken- Driving Arrest Rates	Cirrhosis Mortality
Social Rank					
Median income of families and unrelated individuals	(MHH5C07)	-.30	-.21	-.08	-.08
% Families living below poverty line	(MHH5C08)	.52 (.50)	.44 (.45)	.26 (.32)	.10 (.06)
% Employed males aged 16 + : operative, service workers and laborers, including farmworkers	(MHH5C09)	.32 (.27)	.29 (.27)	.22 (.05)	.01 (-.08)
% Employed males aged 16 + : professional, technical and kindred workers and managers	(MHH5C10)	-.29 (-.20)	-.33 (-.30)	-.14 (.01)	.02 (.09)
Median years of school completed by persons aged 25 +	(MHH5C11)	-.32 (-.25)	-.54 (-.52)	-.28 (-.24)	-.04 (-.01)
Urbanization					
% Residents outside urban areas in towns of less than 2500 inhabitants	(MHH6C90)	-.05	-.01	.40	.03
% Single-family detached dwellings	(MHH5C19)	-.11 (-.12)	.18 (.21)	.21 (.01)	-.50 (-.60)
Population, 1 April 1970	(MHH5C01)	.00 (.02)	.10 (.14)	-.10 (.08)	.05 (.07)

Urbanization

% Residents outside urban areas in towns of less than 2500 inhabitants	(MHI6C90)	-.05	-.01	.40	.03
% Single-family detached dwellings	(MHI5C19)	-.11	.18	.21	-.50
Population, 1 April 1970	(MHI5C01)	(-.12)	(.21)	(.01)	(-.60)
		.00	.10	-.10	.05
		(.02)	(.14)	(.08)	(.07)

Race-Ethnicity

% Household population Black	(MHI5C12)	.25	.01	-.22	.33
% Population foreign-born or native of mixed parentage	(MHI5C14)	(.25)	(-.03)	(.05)	(.44)
% Population Spanish American	(MHI6C45)	.15	.20	-.18	.28
% Population other non-White	(MHI5C13)	(.13)	(.22)	(-.03)	(.34)
		.30	.44	-.05	.00
		(.24)	(.42)	(.09)	(-.01)
		.35	-.17	-.04	.65
		(.32)	(-.21)	(.05)	(.67)

Household and Family Structure

% Households of husband-wife families	(MHI5C15)	-.13	.17	.11	-.63
% Divorced or separated males	(MHI6C73)	(-.03)	(.26)	(-.02)	(-.68)
% Divorced or separated females	(MHI6C74)	.08	-.14	.18	.69
Number of children under 5 years per 1000 women aged 15-44 years (fertility ratio)	(MHI6C51)	(.10)	(-.13)	(.16)	(.69)
Ratio of males to females	(MHI6C50)	.12	-.09	-.23	.40
% Households female-headed	(MHI6C76)	(.14)	(-.13)	(.07)	(.57)
Number of persons aged < 18 per 1000 persons 18-64 (youth dependency ratio)	(MHI5C17)	.28	.38	-.01	-.29
Number of persons aged 65+ per 1000 persons aged 18-64 (aged dependency ratio)	(MHI5C18)	(.25)	(.36)	(.01)	(-.31)
Median age of household heads	(MHI5C16)	.11	.01	.49	-.06
		(.30)	(.08)	(.32)	(-.09)
		.20	-.14	-.36	.42
		(-.11)	(-.30)	(-.18)	(.55)
		.23	.46	.00	-.39
		(.23)	(.46)	(.05)	(-.40)
		-.13	-.04	-.03	.19
		(-.32)	(-.16)	(-.24)	(.18)
		-.12	-.01	.10	.25
		(-.24)	(-.08)	(-.19)	(.29)

TABLE 2.—continued

		Public Drunkness Arrest Rates	Misdemeanor Drunken-Driving Arrest Rates	Felony Drunken- Driving Arrest Rates	Cirrhosis Mortality
<i>Condition of Housing</i>					
% Occupied housing units with direct access, complete plumbing and kitchen facilities	(MHI5C22)	-.35 (-.38)	-.04 (-.02)	-.18 (-.03)	-.68 (-.73)
% Persons living in units with 1.01 or more persons per room	(MHI5C21)	.50 (.44)	.55 (.53)	.29 (.29)	.09 (.06)
<i>Familism</i>					
% Small households (1 person)	(MHI6C47)	.07 (-.01)	.21 (-.27)	-.06 (-.06)	.70 (.71)
% Large households (6+ persons)	(MHI6C48)	.32 (.30)	.55 (.55)	.07 (.17)	-.24 (-.25)
<i>Residential Instability</i>					
% Recent moves (1969-70)	(MHI5C23)	.13 (-.06)	-.04 (-.18)	.04 (.04)	-.15 (-.23)
<i>Women's Labor Force</i>					
% Females aged 16+ in labor force	(MHI6C22)	-.13 (-.01)	-.18 (-.12)	.03 (.24)	.19 (.29)
<i>Employment</i>					
% Unemployed aged 16+	(MHI6C16)	.20 (.16)	-.01 (-.08)	.22 (.06)	.03 (.00)

* Zero-order correlations significant at $p < .05$, 49 df, $r = .24$ or greater; and partial correlations (controlling for urbanization and income) significant at $p < .05$, 47 df, $r = .25$ or greater. (Both one-tailed t test.)

Women's Labor Force % Females aged 16+ in labor force	r	(N1116C22)	- .13 (- .01)	- .18 (- .12)	.03 (.24)	.19 (.29)
Employment % Unemployed aged 16+		(N1116C16)	.20 (.16)	-.01 (-.08)	.22 (.06)	.03 (.00)

* Zero-order correlations significant at $p < .05$, 49 df, $r = .24$ or greater; and partial correlations (controlling for urbanization and income) significant at $p < .05$, 47 df, $r = .25$ or greater. (Both one-tailed t test.)

and cirrhosis mortality ($r = -.60$, after controls). The former correlation suggests that felony drunken-driving arrests are relatively more common in more rural areas, perhaps because of increased speeds by drivers and poorer road conditions.

For our third hypothesis, that minority race-ethnicity is positively associated with alcohol-related problems, we found moderate support. Without controls for urbanization and income, public drunkenness arrest rates were significantly correlated with 3 of the 4 variables. When urbanization and income were controlled, the percentage of household population Black and the percentage of population other non-White remained significantly correlated ($r = .25$ and $r = .32$, respectively).¹³

While felony drunken-driving arrest rates had no correlations significantly different from zero with the race-ethnicity variables, misdemeanor drunken-driving arrests did have a correlation of $r = .42$, with the percentage Spanish American, after controlling for urbanization and income, indicating a moderate relationship.

Cirrhosis mortality was significantly related to 3 of 4 race-ethnicity variables, with all of the zero-order correlations increasing after removing the effects of urbanization and income. This indicates the moderate degree to which race and ethnicity are positively related to cirrhosis mortality. Only the percentage of population Spanish American remained unrelated to cirrhosis death rates.

The fourth hypothesis that we considered is related to the degree to which individuals are integrated into relatively more traditional family-oriented settings. The socioculturalists have argued that the greater the degree of attachment to traditional family structures, the more likely will norms of moderation with respect to drinking behavior prevail. Accordingly, a series of household composition and family structure variables, as well as indicators of familism, were tested.

The household composition and family structure variables, for the most part, were minimally related to arrest rates, although there were a few exceptions, after controlling for urbanization and income. Public drunkenness arrest rates correlated positively with the fertility ratio even after controls, negatively with the aged dependency ratio, becoming stronger after controls ($r = -.32$),

¹³ In 1970, the other non-White population was composed of American Indians, Asian and Pacific Islanders and a residual designation of "other." Together, these groups accounted for approximately 3% of the population in California.

and positively with the sex ratio, becoming significant ($r = .30$) after controls. Misdemeanor drunken-driving arrest rates, after controls, were just barely significant with the percentage of households composed of husband-wife families ($r = .26$); however, the correlation is not in the expected direction, if one accepts the argument that the proportion of husband-wife households is an indicator of social stability. Misdemeanor drunken-driving arrests were positively and significantly related to the fertility ratio and the youth dependency ratio even after controls. Only the sex ratio remained correlated with felony drunken-driving arrests after controls ($r = .32$).

The familism indicators were correlated in unexpected ways. If, as is argued, familism, as represented by the percentages of small households (1 person) and large households (6+ persons), represents the degree to which social areas are family versus nonfamily oriented, then to that extent it would be expected that alcohol-related social problems should decline. With the three types of alcohol-related arrests this is not the case. The percentages of small households and large households were correlated significantly different from zero ($p < .05$, two-tailed test) with misdemeanor drunken driving ($r = -.27$ and $r = .55$, respectively) after controlling for urbanization and income. Furthermore, large households were correlated with public drunkenness arrests after controls ($r = .30$), but they were unrelated to felony drunken driving; small households were unrelated to either public drunkenness arrest rates or felony drunken driving.

It is with cirrhosis mortality that support for the hypothesis was found. Only 2 of the 9 correlations of the household variables with cirrhosis mortality were insignificant (sex ratio, aged dependency ratio), while most of the significant correlations were significant at the $p < .01$ level.

Although median age of household heads was correlated with cirrhosis ($r = .29$) after controls, suggesting the connection between age and the relatively long period for the development of the disease, cirrhosis mortality rates were not statistically related to the aged dependency ratio.

The most probable explanation of the significant positive relationship between cirrhosis mortality ($r = .25$) and median age of household heads and the positive but insignificant relationship between cirrhosis mortality and the aged dependency ratio ($r = .19$) is that cirrhosis mortality rates are curvilinear with respect to

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age. That is, the probability of dying of liver cirrhosis increases with age until the early sixties, after which it decreases very gradually. Thus, to the extent that median age of household heads varies across the 51 counties studied, it should be positively associated with cirrhosis mortality, as in fact it is in these data. The smaller but positive association between the aged dependency ratio and cirrhosis mortality reflects in part that the aged dependency ratio is constructed as a ratio of the number of persons aged 65 and over per 1000 persons aged 18-64. Therefore, to the extent that this ratio varies across the 51 counties, it might be expected to be either slightly positively correlated, uncorrelated, or negatively correlated with cirrhosis mortality. In these data the relationship between the aged dependency ratio and cirrhosis mortality is positive and insignificant.¹⁴

The familism indicators provided strong evidence that cirrhosis mortality is related to traditional-nontraditional families. The percentage of large households is significantly inversely related to cirrhosis mortality ($r = -.25$) after controls and is positively related to single-person households ($r = .71$, $p < .001$) after controls.

Other MHDPS indicators provide further evidence that social area characteristics are related to alcohol-related problems in the expected ways, given previous research findings, although in some relationships the correlations are not significantly different from zero. Public drunkenness arrest and misdemeanor drunken-driving arrest rates were significantly related to crowding (percentage of persons living in units with 1.01 or more persons per room) and large households (6+ persons) both before and after controls, and felony drunken-driving arrest rates were related to crowding ($r = .29$) with controls, but not large households. Although positive, women's labor force participation and felony drunken-driving arrest rates were not significantly related. Although expected to be related to the alcohol problems, residential instability was uncorrelated with any of them, as was the percentage unemployed.

Per Capita Sales, Outlet Rates and Alcohol-Related Problems.
Our fifth hypothesis concerned the relationships between alcoholic

¹⁴ Another factor which is probably influencing the correlations in these data is the cirrhosis mortality rate itself. The rate is crude since it is constructed by taking actual cirrhosis deaths and dividing them by the population aged 15+ at the time of the 1970 Census. Thus, the base population is composed of both individuals increasingly at risk (18-64) and those decreasingly at risk (65+). We are grateful to Professor Georges Sabagh for pointing this out.

beverage outlets and taxable retail sales and alcohol-related problems. Table 3 shows the correlations and partial correlations (controlling for urbanization and income) obtained between the outlet rates, per capita sales and the 4 alcohol problems. Per capita sales were more strongly related to several outlet types other than the one (Type 21, off-premise, general) on which they are based, even after controls. Furthermore, sales were related only to felony drunken-driving arrest rates after controls ($r = .28$).

When we initially studied the correlations between outlet types divided only by whether they were on- or off-premise outlets, we failed to detect differential relationships between the types of outlets and both alcohol-related problems and MHDPS variables. When we examined rates for the specific types of outlets, we found that they were indeed differentially related to both problems and social area characteristics. One very important result that emerged is that Type 40 outlets (beer bars) are implicated in both public drunkenness arrests and misdemeanor drunken-driving arrests, while several types of outlets (Types 20, 21, 40, 41, 47) are related to felony drunken-driving arrests, even after controls. However, it was Type 21 (off-premise, general) and Type 48 (on-premise, general) outlets that were most strongly correlated with cirrhosis mortality after controls ($r = .32$ and $r = .42$, respectively).

Per Capita Sales, Outlet Rates and MHDPS Variables. Our last hypothesis concerned the relationship between social area characteristics and sales and outlets. It is hypothesized that there will be differential relationships between the various outlet types, sales and social area characteristics. Outlets such as Type 41 (on-premise, beer and wine, for bona fide eating places) and Type 47 (on-premise, general, for bona fide eating places) would be expected to be positively related to social class, inversely related to minority race-ethnicity, inversely related to household composition, family structure and familism, positively related to urbanization, and inversely related to unemployment. Outlets such as Type 21 (off-premise, general), Type 40 (beer only, on-premise), Type 42 (beer and wine, public premises), and Type 48 (general, public premises) might be expected to be positively related to social class, minority race-ethnicity, nontraditional household composition, low familism and other variables indicating social disorganization. Furthermore, sales might be expected to be positively related to social rank and minority race-ethnicity, and inversely related to household composition, family structure and familism, and unemployment.

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TABLE 3.—Intercorrelations (and Partial Correlations Controlling for Urbanization and Income) among the Alcohol-Problem Indicators (N = 51)*

	Type 20	Type 21	Type 40	Type 41	Type 42	Type 47	Type 48	Per Capita Sales	Public Drunkness	Misdemeanor Drunk Driving	Felony Drunk Driving
Type 21	.81 (.70)										
Type 40	.84 (.75)	.60 (.44)									
Type 41	.79 (.75)	.78 (.68)	.58 (.49)								
Type 42	-.03 (-.39)	-.14 (-.40)	.07 (-.22)	-.19 (-.39)							
Type 47	.83 (.74)	.91 (.84)	.62 (.40)	.91 (.88)	-.16 (-.43)						
Type 48	.36 (-.11)	.55 (.32)	.26 (-.16)	.11 (-.25)	.10 (-.12)	.40 (.09)					
Per capita sales	.35 (.54)	.29 (.38)	.26 (.38)	.58 (.68)	.17 (-.15)	.37 (.48)	-.31 (-.35)				
Public drunkness arrest rates	.10 (.22)	.05 (.19)	.27 (.35)	-.03 (.08)	.03 (-.03)	-.01 (.13)	.07 (.12)	-.04 (-.01)			
Misdemeanor drunken-driving arrest rates	.10 (.16)	-.14 (-.12)	.37 (.45)	-.11 (-.08)	.01 (-.03)	-.16 (-.14)	-.13 (-.17)	.02 (.04)	.13 (.06)		
Felony drunken-driving arrest rates	.72 (.68)	.54 (.42)	.59 (.50)	.59 (.50)	-.19 (-.36)	.58 (.47)	.13 (-.14)	.24 (.28)	.12 (.18)	.28 (.32)	
Cirrhosis mortality rate	.05 (.05)	.25 (.32)	.11 (.11)	.10 (.12)	-.06 (-.09)	.12 (.15)	.36 (.42)	.18 (.19)	.33 (.33)	-.09 (-.10)	-.01 (-.02)

* Zero-order correlations significant at $p < .05$, 49 df, $r = .24$ or greater; and partial correlations (controlling for urbanization and income) significant at $p < .05$, 47 df, $r = .25$ or greater. (One-tailed t -test.)

Table 4 reports the correlations and partial correlations (again, controlling for urbanization and income) between these variables. Although none of the social-rank variables correlated significantly with sales (even after controls) they were, in general, in the expected direction. And, although all are positive with respect to the minority race-ethnicity variables, sales is significantly correlated only with the percentage of population other non-White, after controls ($r = .28$). Three of the nine household composition variables correlate significantly with per capita sales after controls (percentage of husband-wife families, percentage of divorced men and percentage of divorced women). As expected, per capita sales is inversely related to unemployment and positively related to female labor force participation, suggesting that those areas where relatively more women are engaged in the workplace are areas where packaged liquor store sales per capita are relatively greater. Per capita sales is, also as expected, inversely related to familism (percentages of large and small households).

It is with the outlet rates that we find several important relationships. As hypothesized, restaurants with on-premise licenses (Types 41 and 47) are positively related to social rank and either inversely or insignificantly related to minority race-ethnicity. Only the percentage of population other non-White shows a significant positive relationship with both Type 41 and Type 47 outlets after controls ($r = .27$ and $r = .25$, respectively). As predicted by the hypothesis, both types of outlets are inversely related to unemployment, but positively and strongly related to women's labor force participation (Type 41, $r = .66$; Type 47, $r = .56$, after controls). Furthermore, the correlations of the household composition and family structure variables, as well as the familism variables, support the idea that social areas with traditional family structures have relatively fewer Type 41 and Type 47 outlets.

One point should be noted with virtually all of the outlet rates. Contrary to previous findings (7, 10) we found strong inverse relationships with urbanization as measured by our percent-rural variable, and weak inverse relationships with urbanization as measured by the total population in each county on 1 April 1970. This suggests that caution should be exercised when conducting studies at the aggregate level; outlet rates will not always vary directly with urbanization.

The two public premises outlets (Types 42 and 48) are unrelated to the social rank variables after controls. Although correlated with

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TABLE 4—Correlations (and Partial Correlations Controlling for Urbanization and Income) between Types of Alcoholic Beverage Outlets, per Capita Sales and Mental Health Demographic Profile System Variables

	Type 20	Type 21	Type 40	Type 41	Type 42	Type 47	Type 48	Per Capita Sales
Social Rank								
Median income of families and unrelated individuals	(MHH5C07)	-.19	.01	-.32	.07	.05	-.19	.11
% Families living below poverty line	(MHH5C08)	.33	-.02	.55	-.04	-.05	.05	.01
% Employed males aged 16+; operative, service workers and laborers, including farmworkers	(MHH5C09)	.32	.08	.37	.09	-.02	.29	-.17
% Employed males aged 16+; professional, technical and kindred workers and managers	(MHH5C10)	-.19	.02	-.31	.21	.14	-.23	.22
Median years school completed by persons aged 25+	(MHH5C11)	-.23	.09	-.42	.17	.12	-.10	.10
		(-.09)	(.24)	(-.31)	(.25)	(.26)	(.08)	(.05)
Urbanization								
% Residents outside urban areas in towns of less than 2500 inhabitants	(MHH6C90)	.71	.60	.61	.48	.32	.60	-.04
% Single-family detached dwellings	(MHH5C19)	.24	-.04	.30	-.12	.30	-.01	-.35
Total population, 1 April 1970		(-.21)	(-.51)	(-.03)	(-.49)	(.16)	(-.47)	(-.39)
		-.27	-.16	-.26	-.14	-.13	-.25	.17
		(.03)	(.10)	(.02)	(.05)	(.03)	(.11)	(.16)

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TABLE 4—Correlations (and Partial Correlations Controlling for Urbanization and Income) between Types of Alcoholic Beverage Outlets, per Capita Sales and Mental Health Demographic Profile System Variables

		Type 20	Type 21	Type 40	Type 41	Type 42	Type 47	Type 48	Per Capita Sales
<i>Social Rank</i>									
Median income of families and unrelated individuals									
	(MHI5C07)	-.19	.01	-.32	.07	-.29	.05	-.19	.11
% Families living below poverty line									
	(MHI5C08)	.33 (.35)	-.02 (-.09)	.55 (.56)	-.04 (-.01)	.19 (-.07)	-.05 (-.07)	.05 (-.23)	.01 (.15)
% Employed males aged 16 + : operative, service workers and laborers, including farmworkers									
	(MHI5C09)	.32 (-.07)	.08 (-.24)	.37 (-.02)	.09 (-.39)	.21 (-.07)	-.02 (-.39)	.29 (-.03)	-.17 (-.15)
% Employed males aged 16 + : professional, technical and kindred workers and managers									
	(MHI5C10)	-.19 (.18)	.02 (.29)	-.31 (.02)	.21 (.46)	-.32 (-.12)	.14 (.47)	-.23 (.03)	.22 (.20)
Median years school completed by persons aged 25 +									
	(MHI5C11)	-.23 (-.09)	.09 (.24)	-.42 (-.31)	.17 (.28)	-.03 (.15)	.12 (.26)	-.10 (.08)	.10 (.05)
<i>Urbanization</i>									
% Residents outside urban areas in towns of less than 2500 inhabitants									
	(MHI6C00)	.71	.60	.61	.48	.32	.60	.59	-.04
% Single-family detached dwellings									
	(MHI5C19)	.24 (-.21)	-.04 (-.51)	.30 (-.03)	-.12 (-.49)	.30 (.16)	-.01 (-.47)	.17 (-.19)	-.35 (-.39)
Total population, 1 April 1970									
	(MHI5C01)	-.27 (.03)	-.16 (.10)	-.26 (.02)	-.14 (.05)	-.13 (.03)	-.15 (.11)	-.25 (.00)	.17 (.16)
continued									

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TABLE 4.—continued

		Type 20	Type 21	Type 40	Type 41	Type 42	Type 47	Type 48	Per Capita Sales
<i>Race-Ethnicity</i>									
% Household population		-.40	-.22	-.30	-.29	-.24	-.29	-.17	.08
Black	(MHI5C12)	(.09)	(.29)	(.12)	(.07)	(.09)	(.20)	(.31)	(.09)
% Population		-.34	-.27	-.18	-.16	-.38	-.23	-.16	.19
foreign-born or native									
of mixed parentage	(MHI5C14)	(-.03)	(.06)	(.17)	(.12)	(-.28)	(.13)	(.19)	(.20)
% Population		-.19	-.37	.12	-.30	-.24	-.32	-.24	.06
Spanish American	(MHI6C45)	(.04)	(-.20)	(.37)	(-.14)	(-.23)	(-.12)	(-.09)	(.08)
% Population		-.01	.15	-.09	.09	-.22	.03	.11	.26
other non-White	(MHI5C13)	(.19)	(.39)	(.01)	(.27)	(-.21)	(.25)	(.29)	(.28)
<i>Household and Family Structure</i>									
% Households of		.16	-.07	.21	-.11	.18	-.05	-.06	-.28
husband-wife families	(MHI5C15)	(-.09)	(-.42)	(.09)	(-.41)	(.16)	(-.41)	(-.31)	(-.32)
% Divorced or		.21	.38	.10	.37	-.13	.31	.19	.27
separated males	(MHI6C73)	(.21)	(.41)	(.06)	(.39)	(-.16)	(.32)	(.18)	(.28)
% Divorced or		-.40	-.27	-.42	-.09	-.21	-.24	-.39	.28
separated females	(MHI6C74)	(.16)	(.23)	(.00)	(.38)	(.02)	(.30)	(.03)	(.34)
Number of children									
under 5 years per 1000									
women aged 15-44		-.04	-.28	.18	-.32	-.02	-.27	-.14	-.12
(fertility ratio)	(MHI6C51)	(-.04)	(-.32)	(.21)	(-.34)	(-.05)	(-.29)	(-.17)	(-.11)
Ratio of males to		.65	.66	.56	.48	.01	.55	.34	-.03
females	(MHI6C50)	(.35)	(.42)	(.33)	(.20)	(-.24)	(.21)	(-.06)	(-.03)
% Households		-.51	-.37	-.47	-.26	-.11	-.33	-.17	.15
female-headed	(MHI6C76)	(-.21)	(.01)	(-.31)	(.14)	(.00)	(.15)	(.25)	(.20)
Number of persons aged under									
18 per 1000 persons 18-64		-.09	-.36	.17	-.36	.02	-.36	-.31	-.19
(youth dependency ratio)	(MHI5C17)	(-.02)	(-.37)	(.28)	(-.35)	(.04)	(-.38)	(-.32)	(-.19)
Number of persons aged									
65+ per 1000 persons									
aged 18-64 (aged		.17	.10	.15	.01	.43	.09	.40	.02
dependency ratio)	(MHI5C18)	(-.21)	(-.12)	(-.27)	(-.15)	(.28)	(-.10)	(.22)	(.10)

Females	(MHI6C50)	(.35)	(.42)	(.33)	(.20)	(-.24)	(.21)	(-.06)	(-.03)
% Households		-.51	-.37	-.47	-.26	-.11	-.33	-.17	.13
female-headed	(MHI6C76)	(-.21)	(.01)	(-.31)	(.14)	(.00)	(.15)	(.25)	(.20)
Number of persons aged under 18 per 1000 persons 18-64 (youth dependency ratio)	(MHI5C17)	-.09	-.36	.17	-.36	.02	-.36	-.31	-.19
		(-.02)	(-.37)	(.28)	(-.35)	(.04)	(-.38)	(-.32)	(-.19)
Number of persons aged 65 + per 1000 persons aged 18-64 (aged dependency ratio)	(MHI5C18)	.17	.10	.15	.01	.43	.09	.40	.02
		(-.21)	(-.12)	(-.27)	(-.15)	(.28)	(-.10)	(.22)	(.10)

Median age of household heads	(MHI5C16)	.29	.23	.25	.06	.42	.19	.54	-.06
		(-.26)	(-.15)	(-.27)	(-.08)	(.25)	(-.22)	(.29)	(-.01)

Condition of Housing

% Occupied housing units with direct access, complete plumbing and kitchen facilities	(MHI5C22)	-.38	-.50	-.45	-.33	-.04	-.38	-.38	-.21
		(-.15)	(-.40)	(-.26)	(-.22)	(.13)	(-.24)	(-.38)	(-.21)

% Persons living in units with 1.01 or more persons per room	(MHI5C21)	.30	.01	.55	.01	-.03	-.02	-.06	.13
		(.29)	(-.02)	(.55)	(.02)	(-.21)	(-.04)	(-.23)	(.20)

Familism

% Small households (1 person)	(MHI6C47)	.03	.29	-.06	.21	-.02	.22	.34	.24
		(.03)	(.41)	(-.14)	(.29)	(-.08)	(.34)	(.41)	(.27)

% Large households (6 + persons)	(MHI6C48)	-.07	-.31	.23	-.28	-.14	-.31	-.34	-.05
		(.11)	(-.22)	(.45)	(-.19)	(-.11)	(-.22)	(-.29)	(-.04)

Residential Instability

% Recent moves (1969-70)	(MHI5C23)	.18	.04	.19	.18	.06	.11	-.13	.03
		(.23)	(.14)	(.11)	(.36)	(-.09)	(.30)	(-.27)	(.10)

Women's Labor Force

% Females aged 16 + in labor force	(MHI6C22)	.03	.14	-.21	.35	-.56	.20	-.29	.43
		(.45)	(.47)	(.16)	(.66)	(-.46)	(.56)	(-.07)	(.46)

Employment

% Unemployed aged 16 +	(MHI6C16)	.26	.24	.22	-.06	.27	.09	.48	-.43
		(-.12)	(.01)	(-.18)	(-.33)	(.08)	(-.20)	(.29)	(-.46)

the social rank variables in the expected directions without controls, beer bars (Type 40), after controls, have a significant positive relationship to the percentage of families in poverty ($r = .56$).

Type 48 outlets (all beverages) are related to the percentage of population Black and other non-White after controls, while beer bars (Type 40) are related only to the percentage Spanish American, after controls ($r = .37$). The household composition variables show no consistent patterns with Types 42 and 48 outlets after controls, although the percentage of female-headed households is correlated positively with Type 48 outlets ($r = .25$). Beer bars are positively related to the sex ratio and the youth dependency ratio but they are inversely related to the percentage of female-headed households and the aged dependency ratio. This suggests that beer bars are primarily frequented by men and the relatively young, the correlation with the median age of household heads being $r = -.27$ after controls.

As expected, the familism variables are inversely related to Type 48 outlets, while their relationships with Type 42 outlets are not significant. And, of the other variables, only the percentage of females aged 16 + in the labor force is inversely related to Type 42 outlets, while the percentage unemployed is positively related to Type 48 outlets.

Beer bars (Type 40) are unrelated to social areas where there are larger numbers of single persons, but are moderately related to areas where there are relatively large households ($r = .45$). Beer bars are unrelated to residential instability, women's labor force participation, and unemployment both with and without controls for urbanization and income. However, just as beer bars are correlated strongly with the percentage of families in poverty ($r = .56$), they also correlate strongly with crowding ($r = .55$). All in all, these results demonstrate that on-premise outlet rates are not uniformly distributed across the 51 counties and are differentially related to the MHDPS social area characteristics.

The correlations of off-premise outlet rates present a somewhat different picture. The only significant correlations with the social rank variables occur between the percentage of families in poverty and Type 20 (off-premise, beer and wine only), and the percentage of high-occupational-status men and Type 21 (off-premise, general). The race-ethnicity variables are uncorrelated with Type 20 outlets, while only the percentage of household population Black and the percentage of population other non-White are significantly correlated with Type 21 outlets ($r = .29$ and $r = .39$, respectively).

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With respect to the household composition variables, Type 20 outlets are positively related to the sex ratio ($r = .35$) and inversely correlated with median age of household head ($r = -.26$) after controls. Type 21 (off-premise, general) outlets are inversely related to the percentage of husband-wife families ($r = -.42$) and positively related to the percentage of divorced or separated men ($r = .41$) and the sex ratio ($r = .42$) after controls.

The familism variables correlated inversely, as expected, with Types 20 and 21 outlets, although only the correlation between Type 21 outlets and single-person households remained significant after controls ($r = .41$), while among the rest of the MHDPS variables, only the percentage of women 16+ in the labor force correlated positively with both types after controls ($r = .45$ and $r = .47$, respectively).

Multiple Regression Models. We employed simultaneous multiple regression in a series of models to examine the net effects of both the sociodemographic and availability variables on the dependent variables of interest. Table 5 reports the results of several of these regressions.¹⁵

Public drunkenness arrest rates were first regressed on the percentage of families in poverty and the percentage of population Black (column 1). Both beta coefficients were significant at the .05 level; however, the percentage of families in poverty showed the strongest effect (beta = .526, $p < .001$). The adjusted R^2 in this model accounted for 31.2% of the variance in the dependent variable and overall the equation was significant ($F = 12.332$, $p < .001$).

The addition of Type 40 (beer bars) outlets into the model lowered the beta coefficient for the percentage in poverty (beta = .474, column 2). Both the percentage Black and Type 40 outlets are not significant in this model, while the percentage in poverty remained significant at the .01 level. The overall equation remains significant ($F = 8.259$, $p < .001$) and the adjusted R^2 accounts for slightly less of the variance explained in public drunkenness arrests (30.3%).¹⁶

The third regression reported shows the relationship between public drunkenness arrest rates and the two sociodemographic

¹⁵ Since we were constrained by the small number of cases, no regression possesses more than four predictor variables.

¹⁶ Type 40 outlets correlated with the percentage of families in poverty, $r = .55$.

TABLE 5.—Standardized Regression Coefficients (and Unstandardized Regression Coefficients) for Several Predictor Variables of Public Drunkenness, Misdemeanor Drunken-Driving, and Felony Drunken-Driving Arrest Rates, and Cirrhosis Mortality Rates) (N = 51)

Sociodemographic Characteristics	Public Drunkenness Arrest Rates			Misdemeanor Drunken-Driving Arrest Rates		Felony Drunken-Driving Arrest Rates			Cirrhosis Mortality Rates	
	(1)	(2)	(3)	(1)	(2)	(1)	(2)	(3)	(1)	(2)
% Families living below poverty line (MHI5C08)	.526† (2.079)	.474† (1.871)	.529† (2.089)	.315* (.617)	.182 (.387)	.308* (.017)	.171* (.009)		.232* (.083)	
% Household population Black (MHI5D12)	.253* (.827)	.281 (.919)	.279* (.913)			-.185 (-.008)	.010 (.0005)			
% Household population Spanish American (MHI6D45)				.313* (.208)	.338* (.225)	-.122 (-.002)	-.014 (-.0002)			
% Small Households (1 person) (MHI6C47)									.843† (.236)	.874† (.245)
Aged dependency ratio (MHI5C18)									-.270* (-.047)	-.245* (-.042)
% Population outside urban areas in towns of less than 2500 inhabitants (MHI6C90)							.358* (.002)	-.054 (-.0003)		
Outlet Availability										
Type 21 (off-premise, general)			.121 (1.564)					.313* (.056)		-.146 (-.172)
Type 40 (on-premise, beer only)		.097 (3.106)			.227 (3.931)			.438† (.196)		.285* (.832)
Constant	-9.004	-9.137	-11.097	5.911	6.085	.161	.109	.106	-1.442	-1.172
R ²	.3394	.3452	.3534	.2740	.3097	.1263	.1872	.4082	.5652	.5636
Adjusted R ²	.3119	.3034	.3122	.2438	.2656	.0706	.1165	.3704	.5374	.5256
F	12.332†	8.259†	8.564†	9.058†	7.028†	2.266	2.648*	10.804†	20.364†	14.850†

*P < .05 †P < .01 ‡P < .001.

The third set of regressions is reported for felony drunken-driving arrests. The first regression shows the coefficients for the percentage of families living in poverty, the percentage Black and the

percentage Spanish American. Only the percentage in poverty is significant ($\beta = .308$, $p < .05$), and the model explains only 7.1% of the variance in felony drunken-driving arrest rates. Since these rates were moderately correlated with our measure of urbanization ($r = .40$), we regressed felony arrest rates on the same three variables and the percentage of population outside metropolitan areas (column 2). Both "ruralization" and the percentage of families living in poverty are significant ($\beta = .358$ and $.171$, respectively).

When we removed the percentages Black and Spanish American from the model (both having virtually no effect on felony arrests) and added the availability variables (Types 21 and 40),¹⁷ the percentage of families living in poverty was unrelated to felony arrests.

In the final regression (column 3) we retained the percentage of population outside metropolitan areas with the two availability indicators. Both availability variables remained significant at the .05 level net of urbanization, while the urbanization coefficient became insignificant. The overall model for the last regression is significant ($F = 10.804$, $p < .001$) and the adjusted R^2 shows substantial improvement over the first two regressions (adjusted $R^2 = .37$).

With the last dependent variable, cirrhosis mortality rates, we considered as predictors in the first regression (column 1) the percentage of families in poverty, the percentage of one-person households and the aged dependency ratio. It is clear that the percentage of one-person households is the single best predictor of cirrhosis mortality ($\beta = .843$, $p < .001$) while the percentage of families living in poverty is significant at the .05 level ($\beta = .232$). The aged dependency ratio, although correlated with the percentage of one-person households ($r = .46$), was entered into the model to adjust for differences in distribution of the elderly across counties. Given the zero-order correlation of .18 between the aged dependency ratio and cirrhosis mortality, the negative regression coefficient was unexpected ($\beta = -.270$, $p < .05$).

When the two availability indicators were entered into the model the coefficient for the percentage of families living in poverty was not significant (regression not reported). In the final regression

¹⁷ We do not report these regressions.

(column 2), the percentage of one-person households remains an important predictor ($\beta = .874, p < .001$) of cirrhosis mortality, both the dependency ratio and Type 40 outlets are significant at the .05 level, and the adjusted R^2 is slightly less (.525) than that in the model in column 1. Again, given the correlation of $r = .25$ between cirrhosis mortality and Type 21 outlets, the negative coefficient ($\beta = -.146$) was unexpected. Most of the unexpected negative coefficients result from a moderate degree of multicollinearity between the independent variables and represent a good example of net suppression under the condition that all zero-order correlations are positive (27).

SUMMARY AND CONCLUSIONS

Following Donnelly (17), we employed the MHDPS in the analysis of alcohol availability, packaged liquor store sales and alcohol-related problems in California. In general, our results substantially support the findings of Donnelly and confirm the usefulness of a census-based system of sociodemographic information in analyses of alcohol-related problems. However, there are several differences in the findings for California and Pennsylvania. A brief review of the results in the two studies is informative.

Whereas Donnelly found that increases in per capita sales are positively correlated with alcohol-related problems, such as total alcohol-related arrests and cirrhosis mortality, our findings indicate that per capita sales of packaged liquor stores (an admittedly weak measure) is not related to public drunkenness arrest, misdemeanor drunken-driving arrest and cirrhosis mortality rates, but is correlated with felony drunken-driving arrest rates, after controlling for urbanization and income.

Although Donnelly found contradicting evidence of a positive relationship between alcohol-related arrest rates and social rank variables, we find confirmatory evidence for the hypothesized inverse relationships (4-6). Most of our social rank variables correlate with the arrest rates in the expected directions, although only the percentage of families living in poverty and median education are consistently and significantly related to arrest rates after controlling for urbanization and income. And, although hypothesized to be negatively associated with cirrhosis mortality, none of the correlations with the social rank variables was significantly different from zero. This is in accord with Donnelly's findings, even after controlling for urbanization and income.

Whereas Donnelly found strong relationships between minority race-ethnicity and his problem measures, we found only a few moderate correlations with specific types of arrests. Three of our 4 minority race-ethnicity variables are correlated significantly with cirrhosis mortality, while only 2 of Donnelly's correlations are significant. Interestingly, in both sets of correlations, the percentage Spanish American is unrelated to cirrhosis mortality.

In both Donnelly's data and our own, there is support for the hypothesized inverse relationship between relatively more traditional family structures and alcohol-related arrests, although in our correlations the relationships are modest. We found, as did Donnelly, relatively strong zero-order correlations between cirrhosis mortality and several household composition and family structure variables, although there are differences, e.g., Donnelly's low correlations between cirrhosis and male and female divorce and our moderately strong correlations. And, though Donnelly did not include familism variables, we found a strong relationship between the percentage of households with one person and cirrhosis mortality ($r = .70$), confirming findings made by Gove and Hughes (28) for 389 American cities.¹⁸

Donnelly did not investigate on- and off-premise outlet rates and therefore we have no information with which to make direct comparisons on the MHDPS variables at the county level in Pennsylvania. However, as mentioned above, several researchers (7-10) have studied the effects of availability on consumption and cirrhosis mortality at the state aggregate level, employing different measures of availability, and statistical controls for income, urbanism and restrictions on outlets by population. These studies have provided conflicting results. For example, Smart (7), using a composite measure of availability, found that income and urbanism were more closely associated with consumption and alcoholism than was availability. Parker et al. (9) re-examined Smart's data, but used on- and off-premise outlet rates as their indicator of availability and found strong effects of outlet rates on consumption and cirrhosis mortality rates in states without controls on the number of outlets per unit of population. Harford et al. (10), restricting the analysis to on-premise outlets per 100,000 persons, found that on-premise rates were significantly related to alcoholism rates in 39 jurisdic-

¹⁸ Gove and Hughes (28), employing ecological data, found that living alone was strongly correlated ($r = .64$) with and was, by far, the best predictor of cirrhosis mortality.

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tions. The authors concluded that, although rates of on-premise outlets and urbanization contribute to cirrhosis mortality rates when per capita consumption and income are controlled, the limitations of the overall small sample size and the considerable heterogeneity of state jurisdictions made firm conclusions about the relationships between the variables premature. They called for further study of the variables at the Standard Metropolitan Statistical Area or county level and suggested that a comprehensive model of alcohol availability needed to be developed.¹⁹

In this study we employed five types of on-premise and two types of off-premise outlets as indicators of physical availability, accounting for 90% of all alcoholic beverage outlets in the 51 counties studied. By studying the relationships between social area characteristics, outlet types and alcohol problems, we found several moderate relationships that were masked when the data were analyzed using only on- and off-premise outlet rates. Type 40 outlets (beer bars) are moderately positively correlated with the three types of arrests, even after controlling for urbanization and income, but are minimally correlated with cirrhosis mortality, while Type 21 (off-premise, general) and Type 48 (on-premise, general) outlets were related moderately to cirrhosis mortality.

Furthermore, our analysis revealed important relationships between social area characteristics and specific outlet types. For example, beer bars (Type 40) showed moderate relationships with poverty, crowding and the Spanish American population while off-premise general outlets (Type 21) showed significant positive correlations with the percentage Black, the percentage other non-White and several indicators of social disorganization and disruption of traditional families.

These results suggest that outlet rates (at least in the state of California) are related in important ways to alcohol problems. They lend support to the findings of Parker et al. (9) and Harford et al. (10) regarding the effects of on- and off-premise outlets on liver cirrhosis mortality and, furthermore, they show that specific outlet types are implicated differentially in alcohol-related problems.²⁰

Our multiple regression results showed that physical availability of specific types contributed significantly and positively to the

¹⁹ Smart's study (8) used the formulation of a wider conception of availability developed by Medicine in the Public Interest (21).

²⁰ Neither Smart (7), Parker et al. (9) nor Harford et al. (10) examined the impact of outlets on arrest rates.

explained variance in felony drunken-driving arrest rates and cirrhosis mortality. In the other two dependent variables—public drunkenness arrests and misdemeanor drunken-driving arrests—the two availability variables, although not contributing significant coefficients, did reveal important B values net of the selected sociodemographic variables.²¹ Since we were primarily concerned with an overall description of the data, we did not deal with a number of problems related to technical issues in multiple regression (29). For example, it should be kept in mind that there are statistical outliers in these data.²²

All in all, we found the MHDPS to be quite valuable in ecological research on alcohol-related problems. We found support for a number of hypotheses recently offered in the literature on social factors influencing alcohol problems. However, some considerations and adjustments need to be made when analyzing data of this type. First, aggregate data analysis assumes that the units of analysis are of a "comparable" nature (30). One would probably not want to use, as units of analysis, all states in the United States and all counties in California in the same analysis. Second, inspection for "outliers" must be made; we found that several of our alcohol indicators had outliers for one or another of the counties. We surmise that the very small counties are outliers because of small populations. And, third, although we did not present any regression analyses of the outlet rates on the MHDPS variables, previous analyses of these and other data suggest that tourism is a factor influencing consumption and on-premise availability (13).

Furthermore, as Donnelly pointed out, differential enforcement patterns, and that arrests represent number of arrests and not the number of people arrested, also can bias the data in certain ways. Also, there is evidence that cirrhosis mortality rates for small areas, say with populations under 300,000, may be biased (31). Yet we must conclude that a social area-epidemiological system of information, such as the MHDPS first employed by Donnelly, is invaluable in determining the relative influences of social area profile characteristics and outlet availability on alcohol problems. Further

²¹ There were problems of both multicollinearity and some heteroscedasticity in virtually all of the models, to which we did not fully attend.

²² Neither Smart (7) nor Parker et al. (9) considered outliers in their data and Harford et al. (10) only considered the District of Columbia. Donnelly's published data revealed outliers (17, Table 1), but he did not mention them. Anscombe (29) made several suggestions for the analysis of outliers.

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studies of this type are needed in a variety of jurisdictions so that a general social area profile may be developed which can provide the various jurisdictions with policy recommendations.

With respect to the competing theories of alcohol problems and alcoholism we conclude, on the basis of this analysis, that neither the sociocultural model, which emphasizes the normative structure of ethnic, racial, and class subgroups, nor the distribution of consumption model, which emphasizes the level of consumption and restrictions on consumption by manipulation of economic and physical availability, is sufficient to understand alcoholism and alcohol-related problems. Both approaches have something to offer in improving our understanding of drinking behavior and its pernicious consequences (32-34). The two sociological perspectives, perceived as competing, with an empirical test turn out to be complementary. Why an ecological and normative perspective has been treated in isolation is well beyond the scope of this paper. Our complementary perspective, however, suggests that among particular subgroups certain types of outlets (and drinking) are used more frequently. Availability has an effect on alcohol problems independent of social area characteristics. Our work also suggests the need for time series research which could pinpoint the causal direction in this sociocultural and ecological debate.

REFERENCES

1. PITTMAN, D. J. and SNYDER, C. K., eds. *Society, culture and drinking patterns*. New York: Wiley; 1962.
2. HEATH, D. B. A critical review of the sociocultural model of alcohol use. Pp. 1-18. In: HARFORD, T. C., PARKER, D. A. and LIGHT, L., eds. *Normative approaches to the prevention of alcohol abuse and alcoholism*. (NIAAA Research Monogr. No. 3.) (DHEW Pub. No. ADM-79-847.) Washington, D. C.: U.S. Govt Print. Off.; 1980.
3. DE LINT, J. and SCHMIDT, W. Consumption averages and alcoholism prevalence: a brief review of epidemiological investigations. *Br. J. Addict.* 66: 97-107, 1971.
4. WHITEHEAD, P. C. and HARVEY, C. Explaining alcoholism: an empirical test and reformulation. *J. Health Soc. Behav.* 15: 57-65, 1974.
5. DE LINT, J. Epidemiological aspects of alcoholism. *Int. J. Ment. Health* 5: 29-51, 1976.
6. PARKER, D. A. and HARMON, M. S. The distribution of consumption model of prevention of alcohol problems. *Q. J. Stud. Alcohol* 39: 377-399, 1979.
7. SMART, R. G. The relationship of availability of alcoholic beverages to per capita consumption and alcoholism rates. *J. Stud. Alcohol* 38: 891-896, 1977.
8. SMART, R. G. Availability and the prevention of alcohol-related problems. Pp. 123-146. In: HARFORD, T. C., PARKER, D. A. and LIGHT, L., eds. *Normative approaches to the prevention of alcohol abuse and alcoholism*. (NIAAA Research Monogr. No. 3.) (DHEW Pub. No. ADM-79-847.) Washington, D. C.: U.S. Govt Print. Off.; 1980.
9. PARKER, D. A., WOLZ, M. W. and HARFORD, T. C. The prevention of alcoholism: an empirical report on the effects of outlet availability. *Alcsm, Clin. Exp. Res.* 2: 339-343, 1978.

10. HANFORD, T. C., PARKER, D. A., PAULTER, C. and WOLZ, M. Relationship between number of on-premise outlets and alcoholism. *J. Stud. Alcohol* 40: 1053-1057, 1979.
11. FRANKEL, B. G. and WHITEHEAD, P. C. Drinking and damage: theoretical advances and implications for prevention. (Rutgers Center of Alcohol Studies, Monogr. No. 14.) New Brunswick, N.J.; 1981.
12. BRUUN, K., EDWARDS, G., LUMIO, M., MÄKELÄ, K., PAN, L., POPHAM, R. E., ROOM, R., SCHMIDT, W., SKOG, O.-J., SULKUNEN, P. and ÖSTERBERG, E. Alcohol control policies in public health perspective. (Finnish Foundation for Alcohol Studies, Vol. 25.) Helsinki; 1975.
13. WATTS, R. K. and RABOW, J. The role of tourism in measures of alcohol consumption, alcohol availability and alcoholism. *J. Stud. Alcohol* 42: 797-801, 1981.
14. BAILEY, M. B., HABERMAN, P. W. and ALKSNE, H. The epidemiology of alcoholism in an urban residential area. *Q. J. Stud. Alcohol* 26: 19-40, 1965.
15. CAHALAN, D. and CISIN, I. H. American drinking practices: summary of findings from a national probability sample. I. Extent of drinking by population subgroups. *Q. J. Stud. Alcohol* 29: 130-151, 1968.
16. LEVY, L. and ROWITZ, L. The ecology of mental disorder. New York: Behavioral Publications; 1973.
17. DONNELLY, P. C. Alcohol problems and sales in the counties of Pennsylvania: a social area investigation. *J. Stud. Alcohol* 39: 848-858, 1978.
18. TERRIS, M. Epidemiology of cirrhosis of the liver: national mortality data. *Am. J. Ment. Health* 57: 2076-2088, 1967.
19. STERNE, M. W. Drinking patterns and alcoholism among American Negroes. Pp. 66-99. In: PITTMAN, D. J., ed. *Alcoholism*. New York: Harper & Row; 1967.
20. ORNSTEIN, S. I. An economic analysis of the relationship of alcohol control laws to the consumption of alcoholic beverages; a report to the California Department of Alcoholism and Drug Programs. Los Angeles: University of California Alcohol Research Center; 1980.
21. MEDICINE IN THE PUBLIC INTEREST. A study in the actual effects of alcoholic beverage control laws. Washington, D.C.; 1979.
22. REDICK, R. W., GOLDSMITH, H. F. and UNGER, E. L. 1970 census data used to indicate areas with differential potentials for mental health and related problems. (Mental Health Statistics, Ser. C, Methodology Rep. No. 3.) Washington, D.C.; U.S. Govt Print. Off.; 1971.
23. ROSEN, B. M., LAWRENCE, L., GOLDSMITH, H. F., WINDLE, C. D. and SHAMBAUGH, J. P. Mental health demographic profile system: purpose, contents and sampler of uses. (Mental Health Statistics, Ser. C, Methodology Rep. No. 11.) Washington, D.C.; U.S. Govt Print. Off.; 1975.
24. SHEVY, E. and BELL, W. Social area analysis. Stanford: Stanford University Press; 1955.
25. GREER, S. The emerging city: myth and reality. New York: Free Press; 1962.
26. DURKHEIM, E. Suicide: a study in sociology. New York: Free Press; 1951.
27. COHEN, J. and COHEN, P. Applied multiple regression: correlation analysis for the behavioral sciences. New York: Halstead; 1975.
28. GOVE, W. and HUGHES, M. Reexamining the ecological fallacy: a study in which aggregate data are critical in investigating the pathological effects of living alone. *Social Forces* 58: 1157-1177, 1980.
29. ANSCOMBE, F. J. Rejection of outliers. *Technometrics* 2: 123-167, 1960.
30. BORGATTA, E. F. and JACKSON, D. J. Aggregate data analysis: an overview. Pp. 7-11. In: BORGATTA, E. F. and JACKSON, D. J., eds. *Aggregate data analysis and interpretation*. Beverly Hills, Calif.: Sage; 1980.
31. FURST, C. J. and BECKMAN, L. J. Alcohol-related mortality and alcohol consumption statistics: stability of estimates for small areas. *J. Stud. Alcohol* 42: 57-63, 1981.

- M. Relationship between alcohol 40: 1053-1057, 1979.
; theoretical advances and Studies. Monogr. No. 14.)
- ., POPHAM, R. E., ROOM, BERG, E. Alcohol control for Alcohol Studies, Vol.
- s of alcohol consumption, 7-801, 1981.
emiology of alcoholism in 1965.
summary of findings from population subgroups. Q. J.
- r. New York; Behavioral
- of Pennsylvania; a social
- ortality data. Am. J. Ment.
- frican Negroes. Pp. 66-99. Row; 1967.
- alcohol control laws to the California Department of y of California Alcohol
- ects of alcoholic beverage
- nsus data used to indicate elated problems. (Mental ington, D.C.; U.S. Govt
- D. D. and SHAMBAUGH, J. ents and sampler of uses. Washington, D.C.; U.S.
- tanford University Press;
- ree Press; 1962.
Press; 1951.
- relation analysis for the
- allacy; a study in which al effects of living alone.
- 167, 1960.
- ; an overview. Pp. 7-11. gate data analysis and
- and alcohol consumption hol 42: 57-63, 1981.
32. WATTS, R. K. and RABOW, J. Alcohol availability and alcohol-related problems in California cities. *Alcsm, Clin. Exp. Res.* [In press.]
 33. RABOW, J. and WATTS, R. The availability of alcohol and alcohol problems; some theoretical explanations and empirical consequences. In: GALANTER, M., ed. *Recent developments in alcoholism.* [In press.]
 34. RABOW, J., SCHWARTZ, C., STEVENS, N. and WATTS, R. K. Social psychological dimensions of alcohol availability; the relationship of perceived social obligations, price considerations and energy expended to the frequency, amount and type of alcoholic beverage consumed. *Int. J. Addict.* [In press.]

Prediction of Alcoholism from Alcohol Availability, Alcohol Consumption and Demographic Data

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and Wyatt C. Jones, Ph.D.³

SUMMARY. *Cirrhosis mortality in the fifty states and the District of Columbia was predicted by alcohol consumption, alcohol availability, and several social, cultural, economic and demographic variables.*

ALCOHOL AVAILABILITY, alcohol consumption, and demographic variables have all been posited to influence the incidence of alcoholism. The relationship of these variables to liver cirrhosis mortality as an index of alcoholism is the subject of the present study.

In the United States, laws enacted at the state level seek to control alcohol consumption, establish the minimum legal age for purchasing or drinking alcohol, limit the kind and number of retail outlets and their hours of sale, and specify the type and amount of taxation and advertising. Despite these laws having their origins in the compromises of the political process and the economics of alcohol revenues, the manipulations of these statutes reflect social policy and are central to many public-health alcoholism-prevention models.

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Representatives of the beverage alcohol industry and others argue that restrictive control laws are unnecessary impositions on the social drinker and are likely to exert no influence on the problem drinker (1, 2). Such legislation may create an aura of intrigue about alcohol that encourages its use by the young (3). Some analysts argue that strong and consistent social-drinking norms limit intoxication and the consequent problems due to excessive drinking. Others, employing an economic model, contend that price or taxation is the key to the control of alcohol availability and, in turn, of alcoholism. Such legally enforced economic sanctions are presumed to eliminate harmful levels of consumption (4-10). Alcohol is conceptualized as the disease agent, and the distribution and availability laws as the primary means of prevention (11).

The single-distribution (lognormal) model, which has generated some of the strongest arguments for alcohol control legislation short of prohibition, is based on the positive association between alcohol consumption and cirrhosis mortality. The distribution of consumption in a population is said to approximate a lognormal curve, and the daily consumption of 15 cl or more of absolute alcohol is considered the operational definition of alcoholism. For example, Whalley (12) found that this level of consumption distinguished 94% of the hospitalized alcoholics in his sample from a matched set of controls. People consuming this amount tend to be physically dependent on alcohol.

Assuming that consumption is causally linked to cirrhosis mortality, and that there is little variation from society to society in the dispersion of the consumption curve, Bruun et al. (8) concluded that the proportion of heavy drinkers found in the upper limits of the distribution is determined primarily by the average level of consumption, which in turn reflects demographic differences. To the extent that beverage alcohol behaves like most other economic commodities, this model suggests that alcohol can be controlled by such regulatory measures as raising the minimum legal purchasing age, restricting the number of retail outlets, and controlling the price by taxation.

Empirical assessment of the model has focused on the shape of the distribution (8, 9, 13-17). The lognormal curve fits the data better than either the normal or the bimodal distributions, although it does not account for the proportion of abstainers in a population because it erroneously sets their number at zero (18). In fact, the proportion of abstainers might be negatively related to

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alcohol consumption by drinkers. The lognormal model is suggestive but it requires further empirical evaluation.

Cartwright et al. (19), in a longitudinal study testing several propositions derived from the single-distribution model, found that an increase in consumption in a London suburb between 1965 and 1974 was due to drinkers shifting to higher consumption levels rather than to abstainers becoming drinkers. Increased amounts of consumption per occasion and not more frequent occasions accounted for the increase in alcohol-problem scores. Alcohol problems were associated with younger age and lower occupational status. The factor of alcohol availability could not be assessed unambiguously because the number of outlets selling alcohol for off-premise consumption increased and the number of outlets selling alcohol for on-premise consumption decreased during the study period.

In a cross-sectional study of the states, Smart (20) found no relationship between alcohol availability and consumption or cirrhosis mortality rates when urbanization and income were controlled for. Suggesting its role as an antecedent variable, income is more strongly related to consumption and cirrhosis mortality than it is to availability. Ede and Templer (21) similarly found that the diminished availability occasioned by a 61-day strike by Canadian liquor-store employees had no measurable influence on hospital admissions for acute episodes of alcoholism. In contrast to these two studies, Magruder (22) found alcohol availability to be significantly associated with cirrhosis mortality in the 100 counties of North Carolina.

There are at least two problems in these and other studies that bias their measurement of consumption as based on records of alcohol sales and shipments. Consumption in some jurisdictions is underestimated by the failure to take account of illegally made and distributed "moonshine" and "bootleg" liquor and is distorted (e.g., in Nevada, Florida, and the District of Columbia) by the failure to consider the impact of tourism (5, 23, 24).

In a regression analysis of 67 counties in Pennsylvania, Donnelly (25) found that per-capita liquor sales were best predicted by median income, the percentage of men in high-status occupations, the percentage of the population with foreign parentage, and (negatively) by the percentage of families with children under 18. Police arrests for alcohol-related incidents were best predicted by median income, the percentage of the population Black, the

percentage of other non-Whites, and per-capita liquor sales. Cirrhosis deaths were best predicted by the percentage of the population with foreign parentage, and negatively by both the percentage of husband-wife families and the percentage of families with children under 18. Consumption (as measured by sales) was not among the highest-ranked variables accounting for cirrhosis mortality.

These findings suggest that efforts to link cirrhosis mortality rates to average consumption would be enhanced by the inclusion of sociocultural and demographic factors and by taking account statistically of illegal availability and tourism.

These studies provide evidence for the approximate fit of the lognormal curve of the distribution of consumption and establish the consumption level critical to alcoholism, but the relationship of legislated alcohol availability to consumption and cirrhosis mortality is equivocal. Appropriate controls for illegal availability and tourism are indicated. Economic variables, such as income, and sociocultural and demographic variables account for some part of consumption and cirrhosis mortality. The analyses reported below evaluate the relationship of these variables to consumption and cirrhosis mortality, and assess in turn the role of consumption in accounting for cirrhosis mortality.

METHOD

The units of analysis were the 50 states and the District of Columbia for the Census year 1970. The independent variables included six indicators of alcohol availability (or restrictiveness), numerous demographic and sociocultural characteristics, one indicator of moonshining (illicit production) or bootlegging (illicit sale), and two indicators of tourism. Since some of the variables proved to be quite skewed, appropriate transformations were used to reduce the effect of extreme scores on the product-moment correlations. Nonlinear transformations, the logarithmic transformation in particular, not only alleviate distributional nonnormality (positive skew), but also reduce unequal variability (heteroscedasticity), still maintaining the relative positions of the data points (26).

To test for the possibility that the transformations distorted the relationships obtained among the independent, control, and dependent variables, oblique and orthogonal factor analyses were conducted on both transformed and untransformed variables. Since the observed differences were minimal, only the results of analyses with transformed variables are reported.

An oblique factor analysis was performed to delineate dimensions among the independent variables and to reduce their number. Consump-

liquor sales, percentage of the population by both the age of families (by sales) was used for cirrhosis mortality rates. The inclusion of consumption in the model gave a better fit of the data and establish the relationship of cirrhosis mortality and alcohol availability and income, and some part of the reported below consumption and consumption in

Dependent Variables. Cirrhosis deaths per 1000 people aged 14 years and older were used as the main dependent variable (27). Treatments of the data employing a different dependent variable can be found in Colón (28) and Colón et al. (29).

Employed both as a dependent and an independent variable, the per-capita consumption of absolute alcohol (30) attributed to the population aged 15 years or older was estimated from quantities of distilled spirits, wine and beer shipped, multiplied by their alcohol content (45, 16 and 4.5%, respectively).

Control Variables. Seizures of grain mash (gallons per 1000 population) were used to account for the distorting influences of illicit supplies (30). A 3-year average centered on 1970 was used to smooth year-to-year fluctuations.

Tourism was represented by the proportion of the labor force providing recreation, entertainment and lodging services (31).

Independent Variables. Measures of income, price and state restrictions on alcohol availability were selected to represent the policy elements of the single-distribution model. The measure of income was per-capita disposable personal income.⁴

The price of a popular brand of whisky was used as an index of the price of absolute alcohol (32). It was assumed that the prices of other alcoholic beverages were comparable. The composite Consumer Price Index for alcoholic beverages could not be used because it is a measure of price changes rather than of price levels.

Degree of availability was measured by six policy variables: county-level prohibition, state taxes, method of distribution (state-monopoly vs license state), minimum legal purchasing age, retail outlets, and on-premise licenses. County-level prohibition was measured by the proportion of a state's population residing in dry counties (33).

State control of distribution was represented by a dummy-coded variable (1 = control state; 0 = license state). State alcoholic-beverage taxes (34) were converted to a per-gallon basis by dividing total tax receipts by consumption of absolute alcohol in gallons. The minimum legal purchasing age was measured by the proportional increment in the legal drinking population attributable to the decrease in the drinking age. When a state varied its purchasing age by beverage type (always lower for wine and beer than for distilled spirits), the lowest age was used, as it

⁴ U.S. DEPARTMENT OF COMMERCE. BUREAU OF ECONOMIC ANALYSIS. Disposable personal income by state for 1970. (Data available from source upon request.)

probably best reflects the impact of the policy. The variable of retail outlets was formed by summing U.S. Treasury Department licenses issued for the distribution of distilled spirits, wine and beer (30). The total number of off-premise retail outlets was measured per 10,000 population. On-premise licenses per 1000 were counted for distilled-spirits licenses only (32). Private clubs and dual licenses (on- and off-premise) were included in the on-premise count.

Various sociocultural and demographic concepts were represented in the remaining independent variables. Social control was represented by per-capita churches and police. Churches per 10,000 population is a count of parishes and congregations (35). Inexplicably, the Mormon Church of Utah reported zero churches in that state. An estimate of Mormon churches from national data was made in proportion to each state's Mormon population in the national total. Per-capita police includes both state and local police (36). The proportion of unassimilated groups (Blacks, American Indians, Hispanics, and foreign-born) in the population (37) was included as a variable because these groups exhibit different drinking patterns from those of the general population (5, 38).

Several measures were employed to reflect the concept of isolation: the proportions of men and women over 14 years of age who were single, divorced, widowed (37) or living in single-person households (39).

RESULTS

Factor Analysis. Five factors were extracted in a principal-components analysis with communalities in the diagonals and oblique rotations (direct oblimin, $\Delta = .5$). The Statistical Package for the Social Sciences (40) was used to perform the analyses. The pattern matrix, which best approximates simple structure and consequently is most useful in the overall interpretation of the factors, and the structure matrix, which reveals variables loading on more than one factor, are presented in Table 1 along with simple correlations of the separate variables with consumption and cirrhosis mortality. Correlations (r) among the five sets of factor scores that reflect the interrelationship of nonorthogonal factors are given in Table 2.

Factor 1, entitled Urban Conditions, loaded, in descending order of magnitude, on per-capita police, unintegrated groups, percentage of urbanized population, few churches per capita, high disposable personal income, and state-monopoly distribution. Factor 2, Large Aged Population, was characterized by a low ratio of men to women, a high proportion of divorced or widowed women, high median age, and numerous single-person households. Factor 3, Restricted Availability, was characterized by county prohibition, mash seizures, high per-gallon state taxes, and an absence of on-

TABLE 1.—Transformations, Pattern and Structure Factor Loadings, and Correlations

	Pattern Matrix, Factor Loadings and Communalities										Structure Matrix Factor Loadings				Correlations	
	Large					Low					Urban				Consumption per Capita ^a	Circuits Mortality Rate ^a
	Urban Conditions	Aged Pop.	Restricted Pop.	Liberal Pop.	Adult Pop.	Urban Conditions	Aged Pop.	Restricted Pop.	Liberal Pop.	Adult Pop.	Urban Conditions	Aged Pop.	Restricted Pop.	Liberal Pop.		
State & local police per capita ^a	.907	.034	-.109	-.092	.245	.911	.901	.096	-.388	-.014	.188	.705	.699			
% Foreigners, Native Americans, ^a																
Blacks, Latin surnames	.809	.018	.203	.035	-.058	.735	.828	-.025	.004	.114	-.184	.490	.516			
Urbanization	.783	.257	-.133	-.342	.032	.845	.778	.315	-.348	-.313	.011	.425	.588			
Churches per capita	-.697	-.087	.300	.271	-.145	.779	-.729	-.169	.509	.223	-.160	-.671	-.819			
Disposable personal income per capita	.599	.094	-.483	-.243	-.026	.784	.695	.145	.631	-.208	.042	.677	.871			
Control or license state	-.441	.040	-.196	.013	-.026	.189	-.381	.050	-.085	-.038	.070	-.162	-.188			
Sex ratio ^a	-.079	-.844	-.209	-.147	.353	.902	-.062	-.829	.218	-.001	.195	.069	.250			
% Divorced & widowed women ^b	.067	.894	.500	.258	.161	.901	-.119	.771	.421	.134	.232	-.153	.062			
Median age	-.002	.748	-.141	-.050	.035	.621	.013	.772	-.189	-.173	.234	.086	.342			
Single-person households ^a	.184	.696	-.308	.265	.177	.782	.255	.706	.427	.167	.385	.408	.541			
Dry-county population ^c	-.037	.039	.857	-.019	.148	.718	-.903	.029	.831	-.020	-.029	-.513	-.401			
Callous of man seizures per capita	.116	.071	.676	-.155	.134	.432	-.078	.087	.613	-.148	-.012	-.317	-.311			
State tax per gallon of alcohol	.116	-.245	.636	.276	.015	.561	.012	-.323	.620	.333	-.190	-.233	-.345			
On-premise licenses per capita	.143	-.188	-.538	.099	.140	.427	.273	.145	-.593	.138	.203	.563	.300			
Retail outlets	-.201	-.068	-.388	.582	.304	.639	-.082	.070	.397	.572	.398	.357	.050			
Minimum legal purchasing age ^a	-.067	.090	.059	.519	.130	.278	-.022	.028	.104	.408	-.113	-.122	-.126			
Price	.083	-.108	.038	-.201	-.092	.105	.058	.085	.041	-.266	.135	-.163	-.000			
% Single women ^a	.381	.145	-.322	.319	.737	.682	.559	-.061	.257	.322	.668	.302	.356			
% Divorced & widowed men ^a	.193	.347	-.057	.032	.700	.755	.136	.499	-.277	-.003	.772	.377	.427			
Recreation, entertainment & lodging employees	-.270	-.089	.086	.177	.641	.452	.204	.018	-.115	.221	.577	.308	.266			
Single men	.331	-.321	-.203	.423	-.478	.812	.471	-.491	-.156	.500	-.535	.347	.132			
Eigenvalues	5.001	3.324	2.276	1.544	1.335											
% Total variance	25.0	16.9	12.5	9.0	8.4											

^aLog 10 X transformation.^bX² transformation.^c1/X transformation.

TABLE 2.—*Intercorrelations of the Five Sets of Oblique Factor Scores*

	(2)	(3)	(4)	(5)
(1) Urban conditions	-.015	-.244	.091	-.098
(2) Large aged population		-.055	-.162	.224
(3) Restricted availability			.010	-.221
(4) Liberal availability				.006
(5) Low young-adult population				

premise availability. Factor 4, Liberal Availability, had positive loadings on per-capita retail outlets and minimal legal purchasing age, and a negative loading on price. Factor 5, Low Young-Adult Population, had negative loadings on the proportions of single men and women, and a moderately positive loading on the proportion of divorced and widowed men and the proportion of recreational employees (indicating tourism).

Inspection of the structure matrix indicates that the number of churches, which was negatively loaded on Factor 1, was positively loaded on Factor 3. Income, which loaded positively on Factor 1, loaded negatively on Factor 3. Divorced and widowed men, which loaded primarily on Factor 5, also loaded positively on Factor 2. Single women, which loaded negatively on Factor 5, loaded positively on Factor 1. Single men loaded principally on Factor 5 but also loaded negatively on Factor 2 and positively on Factor 4.

None of the intercorrelations among the factor scores given in Table 2 was statistically significant. Urban Conditions was, however, weakly and negatively related to Restricted Availability; Large Aged Population was positively related to Low Young-Adult Population, which in turn was negatively related to Restricted Availability.

Multiple-Regression Analyses. First, consumption was regressed on the five sets of factor scores. The independent variables were entered stepwise into the equation and are reported in order of variance accounted for. With the exception of variance added at a step (v_A), the statistics given—raw (B) and standardized ($Beta$) regression coefficients, F , p and variance added at the last step, or unique variance (uv)—were obtained when all variables had entered the equation. Simple correlations (r) are also given.

Inspection of Table 3 indicates that the independent variables overall accounted for a large and highly significant proportion of variance (69%). Urban Conditions accounted for the most variance, followed by Restricted Availability (negative relationship), and Liberal Availability. Low Young-Adult Population and Large

Factor Scores

(4)	(5)
.091	-.098
-.162	.224
.010	-.221
	.006

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TABLE 3.—Regression of Consumption on Factor Scores: Unstandardized (B) and Standardized (Beta) Coefficients, Simple Correlation (r), Significance Tests (F) and Unique Variances (UV), All at Final Step, with Variables Ordered by Variance Added (VA) at Step of Entry

	B	Beta	r	F	UV	VA
Urban conditions ‡	.168	.514	.639	33.490	.234	.408
Restricted availability ‡	-.143	-.426	-.608	22.211	.155	.197
Liberal availability †	.081	.232	.260	7.449	.052	.051
Low young-adult population	.058	.174	.217	3.813*	.027	.030
Large aged population	.006	.019	.030	.048	.000	.000
Constant = .923						

$$R^2 = .686, F = 19.628, 5/45 \text{ df}, p < .001$$

† $P < .01$. ‡ $P < .001$.

TABLE 4.—Regression of Cirrhosis Mortality on Factor Scores: Unstandardized (B) and Standardized (Beta) Coefficients, Simple Correlation (r), Significance Tests (F) and Unique Variances (UV), All at Final Step, with Variables Ordered into the Equation by Variance Added (VA) at Step of Entry

	B	Beta	r	F	UV	VA
Urban conditions ‡	.192	.538	.621	30.005	.256	.386
Large aged population ‡	.122	.343	.352	12.716	.109	.129
Restricted availability †	-.115	-.315	-.487	9.906	.085	.098
Low young-adult population	.018	.048	.146	.240	.002	.002
Liberal availability	.009	.025	.013	.069	.001	.001
Constant = 2.893						

$$R^2 = .616, F = 14.409, 5/45 \text{ df}, p < .001$$

† $P < .01$. ‡ $P < .001$.

Aged Population did not account for significant proportions of variance when all other variables were in the equation.

The regression of cirrhosis mortality on the five sets of factor scores was similarly conducted and is presented in Table 4. The overall R^2 (62%) is highly significant. Urban Conditions, Large Aged Population, and Restricted Availability (negative relationship) accounted for significant proportions of variance with all variables in the equation.

The regression of cirrhosis mortality on consumption and the five sets of factor scores is reported in Table 5. The overall R^2 (76%) is highly significant, as were the individual variables of Consumption and Large Aged Population. With Consumption in the equation, Urban Conditions and Restricted Availability no longer accounted for significant proportions of variance in predicting cirrhosis mortality.

When the equation contained only Consumption, R^2 is .595; only the other five factor-score variables, R^2 is .616; and all six

TABLE 5.—Regression of Cirrhosis Mortality on
Scores: Unstandardized (B) and Standardized (Beta)
Correlation (r), Significance Tests (F) and Unique
Step, with Variables Ordered into the Equation by
Step of Entry

	B	Beta	r
Consumption ‡	.727	.667	.727
Large aged population ‡	.118	.330	.330
Urban conditions	.070	.196	.62
Liberal availability	-.050	-.130	.013
Low young-adult population	-.025	-.068	.146
Restricted availability	-.011	-.031	.487
Constant = 2.893			

$R^2 = .755$, $F = 27.683$, 6/

‡ $P < .001$.

independent variables, R^2 is .755. Commonality analysis shows that the common variance of consumption (.595) plus the unique variance of factor scores (.616) minus all independent variables (.727) = .456 ($F = 36.88$, 1/44 df, $p < .001$). Consumption added unique variance over the other variables; $uv = .755 - .616 = .140$ ($F = 25.14$, 1/44 df, $p < .001$). The other variables added unique variance over consumption; $uv = .755 - .595 = .160$ ($F = 5.80$, 5/44 df, $p < .001$).

Inspection of significant effects in the three analyses suggests more specifically, that Urban Conditions was positively related to Cirrhosis Mortality. Restricted Availability was negatively related both to Consumption and to Cirrhosis Mortality. Liberal Availability had a small positive relationship to Consumption only, and Large Aged Population was related to Cirrhosis Mortality only. When Consumption was entered as an intervening variable with the factor-score sets, Urban Conditions and Restricted Availability dropped out, their variance having been accounted for by Consumption, to which they were also strongly related. The relationship of Large Aged Population to Cirrhosis Mortality is due in part to the low rate of alcohol intake to develop cirrhosis and to the high rate of alcohol

The two

TABLE 6.—*continued*

	Urban Conditions	Large Aged Population	Restricted Availability	Liberal Availability	Low Young- Adult Population
Rhode Island	1.103	0.100	- 0.472	- 0.221*	- 0.454
South Carolina	- 0.578	- 1.021	1.199	1.315	- 1.166
South Dakota	- 0.979	- 0.663	- 0.144	1.831	- 0.611
Tennessee	- 0.724	0.573	1.609	- 0.536	0.066
Texas	0.206	0.217	0.889	0.120	0.517
Utah	- 0.064	- 0.999	0.147	- 0.862	- 1.434
Vermont	- 0.466	- 0.497	- 0.245	2.359	- 0.176
Virginia	- 0.026	- 0.765	0.736	- 0.273	- 0.718
Washington	- 0.205	0.549	- 0.731	- 0.677	0.851
West Virginia	- 2.320	0.719	0.412	0.447	- 0.206
Wisconsin	0.259	- 0.299	- 1.233	1.631	0.090
Wyoming	- 0.707	- 0.189	- 0.913	- 0.438	1.325
District of Columbia	3.404	2.198	- 0.705	2.613	- 0.434

The restricted-availability dimension characterizes areas with fundamentalist and proscriptive attitudes, e.g., the Southeast. It would seem difficult to argue that availability measures induce such attitudes; more plausibly, availability is an index or result of such attitudes. It is interesting that only this form of availability is significantly related to cirrhosis mortality. The other availability factor, liberal availability, characterizes states that cater to transient populations such as tourists and out-of-state purchasers, e.g., Nevada and Vermont. Although such states may be high on apparent consumption, no relationship was obtained between this factor and cirrhosis mortality.

The factor analysis also revealed that the availability factors were mostly unrelated to urban conditions, which also has a primary loading on disposable personal income. It would appear, then, that a prevention model based solely on economic variables is untenable because these variables are intertwined with sociodemographic variables that are themselves individually predictive of alcoholism. An economic conception of alcoholism prevention is even more problematic when one considers that the only availability factor related to cirrhosis mortality appears to be a reflection of the normative structure of a region.

Consumption appears to be the most immediate cause of liver cirrhosis but examination of the relationship of all the factors to cirrhosis mortality suggests a rather broad approach to prevention.

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F	UV	VA
103	.140	.595
094	.101	.108
186	.019	.033
521	.014	.015
669	.004	.004
097	.000	.001

df, $p < .001$

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high on one set of measures and low on the other (Table 6). The complex interplay of social, political, economic and religious forces that have contributed to the evolution of control laws and the piecemeal manner in which these have been repealed and amended would preclude the emergence of any single unifying policy structure or philosophy.

TABLE 6.—Oblique Factor Scores for the 50 States and the District of Columbia

	Urban Conditions	Large Aged Population	Restricted Availability	Liberal Availability	Low Young- Adult Population
Alabama	- 0.545	0.152	2.013	- 0.773	- 0.452
Alaska	0.617	- 3.796	- 0.966	0.039	0.765
Arizona	0.949	- 0.574	- 0.534	- 0.210	0.242
Arkansas	- 1.180	0.661	1.737	- 0.779	0.807
California	1.447	- 0.297	- 1.504	- 0.896	0.266
Colorado	0.150	- 0.135	- 0.522	0.101	0.097
Connecticut	0.689	0.375	- 0.600	- 0.806	- 1.095
Delaware	0.278	0.057	- 0.308	- 1.156	- 0.630
Florida	0.671	1.262	1.112	- 0.281	1.934
Georgia	- 0.065	0.069	2.487	- 0.357	- 0.544
Hawaii	1.860	- 2.879	- 0.262	0.198	- 0.968
Idaho	- 0.970	- 0.416	- 0.817	0.252	1.046
Illinois	0.841	0.876	- 0.352	- 0.795	0.066
Indiana	- 1.118	0.671	0.021	- 1.279	- 0.107
Iowa	- 1.276	0.586	- 0.885	- 0.915	- 0.109
Kansas	- 0.518	0.521	0.114	- 0.051	0.325
Kentucky	- 1.491	0.177	1.229	- 0.616	- 0.222
Louisiana	0.663	- 0.534	1.006	1.126	- 0.164
Maine	- 0.946	0.441	- 0.271	0.514	0.323
Maryland	0.599	- 0.238	- 0.280	- 1.180	- 0.611
Massachusetts	1.351	0.722	- 0.192	- 0.295	- 1.151
Michigan	0.386	- 0.182	- 0.208	- 0.890	- 0.249
Minnesota	- 0.266	- 0.093	- 0.393	0.374	- 1.138
Mississippi	- 0.321	- 0.537	2.115	1.456	- 0.297
Missouri	0.050	1.113	- 0.452	0.102	0.910
Montana	- 0.336	- 0.395	- 0.695	0.867	1.187
Nebraska	- 0.754	0.473	- 0.578	0.079	- 0.369
Nevada	0.623	0.352	- 1.186	0.041	3.788
New Hampshire	- 0.645	0.081	- 0.857	0.280	- 0.033
New Jersey	1.001	0.506	- 0.454	- 1.406	- 0.795
New Mexico	0.476	- 0.997	0.768	- 0.010	- 0.319
New York	1.625	0.814	- 0.816	0.550	- 0.371
North Carolina	- 0.432	- 0.838	1.661	0.285	- 1.013
North Dakota	- 0.768	- 1.561	- 0.015	1.039	- 1.319
Ohio	- 0.289	0.730	- 0.375	- 0.626	- 0.049
Oklahoma	- 0.692	1.003	0.148	0.290	1.953
Oregon	- 0.541	0.867	- 0.870	- 0.668	1.165
Pennsylvania	- 0.027	1.042	- 0.568	- 0.915	- 0.521

continued—

ty	Low Young- Adult Population
	- 0.454
	- 1.166
	- 0.611
	0.066
	0.517
	- 1.434
	- 0.176
	- 0.718
	0.851
	- 0.206
	0.090
	1.325
	- 0.434

It appears futile to develop a prevention strategy that touches only on alcohol consumption without addressing attitudes, social conditions, and urbanization factors. Community programs that influence the immediate quality of life are indicated rather than centralized approaches that rely on laws and their enforcement. Church, social and recreational programs may be more efficacious than the manipulation of alcoholic beverage control laws. Alcoholics Anonymous and other socially integrative mutual-help groups with and without religious overtones have demonstrated some efficacy in the arrest of alcoholism. The encouragement of groups that increase social integration and work to prevent excess consumption and the consequent alcoholism would seem to be desirable. This is not to say that alcohol-control policies do not serve other fiscal, social, or psychological ends; just that, given the limited resources available to address the problem of alcoholism, care should be exercised in clearly identifying the ends served by a policy.

The results support the idea that the consumption of alcohol is a necessary but not sufficient prerequisite of alcoholism. Consumption is associated with several of the sociocultural, economic and availability factors but is fully explained by none of them. Thus no single, simple or sovereign solution to the problem of alcoholism is available or feasible. Economic and legislative approaches to the problem are readily short-circuited by illegal availability, especially, as noted above, in the areas where the most restrictive legislation is still in force. If the experience with Prohibition is any guide, the overall average reduction in intake accounted for by law-abiding citizens may not serve to reduce heavy drinking among those less scrupulous. Clearly there is still a strong need for a national policy and a personal consensus that recognize the damage caused by excessive alcohol consumption and seek to establish a lifestyle that is free of this disabling practice.

REFERENCES

1. DISTILLED SPIRITS COUNCIL OF THE UNITED STATES, INC. *DISCUS facts book*; 1977. The beverage alcohol industry; public attitudes and economic progress. Washington, D.C.; 1977.
2. Control of availability policy faulted. *DISCUS Newsl.* No. 390: 3,7, 1980.
3. WILKINSON, R. *The prevention of drinking problems; alcohol control and cultural influences*. New York: Oxford University Press: 1970.
4. BLACKER, E. Sociocultural factors in alcoholism. *Int. Psychiat. Clin.* 3: 51-80, 1966.
5. U.S. NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM. *Alcohol and health*.

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- First special report to the Congress. (DHEW Pub. No. HSM 72-9099.) Washington, D.C.; U.S. Govt Print. Off.; 1971.
6. PLAUT, T. F. A. Alcohol problems; a report to the nation. New York; Oxford University Press; 1967.
 7. ALLMAN, A. D. Sociocultural backgrounds of alcoholism. *Ann. Am. Acad. Polit. Soc. Sci.* 135: 48-54, 1958.
 8. BRUUN, K., EDWARDS, G., LUMIO, M., MÄKELÄ, K., PAN, L., POPHAM, R. E., ROOM, R., SCHMIDT, W., SKOG, O.-J., SULKUNEN, P. and ÖSTERBERG, E. Alcohol control policies in public health perspective. (Finnish Foundation for Alcohol Studies, Vol. 25.) Helsinki; 1975.
 9. LEDERMANN, S. Alcool, alcoolisme, alcoolisation. Vol. 1. Données scientifiques de caractère physiologique, économique et social. (Institut National d'Etudes Démographiques, Travaux et Documents, Cahier No. 29.) Paris: Presses Universitaires de France; 1956.
 10. POPHAM, R. E., SCHMIDT, W. and DE LINT, J. The prevention of alcoholism; epidemiological studies of the effects of government control measures. *Br. J. Addict.* 70: 125-144, 1975.
 11. U.S. NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM. Alcohol and health. Third special report to the Congress. (DHEW Pub. No. ADM-79-832.) Washington, D.C.; U.S. Govt Print. Off.; 1979.
 12. WHALLEY, L. J. Alcohol consumption in 50 men alcoholics and a matched control group in Scotland. *J. Stud. Alcohol* 40: 117-119, 1979.
 13. SKOG, O.-J. The distribution of alcohol consumption in a population. Oslo: National Institute for Alcohol Research; 1971.
 14. CELENTANO, D. and MCQUEEN, D. Comparison of alcoholism prevalence rates obtained by survey and indirect estimates. *J. Stud. Alcohol* 39: 420-434, 1978.
 15. DE LINT, J. and SCHMIDT, W. The distribution of alcohol consumption in Ontario. *Q. J. Stud. Alcohol* 29: 968-973, 1968.
 16. LEDERMANN, S. Alcool, alcoolisme, alcoolisation. Vol. 2. Mortalité, morbidité, accidents du travail. (Institut National d'Etudes Démographiques, Travaux et Documents, Cahier No. 41.) Paris: Presses Universitaires de France; 1964.
 17. SCHMIDT, W. and DE LINT, J. Estimating the prevalence of alcoholism from alcohol consumption and mortality data. *Q. J. Stud. Alcohol* 31: 957-964, 1970.
 18. DUFFY, J. C. Comment on "The single distribution theory of alcohol consumption." *J. Stud. Alcohol* 39: 1648-1650, 1978.
 19. CARTWRIGHT, A. K. J., SHAW, S. J. and SPRUELL, T. A. The relationship between per capita consumption, drinking patterns and alcohol related problems in a population sample, 1965-1974. II. Implications for alcohol control policy. *Br. J. Addict.* 73: 247-258, 1978.
 20. SMART, R. G. The relationship of availability of alcoholic beverages to per capita consumption and alcoholism rates. *J. Stud. Alcohol* 38: 891-896, 1977.
 21. EDE, A. and TEMPLER, D. I. The effect of a liquor store strike upon psychiatric admissions for alcoholism. *Can. Psychiat. Ass. J.* 21: 501, 1976.
 22. MACGRUDER, K. M. The association of alcoholism mortality with legal availability of alcoholic beverages. *J. Alc. Drug Educ.* 21: 27-37, 1976.
 23. MILLER, C. H. and AGNEW, N. The Ledermann model of alcohol consumption. *Q. J. Stud. Alcohol* 35: 877-898, 1974.
 24. WALES, T. J. Distilled spirits and interstate consumption effects. *Am. Econ. Rev.* 58: 853-863, 1968.
 25. DONNELLY, P. G. Alcohol problems and sales in the counties of Pennsylvania. *J. Stud. Alcohol* 39: 848-858, 1978.
 26. COHEN, J. and COHEN, P. Applied multiple regression/correlation analysis for the behavioral sciences. New York; Erlbaum; 1975.

27. U.S. PUBLIC HEALTH SERVICE. NATIONAL CENTER FOR HEALTH STATISTICS. Vital statistics of the United States. Vol. 2. Mortality. Part A. Hyattsville, Md.; 1970.
28. COLÓN, I. Alcohol control policies and their relation to alcohol consumption and alcoholism. Ph.D. dissertation, Brandeis University, Florence Heller School of Advanced Studies in Social Welfare, 1979.
29. COLÓN, I., CUTTER, H. S. G. and JONES, W. C. Alcohol control policies, alcohol consumption, and alcoholism. *Am. J. Drug & Alcohol Abuse* 8: 347-362, 1981.
30. U.S. DEPARTMENT OF THE TREASURY. BUREAU OF ALCOHOL, TOBACCO AND FIREARMS. Alcohol and tobacco; summary statistics. Washington, D.C.; U.S. Govt Print. Off.; 1969-1971.
31. U.S. DEPARTMENT OF COMMERCE. BUREAU OF ECONOMIC ANALYSIS. Regional employment by industry; 1940-1970. Washington, D.C.; U.S. Govt Print. Off.; 1975.
32. GAVIN-JOBSON ASSOCIATES. The liquor handbook. New York; Cowles Communications; 1971.
33. DISTILLED SPIRITS COUNCIL OF THE UNITED STATES. Annual statistical review; distilled spirits industry. Washington, D.C.; 1970.
34. DISTILLED SPIRITS COUNCIL OF THE UNITED STATES. Public revenues from alcoholic beverages; 1970. Washington, D.C.; 1971.
35. JOHNSON, D. W. Churches and church membership in the U.S.; 1971. Washington, D.C.; Glenmary Research Center; 1974.
36. U.S. BUREAU OF THE CENSUS. Statistical abstract of the United States; 1972. Washington, D.C.; U.S. Govt Print. Off.; 1972.
37. U.S. BUREAU OF THE CENSUS. Statistical abstract of the United States; 1973. Washington, D.C.; U.S. Govt Print. Off.; 1973.
38. HARPER, F. D. Research and treatment with Black alcoholics. *Alcohol Health & Res. World* 4: 10-16, 1980.
39. U.S. BUREAU OF THE CENSUS. County and city data book; 1972. Washington, D.C.; U.S. Govt Print. Off.; 1973.
40. NIE, N. H., HULL, C. H., JENKINS, J. G., STEINBRENNER, K. and BENT, D. H. SPSS; statistical package for the social sciences. 2d ed. New York; McGraw-Hill; 1975.

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FIFTH SPECIAL REPORT TO THE U.S. CONGRESS ON
ALCOHOL AND HEALTH
FROM THE SECRETARY OF HEALTH AND HUMAN SERVICES

DECEMBER 1983

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Foreword

As this report so amply documents, the price of alcohol abuse to the American people is appallingly high. And that price is paid by all of us. The cost to our economy has been estimated at over \$49 billion each year. But the dollar cost is only the beginning. The cost in human pain and misery, disease, and death is still more staggering. Accidents on our streets and highways claim about 50,000 Americans' lives each year—leaving another 150,000 permanently disabled. Half of those tragedies are alcohol related. Alcohol's role in disrupting family life is less easily documented, but few doubt that it is significant. One in three Americans surveyed last year felt that alcohol caused problems in his or her family.

Fortunately, this long-neglected problem has now become of increasing concern to many Americans. As a result of citizen action, drunk driving, once largely ignored, is now being taken seriously. More and more States are enacting tougher laws to discourage driving after drinking. Treating alcoholics once was of

interest to a few dedicated men and women—themselves often recovering alcoholics. Treatment now involves many thousands in the helping professions as well. In industry, many, perhaps most, companies are coming to recognize that losing a valued employee to alcoholism is costly—and that making provision for treatment just makes good business sense. Many health insurance plans are providing benefits to treat alcoholism itself as they discover it costs much less in the long run than treating its chronic health consequences.

Perhaps the most encouraging sign is the increasing awareness Americans are developing that we ourselves have a primary responsibility for preserving our well-being by practicing better health habits—including moderation in alcohol consumption.

This report deserves to be widely read—and its implications thoughtfully considered. Through our combined efforts, we can sharply reduce alcohol abuse and its devastating consequences.

Margaret M. Heckler
Secretary of Health and Human Services
December 1983

Preface

This has been a century of impressive achievement in public health. The advent of vaccines against such diseases as smallpox, diphtheria, and polio and their widespread application have virtually eliminated those once dreaded illnesses. Tuberculosis, the cause of one in eight deaths in 1900, is now uncommon and still more rarely fatal. Antibiotics have made most infectious diseases easily curable. But the devastation of one disease has not yet been significantly eroded. It plays a role in 10 percent of all deaths in the United States. In some segments of the population, it produces still higher mortality. It is the principal cause of deaths through accidents among those aged 15 to 24. That malady is, of course, alcohol abuse.

Alcohol abuse during pregnancy has been found to cause fetal alcohol syndrome and other alcohol-related birth defects. Fetal alcohol syndrome appears to be one of the leading known teratogenic causes of mental retardation. There is reason to believe that alcohol may be injurious to the developing fetus at dose levels no higher than those often consumed by those who drink moderately.

In adults, alcohol abuse is the leading cause of liver cirrhosis (the eighth leading cause in the United States), the leading contributing factor for chronic pancreatitis, and has been

implicated in a wide range of other illnesses as well.

As this and previous Alcohol and Health reports indicate, the increased Federal role in this area has markedly altered an earlier pessimistic outlook with respect to treating those dependent on alcohol. We no longer believe that the alcoholic must reach the late stages of the illness before he or she can benefit from help. On the contrary, as in other illnesses, there is good evidence that the earlier treatment begins, the greater is the likelihood of successful recovery.

Alcohol research has provided us with greater insight into the underlying mechanisms of alcohol dependence. It is by no means visionary to say that our enhanced understanding holds the promise not only of improved treatment, but of preventing alcohol dependence before it develops. We may not be far from the day when simple laboratory tests can determine those who are at greatest risk of becoming alcoholic—before they take that first drink.

The Fifth Special Report, together with its predecessors, provides valuable insight into the impressive progress that has been made—and the distance yet to be traveled before we resolve this major public health problem.

Edward N. Brandt, Jr., M.D.
Assistant Secretary for Health

Acknowledgments

This Fifth Special Report on Alcohol and Health is the product of many people's efforts in reviewing and summarizing hundreds of scientific papers, begun shortly after the Fourth Special Report was forwarded to the U.S. Congress in 1981. The chief contributors, many of whom also contributed to previous reports, include some of the most distinguished scientists and knowledgeable alcoholism researchers and medical authorities in the world today.

Equally important roles were played by individuals who provided professional review of the preliminary report. The combined efforts are reflected in every chapter of the report.

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Other individuals who made significant contributions were Robert Petersen, Ph.D., who served as a writer and provided technical assistance, particularly in the overview and other front matter, and Samuel Rosenfeld, a science writer who participated during the early draft stages of the report.

Introduction

To know where you are, it is often useful to look back at where you've been. Less than 15 years ago, a major U.S. Government publication described the problem of alcohol abuse in America as follows:

No other national health problem has been so seriously neglected as alcoholism. Many doctors decline to accept alcoholics as patients. Most hospitals refuse to admit alcoholics. Available methods of treatment have not been widely applied. Research on alcoholism and excessive drinking has received virtually no significant support.¹

It went on to laud the transition from an "atmosphere of moral disapproval" in which alcoholics were treated as "sinners or criminals" to the recognition that alcoholism is an illness. Only 3 years before, a U.S. Court of Appeals, in a precedent-setting decision, held that a homeless alcoholic's public drunkenness was involuntary. Up until then, nearly half of all arrests (excluding traffic offenses) were related to drunkenness. By 1968, Congress enacted the Alcoholic Rehabilitation Act (P.L. 90-574), declaring that "alcoholism is a major health and social problem" and that "the handling of chronic alcoholics within the system of criminal justice perpetuates and aggravates the broad problem of alcoholism whereas treating it as a health problem permits early detection and prevention . . . and effective treatment and rehabilitation. . . ."

This Act and subsequent legislation (P.L. 91-616) establishing the National Institute on Alcohol Abuse and Alcoholism (NIAAA) translated the long-held position of knowledgeable professionals that alcoholism was a treatable disease into national public policy. It officially declared, "Our task is not to punish, but to heal."

It is ironic that it took so long for public policy to reflect an informed consensus. Nearly 200 years ago, one of our country's most distinguished physicians, Dr. Benjamin Rush, labeled alcohol abuse an "addiction and a disease."² He also provided the first epidemiological estimate of the number of deaths each year from alcohol abuse. His estimate of 4,000 deaths annually in a population of 6 million (66.7 per 100,000) is surprisingly close to present estimates (as high as 93.2 per 100,000).

While the problems associated with alcohol are still very much with us, we at the Institute take satisfaction in what has been achieved in the 12 years since NIAAA was established. The first edition of the *Alcohol and Health* reports (1971) referred to alcoholism as an "iceberg problem," because social pressures caused so many with the disease to deny it. In the years since, the hidden portion of that iceberg has become more and more exposed. There have been important shifts in our attitudes toward drinking. Removing the moral onus from alcoholism made it easier for alcoholics to admit to having a disease rather than a defect of will. Organizations that previously stressed anonymity in their rehabilitation efforts have become more sympathetic to individual self-disclosure. Many of our leading citizens are now willing to "come out"—to acknowledge their own hard-fought battles with alcohol addiction. Their courage has made it easier for others to recognize that they too have alcohol problems—and to do so at an earlier stage.

There have been other benefits of more honestly recognizing that problems from alcohol abuse can occur at all levels of drinking and of the society. The still prevalent myth that drinking is "sophisticated," a necessary part of the "successful" life, is slowly being eroded. It now requires less social courage to refuse a drink or to request a nonalcohol substitute. The belief that drinking is essential to masculinity has been very much a part of

¹ U.S. Department of Health, Education, and Welfare. *Alcohol and Alcoholism*. National Clearinghouse for Mental Health Information Pub. No. 5011. Washington, D.C.: U.S. Government Printing Office, 1969.

² Rush, B. *An Inquiry into the Effects of Ardent Spirits upon the Human Body and Mind; With an Account of the Remedies for Curing Them*. (1785) Brookfield, Merriam, 8th ed. 1814.

American folklore. It is being supplanted by an increasing recognition of the health hazards of alcohol abuse and a new commitment to affirmation of good health practices. A public health revolution is underway with official recognition that the individual can do more for his or her own health than any doctor, hospital, drug, or other medical innovation. The past decade has clearly shown that habits as basic to personal health as smoking, diet, and exercise can be changed--and in a remarkably short time. Our success in changing behaviors that were once thought immutable provides good reason for optimism about alcohol abuse. As has become true of smoking, not drinking or drinking more moderately may become the "in" thing.

The carnage on our highways documented here and in earlier reports has also led to a new public appreciation of the gravity of mixing drinking with driving. Citizen groups have been impressively effective in changing public attitudes toward drinking and driving, making individuals more aware of just how much alcohol impairs driving skill.

Primary prevention--before drinking has even begun--may be one key to sharply reducing the high casualty rate from alcohol described in this report. Early intervention, between the sixth and ninth grades, is one means of educationally "inoculating" children against alcohol (and tobacco) abuse. Such efforts to teach good health habits early need to be carefully evaluated. As indicated here, most alcohol education efforts at older ages have been more successful in increasing knowledge about the drug, possibly altering expressed attitudes, but not in changing actual drinking.

Drinking during pregnancy is now known to pose risks for the health of the newborn. Programs directed toward women who have alcohol problems could sharply reduce the number of newborn who suffer such serious consequences of maternal drinking as the fetal alcohol syndrome and other alcohol-linked birth anomalies.

Treatment of alcoholism has come of age. Once largely confined to dedicated members of groups like Alcoholics Anonymous and a few committed professionals, treatment efforts in recent years have grown almost exponentially. Treating alcoholics is now "respectable"--and training for physicians and others to treat the alcohol-dependent person reflects this new found status.

There is an increased willingness to subject therapeutic efforts to research scrutiny--to

determine what works best with whom and under what circumstances. Innovative employee assistance programs are now challenging the traditional belief that treatment must be completely voluntary to be successful. There is evidence that job-based referrals to treatment, provided early and in lieu of disciplinary action on the job, can be as effective as treatment sought on one's own. Treating alcohol dependence early is far more likely to succeed and is less costly to treat than intervention when this insidious disease is far advanced.

The fact that treating alcoholism within the framework of health insurance plans is cost-effective has now been proven repeatedly. There is no question that this leads to lower long-term health costs to the individual and the plan. Encouraging expansion of health insurance coverage to include adequate treatment of alcohol dependence is an important and continuing priority of the Institute.

The high costs of medical care are coming under more careful scrutiny. While a hospital was once thought to be the only suitable setting for detoxifying alcoholics, other settings may be equally effective--and much more cost-effective. Alternative treatment models employing certified alcoholism counselors have had demonstrated success. As the capabilities of these less expensive alternatives become apparent, health insurance programs should include them within their benefit structure.

A lack of agreement as to just what is meant by such terms as "alcohol abuse" and "alcoholism" has plagued us for many years. Because of it, comparing research and treatment outcomes has sometimes been difficult. We now recognize that alcohol abuse and alcoholism are both multidimensional with multiple causes. The newer modes of classification described in this report attempt to take this complexity into consideration.

One of the most satisfying developments in recent years has been research innovation. Federal support for alcohol research, once almost nonexistent, has had significant impact. By its very nature, basic research is a long-range enterprise, and its practical implications are often not immediately apparent. But studies of the ways in which alcohol alters the cellular anatomy and neurophysiology of the brain are already enlarging our understanding in ways that have practical "payoffs." A sensitive measure of the brain's electrical responses to sound and light stimulation may prove useful in determining those at greater

risk of becoming alcohol dependent. Other indications of the effects of alcohol on chemicals that govern nerve transmission are providing better explanations of just why certain symptoms of alcohol abuse occur. New neurophysiological explanations of how alcohol and other dependency-producing drugs operate offer the tantalizing possibility of some form of chemotherapy to reduce or even eliminate the likelihood of becoming alcohol dependent. While this is not a promise for the immediate future, it is an example of the kind of payoff that is unpredictable in the initial stages of basic research.

Biological markers described here--tests that distinguish heavy drinkers from others by measuring effects on the blood or other biological variables--are becoming valuable in the early diagnosis of those with drinking problems. Used in many kinds of settings, such tests can serve to alert the individual to the reality that he or she has an alcohol problem. There is now evidence that making patients aware of the results of such tests, together with limited followup treatment, can be effective.

Advanced computer technology applied to brain scanning has also shown evidence of alcohol-induced brain damage--even in the absence of obvious clinical signs.

In earlier reports, the significance of heredity--a biological predisposition toward alcoholism--was in doubt. A substantial body of animal and human research now leaves little question that heredity plays a role in susceptibility to some of alcohol's effects and in some individuals increases their likelihood of becoming dependent on it. The distinct possibility of identifying such potential alcoholics early may enable us to focus prevention efforts on those at high risk.

Our advances in knowledge have led to a measure of optimism in dealing with alcohol abuse. The 1990 Objectives for the Nation cited in this report reflect this. We are hopeful, for example, that, by 1990, 80 percent of high school seniors will feel, as only a third now do, that there is "great risk" in becoming intoxicated. It may be possible to reduce the

percentage of problem drinkers of all ages by 20 percent. The increased health awareness in our country should facilitate achieving that goal. At the same time, we hope to attain reductions in alcohol-related accidents of over 20 percent and decrease cirrhosis mortality by 10 percent.

The risk of women's drinking while pregnant is now recognized by only three-quarters of women of childbearing age. Raising the consciousness of women to the point where at least 9 out of 10 are aware of the potential risk by the end of the decade is a realistic goal for our public information efforts.

While over half of Fortune 500 firms now have alcohol prevention and referral programs, we believe that a heightened awareness of the value of employee assistance programs will increase their numbers of 70 percent by 1990.

Building on the significantly improved epidemiological effort of the past decade, it should be possible by 1990 to collect far more comprehensive data on the impact of alcohol abuse on American society that will enable us to better monitor what is happening--and to modify our programs as we become more aware of changing national needs.

If this edition of Alcohol and Health provides some grounds for optimism, there is little basis for complacency. Alcohol continues to exact a fearsome toll. We sometimes forget that the cost of alcohol abuse to American society is higher than that for cancer, and at least equal to that for heart and vascular disease (Alcoholism and Related Problems: Opportunities for Research. Report of the Institute of Medicine of the National Academy of Sciences, Washington, D.C., 1980). The amount of our national resources devoted to reducing these costs is still modest compared with that earmarked for more "acceptable" disease. The moralistic stance of an earlier era, to that extent at least, is still with us. Many--perhaps most of us--continue to experience conflicts about our use of alcohol. As we resolve those conflicts, a more rational individual and collective approach becomes possible.

Robert G. Niven, M.D., Director
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Alcohol and Health—An Overview

Alcohol is undoubtedly the most widely used—and abused—drug in America. In 1981, the latest year for which figures are available, the equivalent of 2.77 gallons of absolute (pure) alcohol was sold per person over age 14. Translated into alcoholic beverages, this is about 591 12-ounce cans of beer or 115 bottles (fifths) of table wine or 35 fifths of 80 proof whisky, gin, or vodka.¹ However, Americans are far from equal in their drinking habits. In national surveys, a third of adults report they don't drink. Another third report drinking just over 2 drinks per week (0.21 oz. absolute alcohol/day), and the remaining third report consuming an average of 14 drinks per week. But such averages are deceiving—a tenth of the drinking population consume half the alcoholic beverages sold.

While drinking is nominally restricted to adults, it actually begins much earlier. By ninth grade, more than half (56 percent) of high school seniors responding to an annual 1982 national survey had tried alcohol. But by their senior year, more than 9 out of 10 (93 percent) had done so. Almost all seniors (90 percent) had drunk alcoholic beverages in the previous year. Two out of 5 seniors (41 percent) reported they had had five or more drinks—enough to become drunk—on a single occasion in the 2 weeks prior to the survey. Six percent reported drinking on a daily or near daily basis. High school drinking patterns over the 7 years sampled have changed very little. Male seniors drink more often than females and are more likely to do so daily.

A third (31 percent) of the high school seniors did not disapprove of drinking one or two drinks daily, although two-thirds (65 percent) felt that as many as four or five drinks per day would be harmful.

Defining Alcoholism and Heavy Drinking

Previous editions of this report, like this one, describe in detail many health and social

¹ Assuming 0.6 oz. absolute alcohol = 1.5 oz. 80 proof spirits = 5 oz. table wine (12 percent alcohol) = 12 oz. beer (5 percent alcohol).

consequences of alcohol abuse. These consequences can be summarized most succinctly by saying that there is much evidence—on many grounds—for concluding that alcohol used heavily is costly in human as well as economic terms. New evidence of that cost will be discussed here. But the question of how much must be used to produce a given effect is far more difficult to answer. Research on humans and animals indicates large individual differences in susceptibility to the drug.

When the term "alcoholic," "alcohol abuser," or "problem drinker" is used to designate an alcohol abuser, it must be kept in mind that these designations are somewhat less than precise. What is usually meant by an alcoholic is an individual who has been formally diagnosed as such or has (or has had) serious alcohol problems that meet implicit or explicit criteria of alcoholism, whether or not these have ever been formally diagnosed. It is hoped that, in this report, context will clarify meaning. Similarly, the term "heavy use" sometimes means use that is statistically more frequent than is true of American users generally (as in: "The heaviest using third of the population consume an average of 14 drinks per week."). More often, it is used to indicate a level at which pathological (or adverse behavioral) changes occur more frequently. Some types of episodic or binge drinking may fit neither definition of heavy use, but may nevertheless have serious implications (e.g., drunk driving).

While we have indicated the drink equivalent of a particular amount of absolute alcohol, in reality typical drinks are by no means uniform, and we are forced to rely on rough approximations. As in describing other areas of real life (e.g., smoking habits), we are obliged to accept a certain lack of precision. But within those limits, meaningful observations are possible, as this and earlier reports demonstrate.

Alcoholism and Heredity

Is alcoholism inherited? New evidence concerning genetic susceptibility to alcoholism is

one of the exciting developments in recent research. The belief that alcoholism somehow "runs in families" is an old one. But growing up with an alcoholic parent as a role model has been the traditional explanation for the higher than normal rate of alcoholism in children of alcoholics. Evidence that alcoholics' children, despite having been raised by nonalcoholics, still have a higher rate of alcoholism is persuasive that genetics plays an important role. Moreover, specific biochemical and physiological characteristics in which heredity appears to be important have recently been identified in alcoholics.

A pioneering study of alcoholics who had half-siblings (i.e., brothers or sisters with whom they had only one biological parent in common) found that half-siblings who were also alcoholic were three times more likely to have had an alcoholic natural parent than the nonalcoholic half-siblings.

Research on children adopted at an early age also helps to clarify the role of heredity and environment. Researchers studied a matched sample of natural sons of alcoholics who were then adopted by others, comparing them with the natural sons of nonalcoholics who were also adopted. Both groups had been raised by nonalcoholic adoptive parents. The alcoholics' sons proved to be three times more likely to become alcoholics than the sons of nonalcoholic parents. When the sons of alcoholics were compared with their brothers who had remained in their alcoholic families of origin, the two groups had the same rates of alcoholism, a finding that heredity plays an important role in the development of alcoholism.

Although too few females were considered in the above studies to draw any conclusions about a genetic factor in women alcoholics, a more recent study of adopted women found that those whose natural mothers were alcohol abusers were four times more likely to become alcoholics than those whose mothers were not. Alcoholic fathers did not seem to have the same effect on their daughters. Interpreting this study is complicated by the possible role of the mothers' alcohol use while pregnant in affecting their daughters' later alcohol problems. While it is the first study to suggest a biological predisposition in women alcoholics, there is no way of knowing whether the predisposition is on an in utero developmental basis or a hereditary basis—or both.

A more recent study identified a male limited form of alcoholism where sons of alcoholic fathers are at ninefold higher risk for developing alcoholism themselves.

While the research just described has stressed the importance of genetics in the eventual development of alcoholism, it is certainly not a simple matter of "nature vs. nurture." Other studies suggest that with a genetic susceptibility toward becoming alcoholic, the additional factor of nurturance by an alcoholic parent doubles the chances of the child's becoming alcoholic. But for many, probably most, alcoholics, genetic factors are not apparent in their immediate family's history. However, this does not rule out heredity. Since studies have not been made of multiple generations, there is no way of knowing whether such a biological tendency was present in earlier generations.

Animal research in which systematic inbreeding for several generations has been done clearly indicates the important role of genetics in determining individual susceptibility to many of alcohol's biological effects. Strains of animals can also be selectively bred to prefer drinking alcohol while other strains do not.

Assuming that some inherited tendency increases the likelihood of a person's becoming alcoholic, there are many ways in which it might operate. To name a few: The individual could be more sensitive to certain kinds of stimulus overload, and alcohol may blunt this sensitivity—a mode of self-medication. The central nervous system might neurochemically function in a way that makes it more readily dependent on the drug. It could also account for a more rapid development of tolerance (the need for greater amounts to produce a given effect) and physical dependence. There is some evidence for all of these and other mechanisms as well.

Studies of human twins suggest that as much as half the variation in the rate at which alcohol is eliminated from the body may be genetically determined. The other half is probably related to how alcohol and other drugs are used, diet, and other personal habits. Twin studies also indicate genetically based variation in susceptibility to such medical complications as cirrhosis of the liver and alcohol-induced mental disorders.

Researchers have recently found that one form of the genetically determined liver enzyme aldehyde dehydrogenase is absent in many Orientals. This likely accounts for the higher rate of facial flushing observed after drinking among many of these individuals. The facial flushing is often accompanied by nausea, vomiting, and a drop in blood pressure. These unpleasant symptoms are similar to the reaction that occurs when a person taking

disulfiram consumes alcohol. Such built-in negative responses to alcohol may partially explain why individuals from similar backgrounds often differ so widely in their alcohol consumption.

The certainty that heredity plays a significant role in the development of harmful drinking behaviors and in affecting individual sensitivity to alcohol's injurious effects offers the possibility of better defining biological subtypes of alcohol abusers. If the initial optimism about finding biochemical markers is justified by further research, it may be possible to identify potential alcoholics early—before they develop serious alcohol problems or alcoholism.

Alcohol and the Brain

Alcohol's effects on the brain are among its most seductive—and destructive—properties. Its pleasure-giving potential, along with many of its behavioral hazards, has been recognized since antiquity. Its natural occurrence through spontaneous fermentation probably makes it our most ancient mind-altering substance. But it is only very recently that we are beginning to understand the complexity of alcohol's effects and their underlying neuroanatomical and physiological basis.

Effects of Alcohol on Skilled Performance

Amounts often drunk socially (two or three drinks in an hour) can cause significant impairment in the skills required in driving, flying, and other complex performance. Recent evidence also indicates that time of day can make a difference in how seriously one's performance is affected. Decrements typically persist for many hours after drinking in the evening. Alcohol combined with fatigue has a more pronounced effect than either alone. Recent evidence also suggests that alcohol and tobacco have a greater combined effect on auditory alertness than when used separately.

Overall, there is compelling evidence that alcohol alone and in combination with other commonly used drugs has serious effects on many kinds of performance. The decrements are generally dose-related—the greater the amount consumed, the greater the impairment that results. Further documentation of this can be found in earlier editions of this report.

Alcohol and Brain Damage

Chronic brain injury caused by alcohol is second only to Alzheimer's disease as a known cause of mental deterioration in adults. Many of the symptoms—loss of ability to think abstractly, speech difficulty, and decreased coordination—are similar in the two types of disease. Both groups of patients show brain atrophy (shrinkage) and similar abnormalities in brain electrical functioning. However, Alzheimer's disease is invariably progressive, whereas alcoholic mental deterioration is not. If the patient stops drinking, the deterioration is arrested and substantial recovery can occur.

(Even in alcoholics without obvious evidence of mental deterioration, CAT scans—computerized techniques for visualizing and measuring the brain—have revealed abnormalities). Another advanced computerized method (average evoked response or AER) measures the response of the brain to light and sound stimulation. Recent work indicates that alcoholics respond differently than do non-alcoholic persons. Alcoholics are more sensitive to the stimulation. One theory is that their sensitivity leads them to drink in an effort to reduce their stimulus overload. Possible evidence of this is the recent discovery that those with family histories of alcohol use—even though they were not themselves alcoholics—were different in their AERs from those who had no alcoholics in the family. If this finding is confirmed, it may provide another means for early identification of those at risk of becoming alcoholics.

Other advances in brain measurement have found that alcohol alters brain activity in parts of the brain involved in memory, emotion, and higher level functioning. Microscopic study of the brains of patients who have died with Korsakoff's syndrome, an alcohol-induced disease characterized by severe memory loss and impaired learning, has disclosed brain injury near nerve pathways involved in these functions. These brain lesions are very like those found in patients who have died of acute vitamin B₁ (thiamine) deficiency, possibly indicating that a similar vitamin deficiency occurs after chronic heavy alcohol use.

Debate took place in the past over whether the brain injury found in alcoholics is the result of alcohol as such or of the dietary deficiencies that are so common in advanced alcoholism. It is now known that alcohol itself injures brain tissue, although the combination of excessive drinking and poor diet may accelerate the process.

Neurochemistry--the study of minute quantities of chemicals in the brain that govern how nerve cells transmit their messages--is also advancing our understanding of alcohol. But the effects of the drug on these neurotransmitters is not simple. They seem to vary depending on the part of the brain involved as well as the dose.

Dose-related opposing effects have been found on the neurotransmitter norepinephrine. At low doses alcohol increases norepinephrine release, but at higher doses release of the chemical is inhibited. This seems to correspond to the everyday observation that alcohol is initially stimulating in small amounts but has a depressant effect as more is consumed.

Another neurotransmitter, GABA, inhibits neuronal firing and can control the tendency toward certain types of convulsions. Animal research indicates that animals chronically given alcohol have a reduced number of receptor sites for GABA. This suggests that GABA may play a role in the seizures often seen in alcoholics.

Other research, on the neurocellular level, is probing alcohol's effects on cell membranes. It may explain how tolerance for alcohol--its decreased effect when used regularly--develops and the neurocellular basis for physical dependence.

Earlier research has found that alcohol can affect parts of the brain involved in reward or pleasure. There is now heightened interest in other mechanisms by which alcohol and similar dependency-producing drugs operate. Naturally occurring opiate-like compounds have been found in the brain. Both these chemicals and externally derived opiates seem to have similar sites of action--the opiate receptors. Chronic doses of alcohol in test animals may alter the function of these opiate receptors. This finding has led scientists to speculate that alcohol and other psychoactive drugs may act similarly in the brain. Some support for this theory comes from the observation that narcotic antagonists--drugs that can block the effects of an opiate by occupying opiate receptor sites--also block some, but not all, of alcohol's effects.

Another provocative preliminary hypothesis is that a group of chemicals called tetrahydroisoquinolines, or TIQs, may play a role in habitual heavy drinking. TIQs are formed when alcohol is metabolized. In some animal experiments, when these chemicals were injected into the brains of animals, their preference for alcohol increased significantly.

All of these preliminary neurophysiological observations fall short of adequately explaining the action of alcohol. Additional research is needed to sort out some of these conflicting--and still clearly incomplete--explanations.

Other Health Hazards

Even a casual observer notices some of the effects of heavy drinking on the brain and the complex behavior it controls. But there are other, more insidious, consequences for other parts of the body as well. And many of these are not apparent even to knowledgeable drinkers.

The Digestive Tract

Cancers of the mouth, tongue, pharynx, and esophagus are more common in alcoholics than in nonalcoholics. While the exact cause or causes are unknown, alcohol's direct irritation of mucous membranes is well established. This, combined with the known carcinogenic properties of tobacco so often smoked by heavy drinkers, is likely to be jointly responsible. Vitamin A and B deficiencies because of poor diet and/or alcohol's interference with the vitamins' absorption may also play a role. Animals that are vitamin A deficient are known to be more susceptible to skin, mouth, and esophageal cancers.

Alcohol alters stomach acid secretion, which along with its irritating gastric lining probably accounts for the stomach problems so common in alcoholics. It weakens the stomach wall, making it more susceptible to bleeding. The use of aspirin by drinkers may increase the likelihood of bleeding from the already weakened wall, sometimes leading to hemorrhage.

Alcohol has multiple effects in the intestines. It impairs vitamin absorption, is an irritant, and results in increased gastrointestinal motility. This often leads to diarrhea in heavy drinkers, which may contribute to their poor nutritional state.

Chronic pancreatitis is common in alcoholics. Seventy-five percent of the cases in the United States are believed to occur in alcoholics. Although pancreatitis often results in abdominal pain and vomiting, half the alcoholic population show alteration in pancreatic function without obvious symptoms. Recent evidence suggests that alcoholics may have

more of a specific genetically determined antigen—a chemical that activates the immune system, which suggests a genetic mechanism behind the vulnerability to this disorder. Pancreatic cancer is also twice as frequent in alcoholics—75 percent of those who contract the disease are moderate to heavy drinkers.

Perhaps the best known effects of excessive alcohol use are those on the liver. These range from fatty liver, the most common—and reversible—problem, to cirrhosis, which is life-endangering and irreversible. While it was once thought that liver damage could be averted by an adequate diet, it is now known that damage occurs regardless of how nourishing the heavy drinker's diet is. Alcoholic hepatitis also results from heavy drinking. The inflammation and later destruction of liver cells from this disease kills 10 to 30 percent of those who develop it. Alcoholic cirrhosis is the most dangerous consequence to the liver from drinking, causing at least 11,000 deaths annually in the United States.

Alcohol and the Heart

Alcohol when used heavily may have detrimental effects on the heart. Cardiac myopathy—damage to the heart muscle—often occurs after 10 or more years of heavy drinking. Symptoms of this disorder range from chronic shortness of breath and ankle swelling to heart failure. Degenerative changes in heart muscle cells can be seen with the electron microscope. If the damage is not too extensive, recovery is possible, although it may require 5 or more years of abstinence.

Alcohol also interferes with the heart's ability to contract. Heart beat irregularities (cardiac arrhythmias), which can lead to heart failure, are common in alcoholics. Two ounces of whisky drunk by an individual who already has heart disease can suppress the heart's pacemaking abilities.

Effects on Other Muscles

The effects of alcohol on muscle tissue are not confined to the heart. Muscular weakness and severe pain from muscle cramping are fairly common in heavy imbibers. Mild symptoms of these myopathies, which may not be severe enough to come to medical attention, occur in about a third of alcoholics. Alcohol also affects involuntary smooth muscle contractions.

Alcohol and the Blood

Excessive drinking causes blood abnormalities. Both enlarged red blood cells and anemia are produced. These are probably the combined result of vitamin deficiency, alterations in fat metabolism (because of liver damage), and other still unknown or poorly understood mechanisms. Abnormally large cells are so common (found in 90 percent of alcoholics) that a measure of this, the mean corpuscular volume or MCV, has been suggested as a means of detecting alcoholism. Four or more months of abstinence may be required before the MCV returns to normal.

Many alcoholics also have reduced white blood cell counts. These cells play an important role in the body's immune response. A diminished immune response has now been reported in alcoholics. The higher rates of infectious disease common in this group are probably attributable to this lowered resistance.

Alcohol and Sexuality

Alcohol interferes with male sexual functioning. That was known in Shakespeare's time—and probably before. But recent research provides both confirmation and an explanation. Impotence and/or reduced sexual drive are found in from 70 to 80 percent of alcoholics. The same percentages have been reported to show testicular atrophy and infertility. Alcoholics have much lower levels of the principal male hormone testosterone than do nonalcoholics, along with increased levels of female hormones. Because of this, 50 percent of male alcoholics develop feminine pubic hair patterns, and 20 percent have breast enlargement.

Like other effects of alcohol, these changes in sexuality were once believed to arise from liver injury. More recent evidence clearly indicates that alcohol reduces testosterone levels and causes direct gonadal injury. It also affects sexual functioning indirectly through its impact on the brain's hypothalamus and the pituitary, the so-called master gland. Recent research suggests that the increase in estrogen levels found in alcoholic men is also partly due to adrenal changes. The adrenal glands produce estrogen precursors—chemicals from which estrogens are later formed, although the mechanisms involved are not yet known.

The alcoholic female's sexuality is also affected by her drinking. Severe gonadal fail-

ure—an inability to produce adequate quantities of female hormones—leads to loss of such secondary sexual characteristics as fat deposits in the breasts and other parts of the body, reduced (or absent) menstruation, and infertility. As with men, the control of hormone production by the brain and the pituitary gland is affected.

Alcohol and Pregnancy

Clinical and experimental research over the past decade underscores the threat to the developing fetus that maternal alcohol misuse represents. The term fetal alcohol syndrome (FAS), first used 10 years ago to describe a set of symptoms in the newborn infant believed to be caused by heavy maternal alcohol abuse, has recently been more carefully standardized. The new standard demands that three kinds of characteristics be found in the newborn to justify being classified as a case of FAS: (1) growth retardation before or after birth, (2) abnormal features of the face and head such as unusually small head circumference and/or flattening of facial features, and (3) evidence of central nervous system abnormality (e.g., mental retardation, abnormal behavior, etc.).

Other birth anomalies are regarded as alcohol related if there is evidence of heavy or frequent maternal drinking during pregnancy and if the specific anomalies occur in greater frequency, after control for confounding variables, among the children of heavier than lighter drinkers or abstainers. Some studies suggest a risk for outcomes including decreased birth weight and increased spontaneous abortion at levels of alcohol consumption as low as two drinks per day, although some investigators question whether these self-reported consumption levels are not in actuality higher. Isolated instances of heavy drinking may also pose significant hazard.

Women's alcohol use during pregnancy has been described by one research group as "the most frequent known teratogenic cause of mental retardation in the Western World." However, the prevalence of fetal alcohol syndrome or of other alcohol-related birth defects is not easy to specify. Moreover, since heavy drinkers often underestimate (or underreport) levels of drinking, it is difficult to be certain how much alcohol consumption is required, at what point or points in pregnancy, to produce abnormalities. Because developing fetuses probably differ in their sensitivity to alcohol as well, the pattern of alcohol use that

will result in congenital anomalies cannot be specified with precision.

One somewhat encouraging finding is that many women who usually drink heavily markedly decrease their drinking while pregnant. Such a reduction might be expected to decrease the risk of congenital defects.

Current estimates are that FAS affects somewhere between 1 and 3 births per 1,000, but other alcohol-related birth defects probably add significantly to that number. A recent calculation suggests that 5 percent of total birth defects may be alcohol related.

Animal research, which can be more carefully controlled than that on humans, leaves little doubt that alcohol ingestion during pregnancy has serious consequences. Birth defects closely resembling those found in humans have been produced in many animal species. Scientists who administered two doses of alcohol to mice on one day early in pregnancy (an animal model resembling human binge drinking) found abnormal facial features in offspring like those reported in human FAS. Extensive animal—and human—findings amply justify discouraging women from drinking while pregnant.

Lowered birth weight is the most consistent result of alcohol use during pregnancy. Since low birth weight is associated with more frequent neonatal deaths and is frequently associated with mental retardation and neurological defects, this finding is important.

Several studies have found low IQ and motor development scores related to maternal alcohol use. Most recently (1983), 4-year-olds whose mothers drank during their gestation were found to have decreased attention spans and slow reaction times. Children with FAS and animals prenatally exposed to alcohol have been found to show hyperactivity.

Paternal Drinking

Although it is known that long-term alcohol use by men can lead to reduced sperm counts, decreased sperm motility, and higher rates of sperm abnormalities, there is no evidence from either animal or human studies that heavy alcohol use leads to congenital defects in the children of such men.

Adverse Social Consequences

Alcohol abuse plays a role in many kinds of social problems. In some—traffic accidents,

for example--what is known about alcohol's effects on complex skills makes it reasonable to ascribe a causal role to the drug. In other problems--child and spouse abuse, for example--excessive alcohol use is probably not the primary cause although it may contribute significantly, perhaps by reducing inhibitions that might otherwise curb expression of anger.

Traffic Accidents

Traffic accidents are the fifth leading cause of death in the United States and its leading cause among those under age 35. In the latest year for which data exist (1981), such tragedies killed 49,000 and permanently disabled another 150,000 persons. The consensus is clear--alcohol is responsible for up to half of these tragedies. A review of 23 studies found a third or more of drivers in fatal accidents had blood alcohol concentrations (BAC) above the usual legal standard for drunk driving of 0.10 percent. Five or six drinks consumed in the 2 hours before driving can cause this level in a 155-pound person. Drivers with blood alcohol concentrations above 0.10 percent have been estimated to be 3 to 15 times more likely to have a fatal accident than nondrinking drivers. Other estimates have concluded that even lower BAC levels--from 0.05 to 0.099 percent--increase the risk from 1.25 to 3.25 times. This is the level that results when a 155-pound person has had three to four drinks or a 120-pound person as few as two to three drinks within 2 hours. Unfortunately, many people are not aware of these risks. A recent Massachusetts survey found that 17 percent of respondents believed the fiction that you can have six or more drinks without becoming too drunk to drive. (Six drinks in a 155-pound person would result in a BAC above 0.10 percent, even if the drinks were consumed during a 3-hour period before driving.)

Pedestrians who have been drinking also have a much higher risk of being injured or killed in a traffic accident than do nondrinkers. In one recent study, more than a third (36 percent) of injured pedestrians and nearly half (45 percent) of those who died from being struck by a motor vehicle had blood alcohol concentrations above 0.10 percent. Only 13 percent of a control sample of uninjured pedestrians passing the same accident sites at a similar time had that high a BAC. As with motorists, the higher the blood level, the higher the risk of accident involvement. Compared with that of the nondrinking pedestrian,

the risk of being hit was twice as high at blood levels above 0.10 percent, five times higher if the BAC was above 0.20 percent.

Industrial Accidents

Less is known about the relationship of alcohol to industrial accidents than to traffic accidents, although such a connection is predictable. A 1982 Maryland study of workers who died in job-related accidents found that 11 percent had BACs above 0.08 percent. While this may not be nationally representative, it indicates a relationship that should be further examined.

Other Accidents

It is hardly surprising that the heavy imbibor is at greater risk in other types of accidents as well. Accident statistics are consistent with this expectation. Alcoholics are 5 to 13 times more likely to die from falls than nonalcoholics. Alcoholics are also much more likely to die in fires--a tenfold increase over nonalcoholics. Heavy drinkers had 2.7 times the accidental death rate (omitting traffic accidents) of other Kaiser-Permanente members during a 10-year followup period. While such statistics do not prove that alcohol played a direct role in the particular hazard, the presumption cannot be dismissed.

Alcohol and Crime

Although many statistical associations have been made between alcohol use and criminal behavior, a direct causal link is more tenuous. One of the earlier homicide studies (1958) found that either the murderer or the victim had been drinking in two-thirds of the slayings reviewed. Subsequent research has also found that a majority of murderers, their victims, or both had been drinking prior to the crime. Whatever the role of drinking in such studies, it is confounded by such factors as a failure to control for age, sex, and race, all of which are related to both violence and drinking. The most that can be concluded is that alcohol is a contributing cause but is probably only one of several factors involved.

Rape

The role of alcohol in rape is even more difficult to assess. Not only is rape very underreported--authorities agree that only a

minority of attacks are reported—but intoxication may be offered as an excuse or extenuating circumstance by the rapist. Studies of rapists, however, indicate that they often drank heavily prior to the crime, and that their rate of alcoholism is two to three times that of the general population. Whether alcohol plays a disinhibiting role, or serves as a justification for the behavior, or plays some other part is unclear.

Alcohol and the Family

Alcohol abuse undoubtedly affects family life. It is noteworthy that a third of persons interviewed in a 1982 survey felt that alcohol caused problems in their families. But the precise ways in which it does so are more difficult to document. The rate of separation and divorce among alcoholics is seven times that of the general population. Two out of five domestic relations court cases involve alcohol.

While few doubt that heavy drinking plays some role in family violence and the abuse of spouses and children, just how and to what extent is again hard to specify. Most studies report that alcoholism or excessive drinking is involved in about half the cases of spouse abuse. As with rape, drinking may be used as an excuse for the violence involved. Early reviews of child abuse have described associations between it and alcohol in as many as 38 percent of cases. However, a recent review has concluded that alcohol involvement in child abuse is no higher than in the general population.

A 1983 review of the effects of parental alcoholism on children indicates that they are more likely to have school problems and to display antisocial behavior. Other studies report that such children have less self-esteem, more anxiety symptoms, more aggression, and more psychosomatic symptoms.

Alcohol and Suicide

Given the high rate of depression in alcoholics, it is not surprising that they have a high rate of suicide. But here, too, it is unclear whether the depression contributes to the drinking and suicide, or the alcohol use causes the depression and ensuing self-destruction. Nevertheless, as the Fourth Special Report noted, as high as four out of five of those who attempt suicide had been drinking at the time. Alcoholics commit suicide from 6 to 15 times more frequently than the general population.

Economic Costs

General agreement exists that alcohol abuse results in lost work productivity and an increase in industrial accidents. In one study of railroad companies, problem drinkers had twice the absenteeism of other employees. Much less is known about the costs associated with episodic drinking on or off the job as it affects work performance.

Estimating the cost of alcohol abuse to American society is difficult. Many of the variables that should be taken into account are simply not known with sufficient accuracy. But even making very conservative assumptions about these factors, a recent calculation places the annual national loss at about \$49.4 billion. The bulk of this amount—\$36.8 billion—represents the value of lost productivity. The cost of various kinds of medical care for alcohol-related illness and injury totals \$6.4 billion. The remaining \$6.2 billion includes such added costs as traffic accidents, fires, crime, and other miscellaneous losses attributable to alcohol.

Perhaps the highest cost is the incalculable pain and misery associated with alcohol abuse for the abusers, their families, and the larger society. There is no disagreement that those costs are profound.

Treatment Trends

A basic problem in treatment is deciding just who is an alcoholic and just what is alcohol abuse. Failure to agree on definitions is the basis for much of the confusion regarding the exact prevalence of alcohol abuse and its implications. The latest revision of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-III) represents an important advance in conceptual and diagnostic clarity. Using a multi-dimensional matrix, it takes into account alcohol-induced symptoms, psychosocial stress, the patient's level of adaptive functioning, and related personality disorders. It is hoped that the new manual will ensure uniform standards that will in turn permit increased comparability of treatment and research findings.

A related development in diagnosis is the improvement in relevant sections of the International Classification of Diseases (ICD) resulting from collaborative effort between the U.S. Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) and the

World Health Organization (WHO). Central to the WHO approach is the concept of a dependence syndrome, an interrelated cluster of cognitive, behavioral, and physiological symptoms.

One of the most promising new developments is the emergence of a group of biochemical markers to assist in identifying alcoholics. While each of these tests has limitations, several studies have now found that a profile of multiple blood tests can quite accurately differentiate alcoholics from non-alcoholics.

The new Munich Alcoholism Test uses both clinical signs and symptoms noted by the examining physician. When combined with self-reporting instruments and the biological marker approach, such testing promises to more usefully discriminate between different types of alcohol-dependent patients. In preliminary trials of this approach, psychosocial problems were most evident in young abusers, with clinical signs and symptoms more common in older alcoholics.

Early Intervention

Early identification through the sophisticated techniques now available offers the possibility of early intervention. And there is now increasing evidence that it works. Even a 35-minute counseling session in a general hospital setting has been found to reduce drinking in a significant number of newly identified problem drinkers who were followed up for 1 year.

A recent Swedish study using a biochemical marker—elevated GGTP scores—to identify heavy drinkers has also found that early intervention can be valuable. Those identified were told their GGTP results and their significance. They were then repeatedly retested, given feedback on their progress, and encouraged to moderate their drinking. The group had lower work absenteeism, fewer hospitalizations, and a lower death rate during the 6-year followup.

In France, a combination of two biochemical tests and a clinical examination is now being used on a nationwide scale to detect early alcohol abuse in industrial, health care, and court settings. When a problem is found, the individual is referred to local early intervention clinics for care. The system, which has yet to be evaluated, illustrates the feasibility of large-scale intervention.

Work-based alcoholism treatment intervention as an alternative to disciplining the

worker has also shown definite promise. Several studies have found that it works as well as voluntarily sought treatment when measured by abstinence and improved job performance.

Detoxification

Detoxification—the process of withdrawing the alcohol-dependent patient from alcohol and controlling his or her withdrawal symptoms—has traditionally been done in a hospital setting. In recent years, partly because of the high cost of in-hospital detoxification, other settings have been tried. Although these alternatives have not been systematically evaluated, a review of the evidence suggests that detoxification can be done humanely, safely, and efficiently in nonhospital settings. It may, however, be important to develop clear criteria of what types of alcohol abusers are most successfully treated by the new alternatives.

Treatment Benefits

A number of studies testing the feasibility of treating alcoholics through medical insurance plans have now been completed. All have found that alcoholics and their families are initially more expensive to treat, as they require more care than those without alcohol problems. However, following treatment for alcoholism, demand for services drops, and the treatment is unquestionably cost-effective in reducing total medical expenses. The median reduction in sick days and accident benefits was 40 percent in one review of 12 programs.

Research comparing inpatient and outpatient treatment settings has not found significant differences in treatment outcome. Since patients are not usually randomly assigned to each type of setting, it is not yet known whether differences in the types of patients normally sent to these two settings are important. (Seriously ill patients with medical complications may still be more appropriately treated in a hospital.)

Client-Program Matching

The problem of developing programs suited to special populations such as women, ethnic minorities, and the elderly has become important. As this and earlier reports indicate, groups such as Native Americans have much higher rates of illness and death from alcohol-related illness than does the general population. It is critical that treatment programs

be designed to overcome cultural, economic, and other barriers that deter some groups from seeking treatment. A survey of California facilities found that women alcoholics were less likely to seek treatment if programs lacked child care and aftercare arrangements and professional staff. More and more programs are being designed to meet these and other needs of special populations.

Treatment Approaches

Several experimental approaches to treatment have been extensively evaluated since the Fourth Special Report. Aversion therapy, which links the sight, smell, and taste of alcohol with a nausea-producing drug, appears to be effective with stable, well-motivated alcoholics, while the same approach substituting electrical shock appears not to be. A related technique—covert sensitization—that pairs verbally induced nausea with imagined alcohol consumption produces greater improvement than insight-oriented therapy, based on a 4-year followup.

A recent study of disulfiram (Antabuse) not discussed in the Fourth Special Report found the drug slightly more effective in producing abstinence after a year than a vitamin B preparation. Another study found that additional training in role playing, communication, and stimulus control along with the use of disulfiram was effective with married clients, but not with the unmarried. Further training in drink refusal, social skills, muscle relaxation, and job and recreational counseling for the single group raised their abstinence levels, however. Closely supervised use of disulfiram in an industrial setting was effective in reducing absenteeism.

Traditional psychotherapy has been found not to be generally effective with alcoholics, but may be useful with some types. Family therapy is also being tried, although it has not yet been fully evaluated.

Early findings suggested that some "definitely alcoholic" clients were capable of drinking moderately 6 months after treatment and up to a year later. However, a followup 4 years later has found that those who were abstinent at 18 months were less likely to have drinking problems at 4 years than those who drank moderately at 18 months.

Patients affiliated with such aftercare groups as halfway houses and Alcoholics Anonymous have been found to have better therapeutic outcomes than those who remain unaffiliated.

Prevention

Alcohol abuse prevention is becoming more important as awareness grows that alcohol problems are present in the entire drinking population. While problems occur at lower rates in groups that drink less, they are rarely totally absent.

There is little question that alcohol education programs can increase knowledge concerning alcohol and may also alter attitudes toward the substance. But the question of the extent to which they modify actual drinking behavior is not so easily answered. Even when such changes have been assessed—and most programs have not tried to do so—they are difficult to interpret. Changes in economic conditions, minimum ages at which drinking is permitted, and law enforcement policy are all likely to overshadow program effects.

A 1982 study found that teaching 7th through 10th graders about alcohol improved their knowledge and also reduced their use. A recent review of prevention programs emphasizes that improved decisionmaking is likely to lead to a reduction in pro-drinking attitudes, although it is overly optimistic to expect significant use reduction based on such limited interventions.

Nontraditional programs emphasizing interactive learning, which involves students in the actual design of the prevention program, seem to be most successful in altering attitudes and possibly drinking behavior.

Intervention—between 6th and 9th grades—to teach youngsters to develop resistance to later use of alcohol (and tobacco) is a promising prevention strategy, although it has not yet been followed up with longitudinal studies.

Because the college years are important in developing long-term drinking habits, a number of programs have been directed toward these students. Improvements in knowledge occur and there is evidence that attitudes shift, but no changes in actual drinking have been found.

Increased sophistication is evident in programs tailored to meet the needs of women (especially during pregnancy), minorities, and other special populations such as the elderly. These new programs need to be tested for effectiveness.

Mass media campaigns are probably too weak an intervention to significantly alter drinking behavior unless they are closely coordinated with local education and training efforts. However, they can be useful in generating heightened interest in alcohol prob-

lems and in mobilizing community resources to deal with the problem.

Lowering alcohol consumption levels is associated with reduction in a number of adverse effects of drinking such as alcohol-related traffic accidents (especially by youth) and diseases such as cirrhosis of the liver. Some effective efforts, documented through research, are:

- Increasing taxes on alcoholic beverages
- Increasing the legal age for purchase of alcoholic beverages
- Altering the number and location of outlets for purchase of alcoholic beverages.

Future Directions

New directions are evident in many parts of this report. Among the most promising are improved early identification of potential problem drinkers and alcoholics and expanding present basic knowledge of just how and why alcohol abuse is so destructive. Basic research may result in developing strategies that reduce the likelihood of those who are geneti-

cally more vulnerable becoming alcohol dependent. It may also enable them to drink moderately with less hazard.

New evidence of the serious threat alcohol poses to the developing fetus argues persuasively for enhanced efforts at preventing maternal drinking by making women more fully aware of the risk. The evidence that many individuals are many times more vulnerable to alcohol's destructiveness demands that better prevention and treatment be developed for those at high risk. Better targeted prevention efforts are also implicit in early detection of genetic vulnerability to alcohol through the use of biological markers.

Treatment offers many opportunities for increased innovation. One is to tailor programs to fit the needs of different types of clients. Many approaches that have been used with reported success have not been adequately evaluated. The cost-effectiveness of early intervention in health maintenance organizations and industry argues for more aggressive efforts by both.

Alcohol problems have plagued most societies throughout history. But never before have we had as much basic knowledge as we now possess, with the promise of still greater understanding to come.

Chapter I

Epidemiology of Alcohol Abuse and Alcoholism

Epidemiological research is useful in identifying many of the social, environmental, behavioral, and biological factors associated with alcoholism and alcohol abuse. New insights and understanding of these factors are complicated by the lack of standard and uniformly applied definitions. Terms such as alcohol abuse, alcohol misuse, problem drinking, alcoholism, and alcohol dependence are often used interchangeably. The development of uniform operational definitions should lead to continued advances in the epidemiology of alcohol-related conditions. Meanwhile, researchers and policymakers must rely on current terminology and the best data available to understand the problems associated with alcohol abuse and alcoholism. The data presented here indicate the scope and magnitude of alcohol abuse and alcoholism in this country and provide a framework for discussing the effects of the excessive consumption of alcoholic beverages.

Consumption of Alcoholic Beverages Measured by Alcohol Sales

Sales of alcoholic beverages are often used as an indirect measure of alcohol consumption. But per capita consumption figures based on sales and excise tax data do not take into account the amount of alcohol consumed in one State but purchased in another, the amount of alcohol purchased but not consumed in any given year, sales of alcohol that go unreported, and the consumption of illegally produced alcohol (e.g., moonshine).

Figure 1 shows the estimated per capita alcohol sales for beer, wine, distilled spirits, and the three beverage types combined. The estimates are expressed in gallons of absolute alcohol per person, per year, for the United States. For statistical purposes, this population is defined as the estimated population 15 years of age or older for years up through

1969, and 14 years of age or older for years 1970 through 1981. For 1981, the average estimated consumption is 2.77 gallons per year or approximately 1 ounce of absolute alcohol per day for each person in this population. Fifty-one percent of the absolute alcohol consumed in 1981 was beer (1.40 gallons per person), 36 percent distilled spirits (1.01 gallons per person), and 13 percent wine (0.35 gallons per person).

Figure 2 displays estimated per capita consumption in 1981 for the three alcoholic beverage types combined for each State and the District of Columbia (NIAAA 1982a, d, e). This map indicates that substantial variation exists across States in quantity of alcohol sold. Statistical data for each State also indicate considerable variation in the relative sales of each type of alcoholic beverage. A number of factors may influence the variation in beverage sales and apparent per capita consumption across States, including:

- Difference in State taxes for each type of beverage
- Difference in the legal drinking age within each State
- Differences in State legislation regarding the location, type of outlets, and hours of operation for selling alcoholic beverages
- Differences in cultural and social habits across geographic regions.

U.S. alcohol per capita sales have increased approximately 35 percent since the early 1960s, but only 7 percent since 1971. While estimated per capita sales of beer rose 22 percent and of wine rose 13 percent, sales of distilled spirits have dropped 10 percent since 1971. Considerable variation in sales rates occurs among States, e.g., a decrease of 17 percent in the District of Columbia and an increase of 36 percent in Hawaii. These data

must be interpreted cautiously, however, because the States are differentially affected by such factors as drinking age, degree of urbanization, tourism, and out-of-State purchasers.

Surveys of Self-Reported Consumption Among Adults

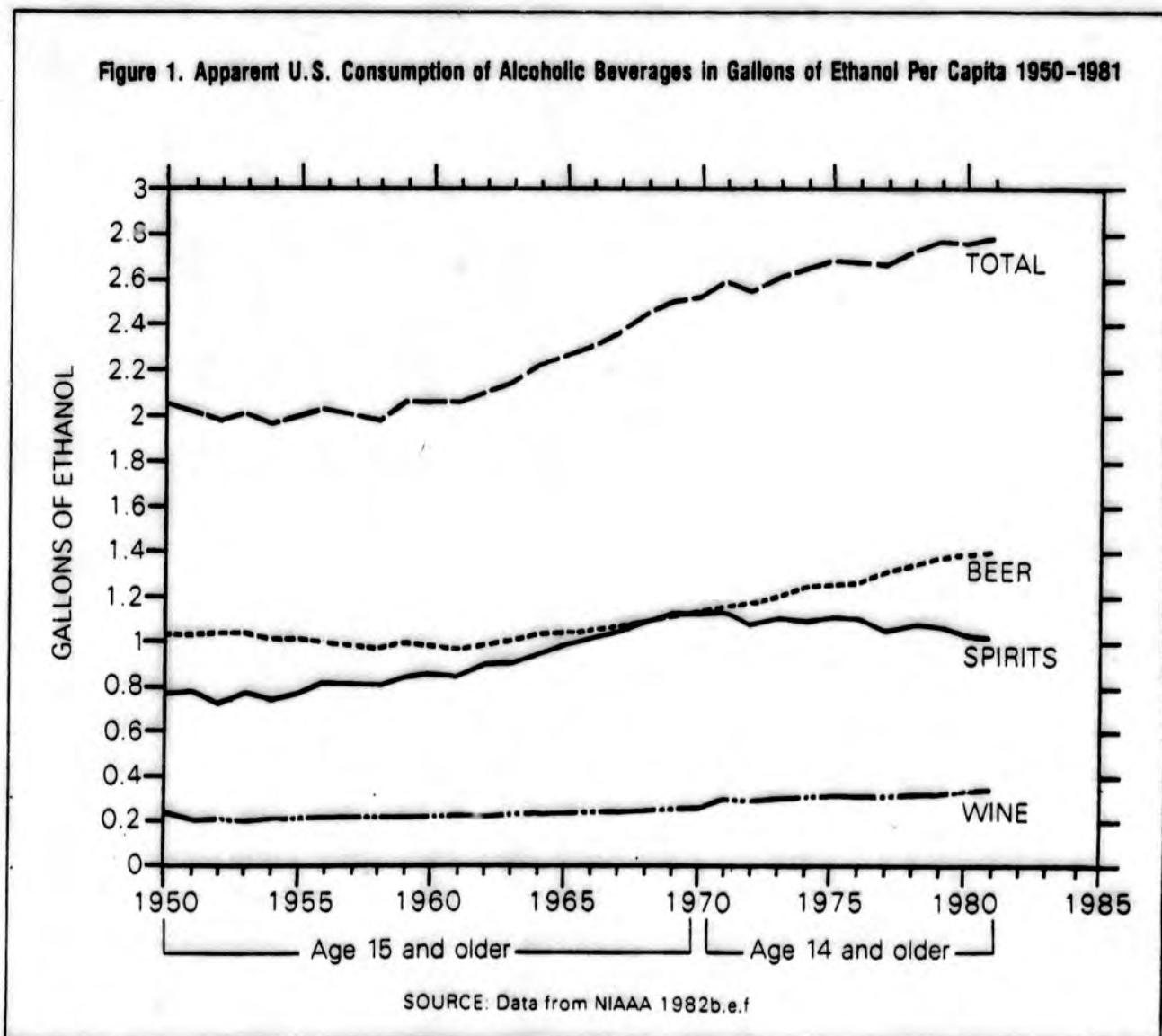
The National Institute on Alcohol Abuse and Alcoholism (NIAAA) has sponsored a series of national surveys that characterize the self-reported drinking patterns of the U.S. adult population. Results across these surveys conducted in the 1970s are similar. Approximately one-third of the adult population described themselves as alcohol abstainers (consumed not a single drink in the year prior to the survey). Another third described themselves as

light drinkers (consuming up to 0.21 ounces of absolute alcohol per day on the average), while the remaining third were moderate (0.22-0.99 ounces) or heavier drinkers (1.0 ounces or more), the latter (heavier drinkers) consuming the equivalent of approximately two drinks or more every day.

Men drink significantly more than women, and younger adults more than older adults (see figure 3). The most recent national survey (1979) confirms these sex and age variations. In addition, the 1979 data indicate that abstaining from alcohol is less common in the New England and Pacific regions and more common in the South, and that the proportion of abstainers falls as income rises and years of schooling increase.

Findings from other national household surveys also confirm that alcohol consumption

Figure 1. Apparent U.S. Consumption of Alcoholic Beverages in Gallons of Ethanol Per Capita 1950-1981



is greater among younger adults than among older adults. During the years 1974 through 1982, the annual prevalence of alcohol use among younger adults, 18 to 25 years old, was approximately 22 percent greater than among adults 26 years and older. Overall, the annual prevalence of alcohol use reveals a slight but gradual increase between 1974 and 1982.

The survey data suggest that a substantial proportion of all alcohol is consumed by a relatively small fraction of the total drinking population (see figure 4). This figure shows that 50 percent of the alcohol is consumed by 10 percent of the population.

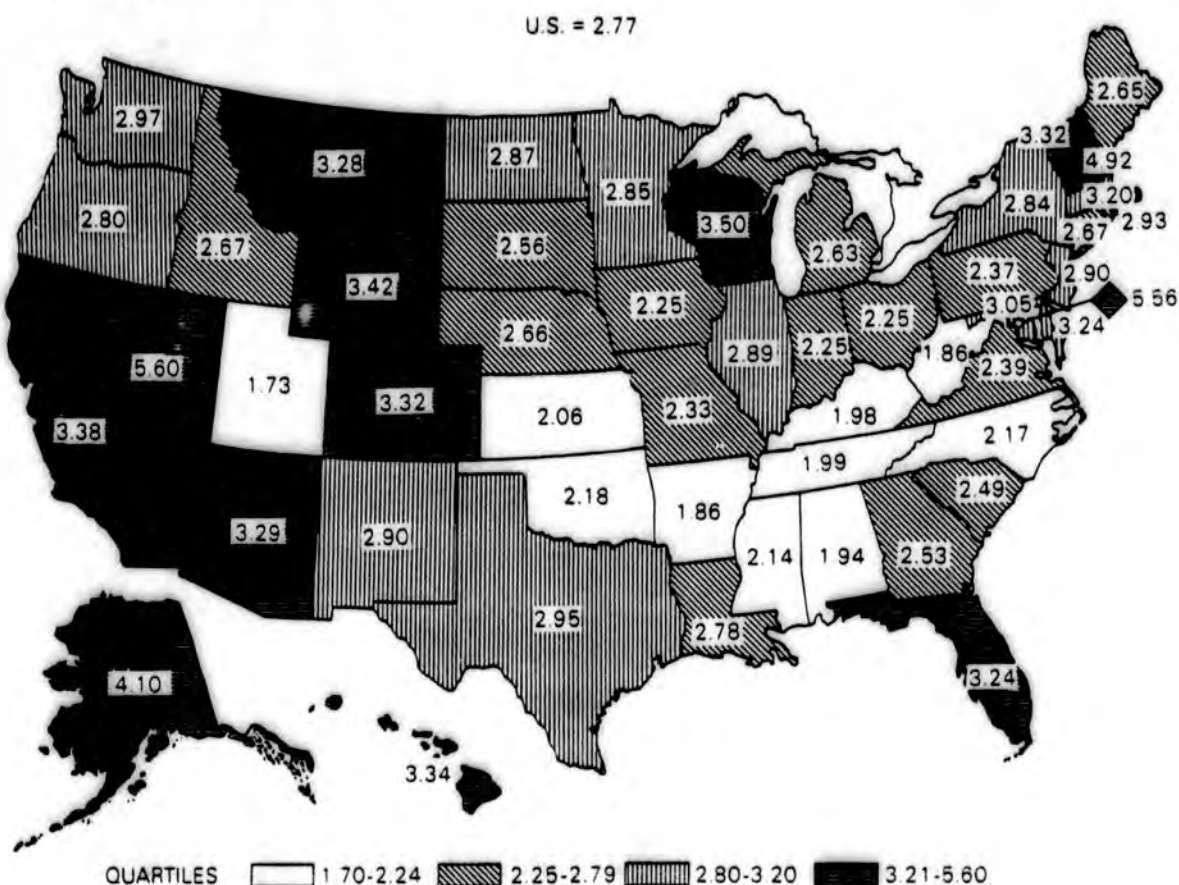
Surveys of Alcohol Consumption Among Youth and Young Adults

NIAAA has also sponsored a number of national surveys to assess alcohol consumption

patterns and trends among the youth and young adult population. Two NIAAA surveys in 1974 and 1978 (Rachal et al. 1975, 1980) were followed in 1980 by the NIAAA-sponsored Gallup Youth Survey. In addition, the National Institute on Drug Abuse (NIDA) sponsors an annual survey of high school seniors and a biennial household survey (NIDA 1983a) that collects data on alcohol consumption of the youth population. These surveys showed a small decrease in the drinking levels among 16- to 18-year-olds for the years 1974, 1978, and 1980 (Zucker and Harford in press). The NIAAA data reveal some increase in the number of abstainers and a slight decrease in the proportion of moderate and heavy drinkers between the years 1978 and 1980.

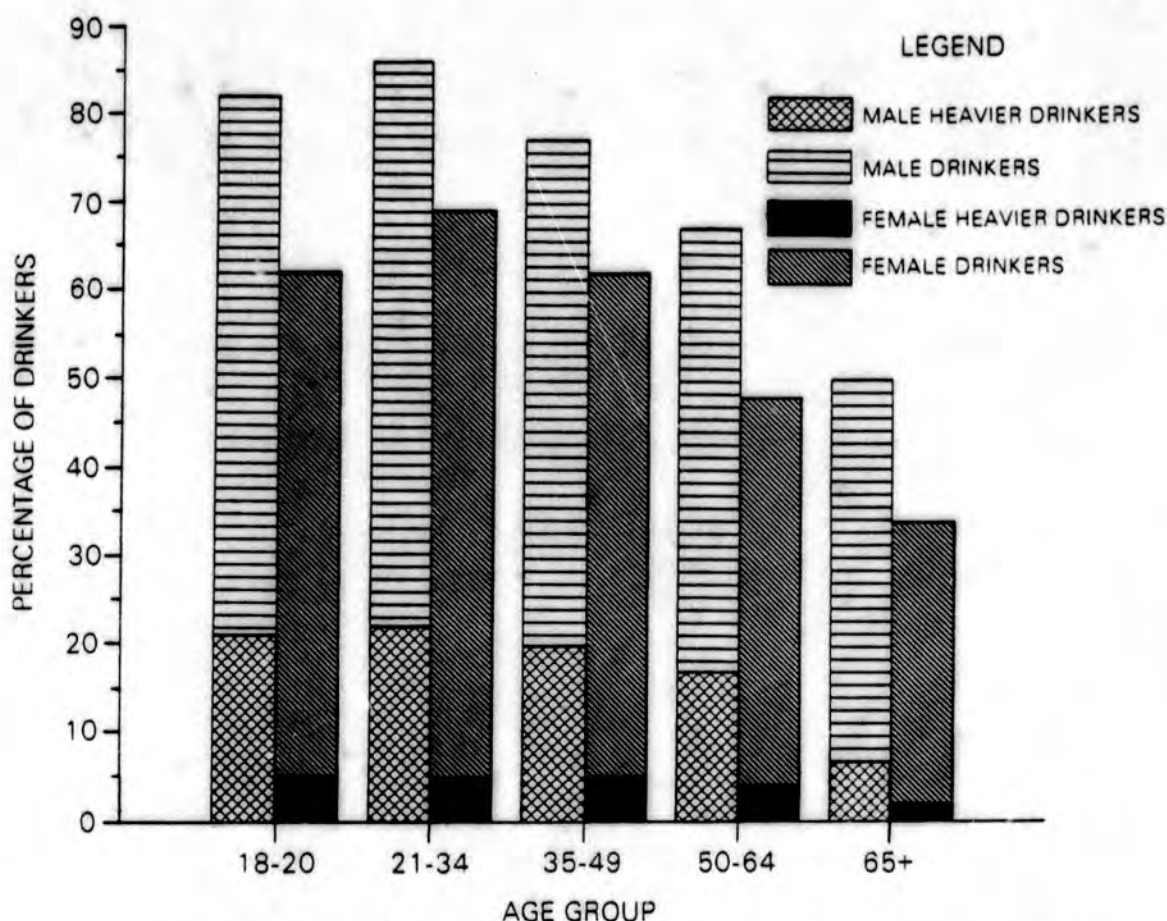
Similar conclusions can be drawn from the national surveys of high school seniors sponsored by NIDA since 1975 (NIDA 1983b). In a sample of approximately 130 public and pri-

Figure 2. Apparent Consumption of Ethanol from All Alcoholic Beverages in U.S. Gallons Per Capita of the Population Age 14 and Older, 1981



SOURCE: NIAAA 1982a,d,e.

Figure 3. Percentage of Drinkers by Age Group and Sex Averaged for the Period 1971-1976



SOURCE: Data from Clark, W.B. and Midanik, L. Alcohol Use and Alcohol Problems Among U.S. Adults: Results of the 1979 National Survey. Alcohol and Health Monograph No. 1 NIAAA, 1982. DHHS Publication No. (ADM) 82-1190

vate high schools selected to produce national estimates of drug-taking behavior among high school seniors, alcohol consumption among high school seniors has remained relatively stable from 1975 through 1982. The data on specific consumption show that more than 90 percent of surveyed seniors reported having used alcohol at least once during their lifetimes, and almost 90 percent reported using it in the past year. Six percent reported daily or nearly daily use during the month prior to the survey, and a significantly greater percentage reported occasional heavy drinking. Approximately 41 percent of the 1982 sample of high school seniors stated that, on at least one occasion during the 2-week period prior to the survey, they drank five or more drinks.

In general, young males report more drinking episodes and more daily use than females, and consumption of larger quantities of alcohol on a single drinking occasion. High school seniors from the South and the West apparently consume less alcohol than do those from the Northeast and the North Central regions.

These surveys of high school seniors also identified the grade at which an alcoholic beverage is first consumed. Approximately 10 percent of those surveyed had their first drink by the 6th grade. About 30 percent began drinking by the 8th grade, about 56 percent by the 9th grade, and about 75 percent by the 10th grade. By senior year (12th grade), fully 93 percent of those surveyed had had a drink. The surveys show very little change in this

pattern over time.

The surveys collected attitudinal information about alcohol consumption. In the 1981 survey, 22 percent believed consumption of one or two drinks daily to be harmful; 36 percent saw significant risk in having five or more daily drinks once or twice each weekend; and 65 percent believed that consumption of four or five drinks daily would result in significant harm. Thirty-one percent of the 1981 high school seniors did not disapprove of taking one or two drinks nearly every day. Little variation in these attitudes occurs over the 1975-1981 period (NIDA 1983a).

Finally, the NIDA survey data show a steady decline (except for the year 1979) in the 12- to 17-year-olds who reported taking a drink in the 30-day period prior to the survey. The drop from 37 percent in 1979 to 26 percent in 1982 may reflect recent changes in the legal age for purchasing alcohol in the majority of States. Future monitoring of the trends in alcohol consumption among teenagers and young adults will clarify these findings.

More detailed results of the NIDA surveys are summarized in Alcohol and Health Monograph No. 1 (Rachal et al. 1982).

Surveys of Alcohol Consumption During Pregnancy

Recently, national surveys have been conducted of a sample of married women who

gave birth to live infants in 1980, as well as a sample of married women whose pregnancies terminated in a reported fetal death. These two surveys conducted by the National Center for Health Statistics (NCHS) collected information from married mothers, hospitals, physicians, and the birth or death certificates (Prager et al. in press). Data were collected on prenatal care, previous pregnancies, diagnostic and other procedures performed during pregnancy, and drinking and smoking behavior before and during pregnancy. (Additional data and discussion are included in the chapter on alcohol and pregnancy outcome.)

Preliminary analysis of 4,405 mothers giving live births show that women substantially reduce their alcohol consumption after they become pregnant (table 1). While 45 percent of these women abstained from alcohol use before pregnancy, approximately 61 percent abstained during pregnancy. The percentage of all mothers who were moderate/heavy drinkers (Level 2) declined from 12 percent before pregnancy to 2 percent during pregnancy.

Table 1 also shows changes in drinking from before pregnancy to during pregnancy. Of the 516 women who drank 0.25 ounces or more of absolute alcohol per day (Level 2) before pregnancy, only 16 percent continued to consume that amount during pregnancy, while 67 percent consumed less than that amount (Level 1), and 17 percent stopped drinking altogether. Similarly, of the 1,908 light drinkers (Level 1), 66 percent remained light drinkers,

Table 1. Number and percentage distribution of change in drinking behavior from before to during pregnancy, 1980

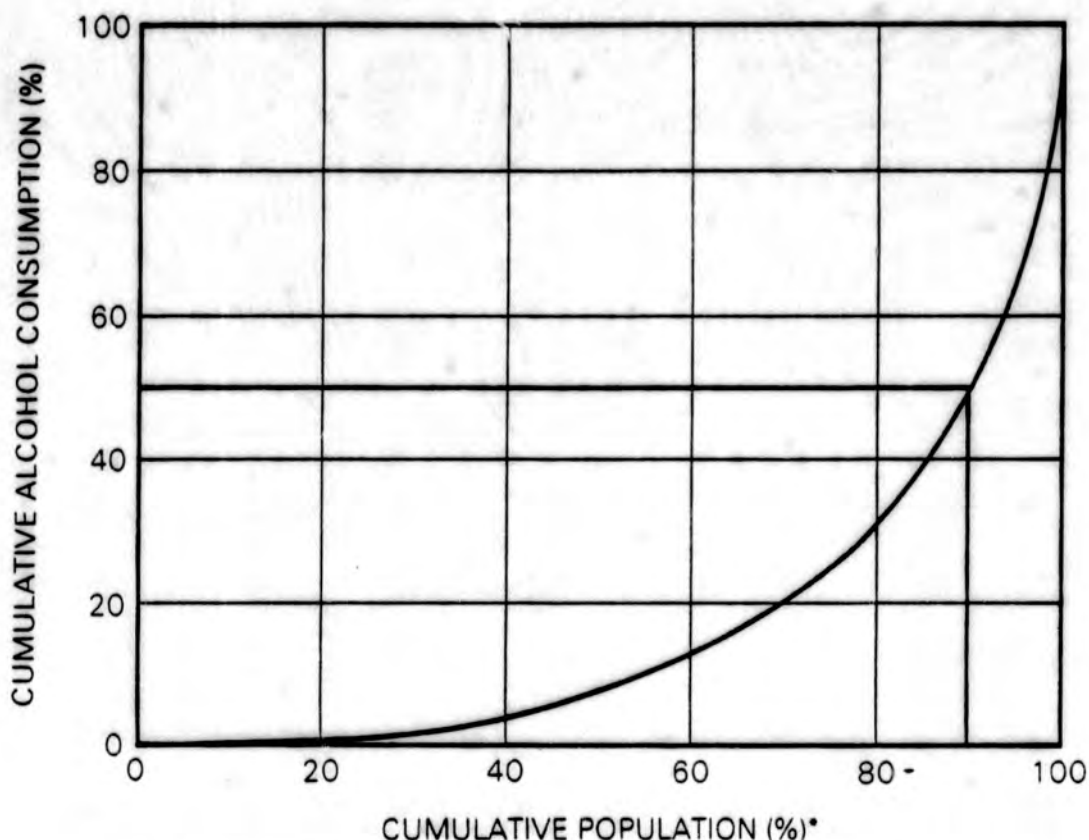
(Married mothers giving live births)

	Before pregnancy		During pregnancy		
	Number	Percent	Nondrinker	Level 1	Level 2
Nondrinker	1,981	45	99	1	1
Level 1	1,908	43	33	66	1
Level 2	516	12	17	67	16
Total	4,405	100	61	37	2

NOTE: Nondrinkers consumed less than one drink less than once a month. Level 1 drinkers consumed less than 0.25 ounces of absolute alcohol per day on the average. Level 2 drinkers consumed 0.25 or more ounces per day.

Source: Prager et al. in press.

Figure 4. Cumulative Distribution of Alcohol Consumption



*Drinkers only, aggregated from several U.S. national surveys, 1971-1975

SOURCE: AEDS data from HANES I Medical History Questionnaire and Louis Harris and Associates Surveys for 1972-1974

while 33 percent stopped drinking. These data confirm other research showing similar decreases (Little et al. 1976; Rosett et al. 1983).

This research also describes drinking behavior before and during pregnancy for several sociodemographic characteristics of married mothers giving live births. Black mothers drink less before and during pregnancy than white mothers. Hispanic mothers drink less than non-Hispanic mothers. Women with less education and from families with lower incomes drink less than women with more education or higher family incomes.

Alcohol-Related Mortality Attributed to Alcoholism, Alcoholic Psychosis, and Liver Cirrhosis

Three important causes of death attributed to excessive alcohol consumption are alcoholism, alcoholic psychosis, and cirrhosis of the liver. By definition, all deaths from alcoholism and alcoholic psychosis are alcohol related; however, alcohol is not the exclusive cause of cirrhosis of the liver. The 1979 U.S. mortality rate for alcoholism was 1.8 per

100,000 population, or approximately 4,000 deaths. The mortality rate in 1979 for alcoholic psychosis was 0.2 per 100,000 population, or about 440 deaths. Cirrhosis of the liver, the eighth leading cause of death in the United States in 1979, accounted for about 29,000 deaths, or 13.2 deaths per 100,000 population.

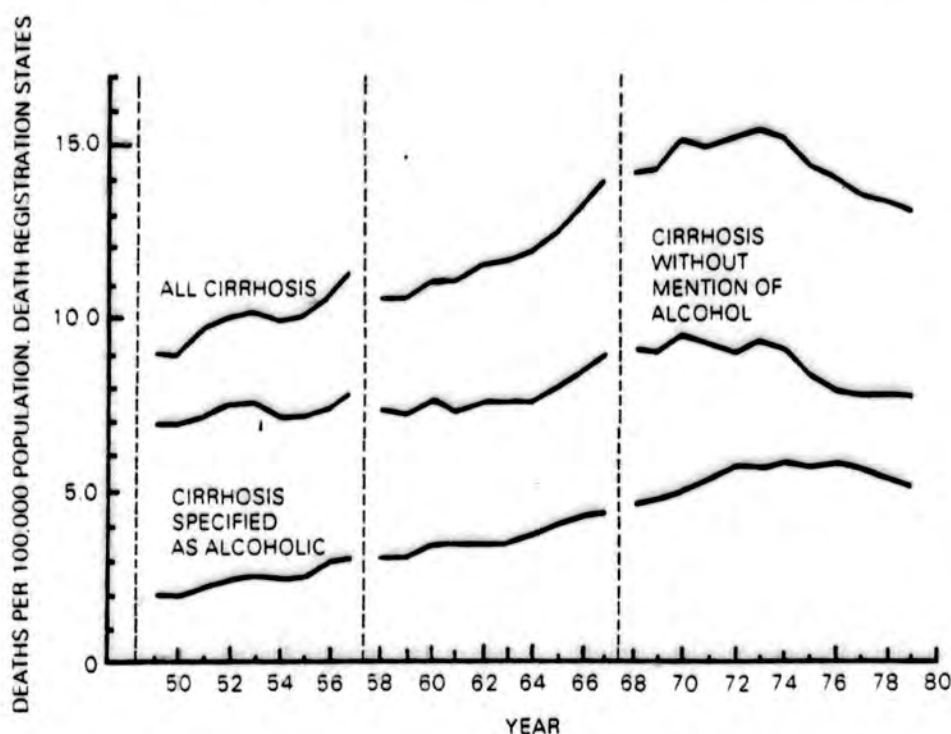
Since 1949, the death rates attributed to alcoholic psychosis and alcoholism have remained relatively stable, while the death rate attributed to cirrhosis of the liver rose until about 1973 and then began to decline (see figure 5). The U.S. mortality rate for cirrhosis decreased 12 percent between 1973 and 1979. The overall decline in the cirrhosis death rate appears to be due to factors other than alcohol; alcohol-related cirrhosis death rates have remained stable at approximately 5 deaths per 100,000 population (Malin et al. 1982a).

Alcohol-Related Causes of Hospitalization¹

The national vital statistics record system of the National Center for Health Statistics (NCHS) collects and tabulates mortality information by cause of death for the entire U.S. population, but no comparable data system exists to collect and tabulate statistics on morbidity (disease) for the entire population. The Hospital Discharge Survey (HDS) operated by NCHS approximates a national morbidity surveillance system by sampling more than 225,000 records from over 400 participating hospitals on an annual basis.

¹ This section summarizes various reports prepared by the National Center for Health Statistics. These reports have been cumulated in the references.

Figure 5. Trends in Cirrhosis Mortality Rates With and Without Mention of Alcohol in the United States, 1949-1979



SOURCE: Malin (1982) and personal communication with Mortality Branch, National Center for Health Statistics

National estimates have been made for the years 1971 through 1980 on the numbers and rates of patients discharged with diagnoses of alcoholism, alcoholic psychosis, and cirrhosis of the liver. Because of sampling variability in the HDS from year to year and the relatively low frequency of some diagnoses, monitoring trends over time must be viewed with caution. Nonetheless, the data suggest a significant increase in the number of persons hospitalized for alcoholism during the 1970s. For example, there were an estimated 200,000 alcoholism discharges in 1971 compared with an estimate of 519,000 alcoholism discharges in 1978, an increase of more than 160 percent. During this same period, the national mortality rates remained relatively stable.

When HDS data are considered by sex, the rates for discharges with alcoholism are 3-1/2 to 4-1/2 times greater for males than for females. While the numbers and rates of discharges with alcoholism have increased for both sexes, this ratio has remained fairly constant. The male rate for hospital discharges for cirrhosis of the liver has remained stable through the 1970s at about 1-1/2 times the female rate.

Alcohol-Related Problems in Veterans

Data from the Veterans Administration (VA 1982) show that a substantial fraction of the nearly 1 million discharges from VA hospitals in 1980 had an alcohol-related disorder, including alcoholic psychosis, alcoholism, organic brain syndrome, and alcoholic cirrhosis of the liver. Such disorders constitute the second largest category of diagnoses (next to heart disease) among patients discharged from VA hospitals. The proportion rose from 15 percent in 1970 to 19 percent in 1974 and 1975 and has dropped slightly to 17 percent in 1977-1980. Nearly two-thirds of these discharges had a principal diagnosis that was alcohol related.

Periodically, the VA conducts a census of hospitalized patients (VA 1982). During the 1970s, the rate of patient hospitalizations with an alcohol-related principal diagnosis was relatively stable at about 8 to 10 percent each year. In some years, the VA has also estimated the number of problem drinkers, who constitute an additional 5 to 6 percent of the patient census. When problem drinkers are added to those patients identified as alcoholic, about 25 percent of the entire VA patient load have alcohol-related problems that contribute to their medical disabilities.

Since 1970, the proportion of VA patients with alcoholism or problem drinking has more than doubled for those under age 35 (15 percent to 35 percent). This marked increase reflects the growth in alcohol-related disabilities from the Vietnam era. The reasons for this substantial increase remain to be identified. It is not clear whether (a) this increase is specific to the VA beneficiary program, (b) the statistics reflect a more general increase among all men of comparable age, (c) younger veterans have a greater proportion of alcohol problems simply because the incidence of other health conditions such as cancer or stroke is less in this population, or (d) the increases are attributable to some combination of these and other reasons. The trend over time does reflect, in part, the aging of this group, increased years of drinking, and a greater probability of being diagnosed as alcoholic. In response to alcohol problems, the VA has promoted specialized alcohol dependence treatment programs, which have multiplied from 8 in 1970 to 100 in 1980.

Alcohol-Related Problems Among Native Americans

Alcohol-related mortality is a major problem among Native Americans, with cirrhosis of the liver the fourth leading cause of death. Accidents are the leading cause, and the contribution of alcohol to fatal mishaps is substantial. Death rates attributable to major alcohol-related causes of death (alcoholism, alcoholic psychosis, and cirrhosis of the liver with mention of alcoholism) are about eight times greater among Native Americans than for the U.S. population as a whole (Indian Health Service 1982a).

Hospital discharge rates from the Indian Health Services (IHS) hospital system for alcohol-related diagnoses are about three times the rates for the entire United States and double the rates for races other than white. This higher IHS alcohol-related discharge rate applies to both males and females. For ages 15 to 44 years, generally years of high productivity, the IHS hospital discharge rate for alcohol-related diagnoses is more than four times that of the rate for the total United States (Indian Health Service 1982b).

Table 2 shows age-specific hospital discharge rates for the IHS and for the United States as a whole in 1981. IHS discharges are from IHS and general hospitals under contract to the IHS, while U.S. discharges are from

non-Federal short-stay hospitals. The alcoholic psychosis rate for all ages for IHS discharges is four times as great (11.8 vs. 2.9 per 100,000) as the discharge rate for the United States as a whole. The discharge rates for nondependent abuse of alcohol and for alcoholic liver disease are 3.4 times as great (5.8 versus 1.7 and 6.4 versus 1.9, respectively), and the alcohol dependence syndrome rate is nearly double that for the total United States (35.8 versus 19.7). The table also shows these comparisons for specific age groups where the differences are even greater.

Alcohol-Related Motor Vehicle Fatalities

Motor vehicle accidents have claimed between 45,000 and 55,000 lives annually over the past decade. While the death rate per 100 million vehicle miles has in general declined by more than 25 percent (perhaps as the result of the post-oil-embargo lower speed limit and more rigorous enforcement), motor vehicle crashes have become one of the Nation's leading causes of death for all age groups and the leading cause of death for persons aged 15 to 24 years. In this age group, motor vehicle fatalities account for about 40 percent of all deaths.

The National Highway Traffic Safety Administration (NHTSA) maintains national sur-

veillance of all traffic crashes involving one or more fatalities, the Fatal Accident Reporting System (FARS). For every fatal accident, FARS provides data on each vehicle, each driver, and each person involved in the accident, including a subjective characterization of alcohol involvement when it is known. In 1981, the blood alcohol concentration (BAC) was known for only 30 percent of the 62,120 drivers involved in fatal traffic crashes (see table 3). Among these 18,540 drivers, 6,349 (34 percent) had no detectable BAC at the time of the testing; 2,450 (13 percent) had BACs between 0.01 and 0.09 percent; while the remaining 9,741 (53 percent) had BACs of 0.10 percent or higher, the level of legal intoxication in all but three States. In addition, of the 48,069 deaths that occurred in 1981, 20,658 (43 percent) were known to involve alcohol.

Alcohol involvement in accidents is especially common in single-vehicle crashes, in which young people are disproportionately involved (see table 4). The 16- to 24-year-old age group accounts for only 16.5 percent of the U.S. population but 41 percent of the single-vehicle accident fatalities and 45 percent of all single-vehicle fatal accidents involving alcohol. In contrast, the 55-and-older age group represents 21 percent of the population but accounts for only 11 percent of all single-vehicle accident fatalities and 5.5 percent of the single-vehicle accidents involving

Table 2. Discharge rates per 10,000 from Indian Health Service and contract general hospitals and from non-Federal short-stay hospitals by first-listed diagnosis and age, 1981

Age at admission	Alcoholic psychosis		Alcoholic dependence syndrome		Alcoholic nondependent abuse of alcohol		Liver disease	
	IHS rate	U.S. rate	IHS rate	U.S. rate	IHS rate	U.S. rate	IHS rate	U.S. rate
All ages	11.8	2.9	35.8	19.7	5.8	1.7	6.4	1.9
Under 15 years	0.0	0.0	0.6	0.6	0.9	0.4	0.0	0.0
15-44 years	19.0	3.1	50.5	22.6	8.6	2.1	8.0	1.1
45-64 years	20.1	6.3	83.2	38.3	8.9	2.3	18.9	5.2
65 years and over	4.9	2.0	21.7	13.6	4.4	1.6	4.2	2.4

Source: Indian Health Service 1982b.

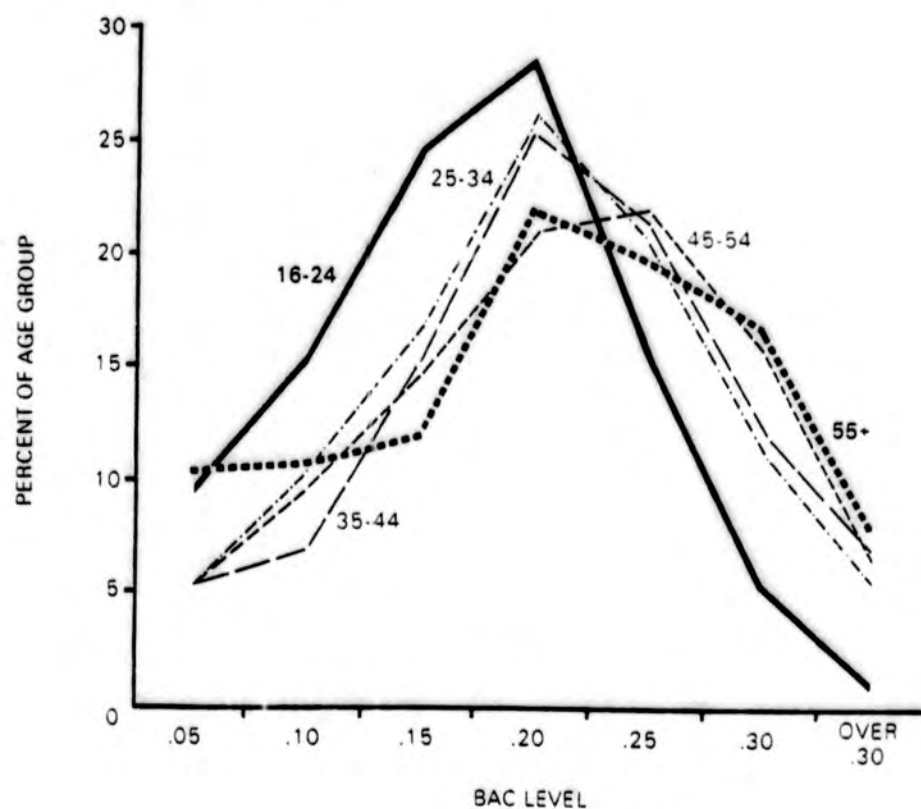
Table 3. Alcohol involvement by performance measure and by alcohol test results (BAC) of drivers in fatal traffic accidents, 1981

Alcohol involvement determined by performance measure ^a	Alcohol test results (BAC)			Persons tested		Drivers in fatal accidents
	0	.01-.09	.10 and +	Number	Percent	
No	6,136	2	2	6,140	14	42,878
Yes	213	2,448	9,739	12,400	64	19,242
Total	6,349	2,450	9,741	18,540	30	62,120

^a When suspected of drinking by an investigating officer, a driver's poor response to performance measures, such as the ability to walk a straight line, generally leads to a label of "alcohol involvement" on the investigative report.

Source: NIAAA 1983.

Figure 6. Percentage of Drivers With Positive Blood Alcohol Concentration (BAC) by Age Group in Single Vehicle Accidents, 1981



SOURCE: Malin et al. 1982b.

alcohol. Young drivers are even more disproportionately involved in single-vehicle accidents when other factors such as average miles driven and proportion licensed to drive are considered. Fewer young drivers are licensed than their older counterparts and they drive less on average.

Figure 6 shows the percentage of drivers in single-vehicle fatal accidents in each age group at various levels of BAC. Greater percentages of young drivers have lower BACs than older drivers. This suggests that younger drivers become involved in fatal accidents at lower BAC levels than older drivers (Malin et al. 1982b).

Drivers involved in alcohol-related fatal crashes have a higher frequency of previously recorded accidents, license suspensions and revocations, and convictions for driving while intoxicated (DWI), as well as a higher proportion of previously recorded speeding convictions and "other harmful moving violations." For the 62,120 drivers involved (but not necessarily killed themselves) in fatal traffic crashes in 1981, data were collected on their previous convictions for DWI. Among the drivers who were tested and found to have no detectable BAC, 2 percent had a previous DWI conviction. Among the drivers whose BAC was in the range from 0.01 to 0.09 percent, 5 percent had a previous DWI conviction. Among the drivers with BACs of 0.10 or greater, 11 percent had previous DWI convictions, a rate 5.5 times greater than the group without detectable BAC (NIAAA 1983).

Alcohol- and non-alcohol-related traffic fatalities appear to follow similar monthly variations. For both groups, fatalities are higher in summer months when more vehicle miles are driven, while in winter they tend to fall below the average monthly rate.

Figure 7 displays the day of week and the time of day for all fatalities in 1981. Fatalities in accidents with no known alcohol involvement peaked between 4 p.m. and 8 p.m. on Sunday through Thursday. In contrast, the alcohol-related fatalities peaked later in the evening (8 p.m. to 4 a.m.) for Monday through Thursday. Similarly, on Friday and Saturday, the greatest number of non-alcohol-related fatalities occurred between 4 p.m. and 4 a.m., while alcohol-related fatalities occurred later (midnight to 4 a.m.) and were much higher than the weekend maximums for fatalities with no known alcohol involvement (NIAAA 1983).

Summary

In summary, it can be said that the acquisition of new knowledge about the epidemiologic factors affecting alcohol abuse and alcoholism is increasing. Advances in knowledge have been hampered by a lack of uniformly applied definitions of terms such as alcoholism and alcohol abuse, but there are encouraging signs of progress.

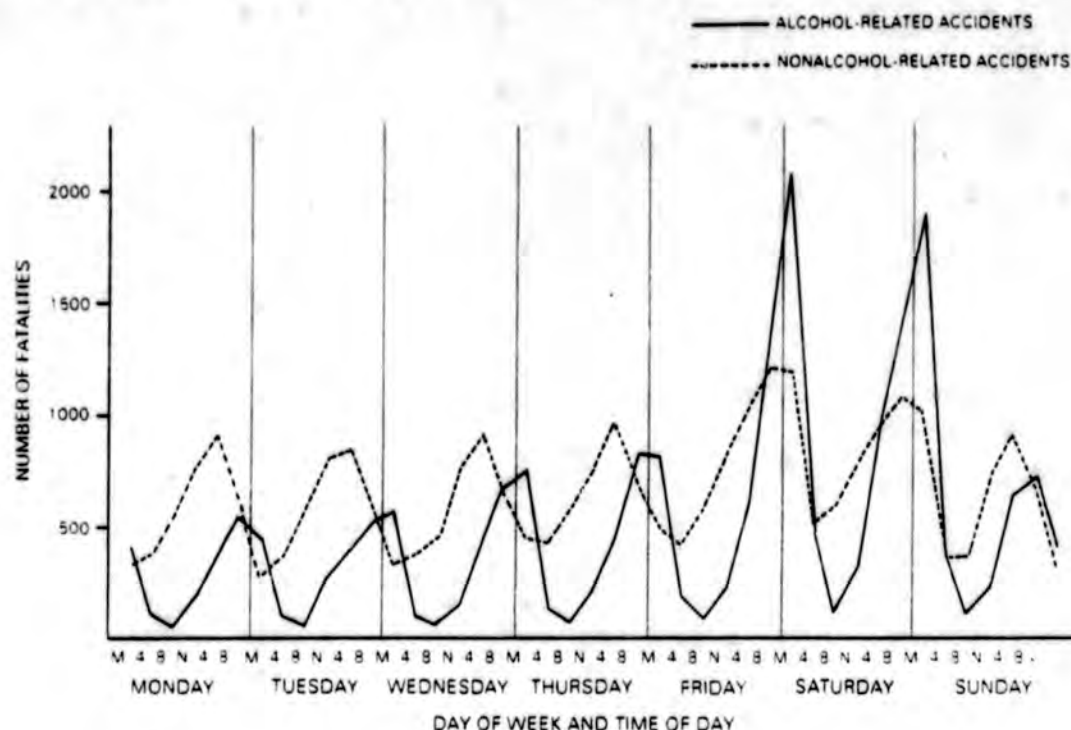
The per capita alcohol consumption continues to rise, although not as steeply as in

Table 4. Fatal single-vehicle accidents by age of driver and alcohol involvement, 1981

Age group	U.S. population (percent)	Number single- vehicle accidents	Total (percent)	Number accidents with alcohol	Alcohol-involved accidents (percent)
16-24	16.5	10,295	41.0	3,190	44.6
25-34	16.8	6,977	27.8	2,193	30.6
35-44	11.5	3,165	12.6	902	12.6
45-54	9.9	2,002	8.0	476	6.6
55+	21.1	2,656	10.6	397	5.5
Totals		25,095	100.0	7,158	100.0

Source: Malin et al. 1982b.

Figure 7. Number of Fatalities by Day of Week, Time of Day, and Alcohol Involvement, 1981



SOURCE: AEDS data from NHTSA Fatal Accident Reporting System (FARS) for 1981

earlier decades. About a third of the total population abstains from alcohol consumption during the typical year, another third consumes an average of less than one drink every other day, while the remaining third drinks more heavily.

Women substantially reduce their alcohol consumption after they become pregnant. While 45 percent abstain before pregnancy, 61 percent are abstainers during their pregnancy period. Similarly, 12 percent are moderate to heavy drinkers before pregnancy, while only 2 percent continue drinking to this extent after they become pregnant.

Overall mortality from major alcohol-related causes of death has remained relatively stable. Alcohol-related disease appears to have fallen from a high point in 1973, but liver cirrhosis has decreased only slightly.

Within the Veterans Administration hospital system, alcohol-related disorders have dropped from previous high levels in the 1970s. However, the proportion of veterans under age 35 with alcohol-related problems has increased

substantially. Alcohol-related problems among Native Americans continue to be a major source of both morbidity (disease) and mortality, with alcohol-related disorders among Native Americans being several times higher than for the U.S. population as a whole.

A substantial fraction of the Nation's traffic fatalities continues to be associated with alcohol consumption. More than half the drivers involved in fatal accidents are intoxicated. Drinking drivers involved in fatal crashes tend to have poorer driving records, including a higher frequency of previously recorded accidents, a higher frequency of previously recorded license suspensions and/or revocations, and a higher proportion of previously recorded speeding convictions than nondrinking drivers in similar accidents.

Epidemiological research continues to clarify the nature and extent of alcohol abuse and alcoholism. Increased knowledge and application of that knowledge to prevention and treatment could help reduce the individual and social costs of alcohol abuse and alcoholism.

References

- Clark, W.B., and Midanik, L. Alcohol use and alcohol problems among U.S. adults. Results of the 1979 national survey. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM) 82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 3-52.
- Indian Health Service. Analysis of Fiscal Year 1981 IHS and U.S. Hospital Discharge Rates by Age and Primary Diagnosis. Rockville, Md.: IHS, August 1982a.
- Indian Health Service. "Chart Series" Tables. FY 1983 ed. Rockville, Md.: IHS, Office of Program Statistics, March 1982b.
- Little, R.E.; Schultz, F.A.; and Mandell, W. Drinking during pregnancy. Journal of Studies on Alcohol 37:375-379, 1976.
- Malin, H.; Coakley, J.; Kaelber, C.; Munch, N.; and Holland, W. An epidemiologic perspective on alcohol use and abuse in the United States. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM) 82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982a. pp. 99-153.
- Malin, H.J.; Trumble, J.; Kaelber, C.T.; and Lubran, B. Alcohol-related highway fatalities among young drivers—United States. Morbidity and Mortality Weekly Report 31 (48):641-644, 1982b.
- National Center for Health Statistics. Detailed diagnoses and surgical procedures for patients discharged from short-stay hospitals: United States, 1977, DHHS Pub. No. (PHS) 79-1274; 1977, DHHS Pub. No. (PHS) 80-1274. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1979, 1980.
- National Center for Health Statistics. Inpatient utilization of short-stay hospitals by diagnosis: United States, 1971. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 16. DHEW Pub. No. (HRA) 75-1767; 1972. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 20 DHEW Pub. No. (HRA) 76-1771; 1973. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 25 DHEW Pub. No. (HRA) 77-1776; 1974. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 30 DHEW Pub. No. (HRA) 77-1785; 1975. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 35 DHHS Pub. No. (PHS) 81-1716. Hyattsville, Md.: NCHS, 1974, 1975, 1976, 1977, 1981.
- National Center for Health Statistics. Standardized microdata tape transcript. 1975 Hospital Discharge Survey. Hyattsville, Md.: NCHS, n.d. NTIS No. PB-270-773; 1976 Hospital Discharge Survey. Hyattsville, Md.: NCHS, n.d. NTIS Order No. PB-82-179-227.
- National Center for Health Statistics. Utilization of short-stay hospitals: Annual summary for the United States, 1979. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 60. DHHS Pub. No. (PHS) 82-1721; 1980. Vital and Health Statistics, Series 13: Data from the National Health Survey, No. 64 DHHS Pub. No. (PHS) 82-1725. Hyattsville, Md.: NCHS, 1981, 1982.
- National Institute on Alcohol Abuse and Alcoholism, Alcohol Epidemiologic Data System. Analysis of Data From FARS for the Year 1981. Rockville, Md.: NIAAA, April 1983.
- National Institute on Alcohol Abuse and Alcoholism, Alcohol Epidemiologic Data System. U.S. Apparent Consumption of Alcoholic Beverages Based on State Sales, Taxation, or Receipt Data. U.S. Alcohol Epidemiologic Data Reference Manual, Section 1. Rockville, Md.: NIAAA, 1982a. NTIS No. PB-82-187-113.
- National Institute on Alcohol Abuse and Alcoholism, Alcohol Epidemiologic Data System. Cirrhosis of Liver Mortality in the United States, Each State and County and Selected SMSAs. U.S. Alcohol Epidemiologic Data Reference Manual, Section 2. Rockville, Md.: NIAAA, 1982b. NTIS No. PB-82-187-097.
- National Institute on Alcohol Abuse and Alcoholism, Alcohol Epidemiologic Data System. Hospital Discharges With Alcohol-Related Conditions. Hospital Discharge Survey, 1975-1977. U.S. Alcohol Epidemiologic Data Reference Manual, Section 4. Rockville, Md.: NIAAA, 1982c. NTIS No. PB-82-187-121.
- National Institute on Alcohol Abuse and Alcoholism, Alcohol Epidemiologic Data System. U.S. Apparent Consumption for Calendar Year 1980. AEDS Working Paper #50. Rockville, Md.: NIAAA, June 1982d.
- National Institute on Alcohol Abuse and Alcoholism, Alcohol Epidemiologic Data System. U.S. Apparent Consumption for Calendar Year 1981. AEDS Working Paper #55. Rockville, Md.: NIAAA, June 1982e.
- National Institute on Drug Abuse. National household survey on drug abuse. NIDA Capsules C83-1:1-3, February 1983a.
- National Institute on Drug Abuse. Student Drug Abuse in America, 1975-1982. DHHS Pub. No. (ADM) 83-1260. Rockville, Md.: NIDA, 1983b.
- Prager, W.; Malin, H.; Graves, C.; Spiegler, D.; Richards, L.; and Placek, P. "Smoking and Drinking Behavior Before and During Pregnancy of Mothers of Live-born Infants." U.S. Department of Health and Human Services. National Center for Health Statistics, Health U.S. 83, in press.
- Rachal, J.V.; Guess, L.L.; Hubbard, R.L.; Maisto, S.A.; Cavanaugh, E.R.; Waddell, R.; and Benrud, C.H. Adolescent Drinking Behavior *M-1978, Vol. 1. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1980.

- Rachal, J.V.; Maisto, S.A.; Guess, L.L.; and Hubbard, R.L. Alcohol use among youth. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM) 82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 55-95.
- Rachal, J.V.; Williams, J.R.; Brehm, M.L.; Cavanaugh, B.; Moore, R.P.; and Eckerman, W.C. A National Study of Adolescent Drinking Behavior, Attitudes and Correlates. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1975. NTIS No. PB-246-002.
- Rosett, H.L.; Weiner, L.; Lee, A.; Zuckerman, B.; Dooling, E.; and Oppenheimer, E. Patterns of alcohol consumption and fetal development. Obstetrics and Gynecology 61 (5):539-546, 1983.
- U.S. Department of Health, Education, and Welfare. Third Special Report to the U.S. Congress on Alcohol and Health. Noble, E.P., ed. DHEW Pub. No. (ADM) 79-832. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1978.
- Veterans Administration. Office of Reports and Statistics. Alcoholism and Problem Drinking, 1970-1975: A Statistical Analysis of VA Hospital Patients. 1980 Supp. Biometrics Monograph No. 14. Washington, D.C.: VA, 1982.
- Zucker, R.A., and Harford, T.O. A national study of adolescent drinking practices in 1980. Journal of Studies on Alcohol, in press.

Chapter II

Genetics and Alcoholism

One of the most keenly debated issues in alcoholism research has been whether genetic factors play an important part in the development of alcoholism. In other words, is susceptibility to alcoholism inherited, at least in some individuals? If so, what are the predisposing factors? To what extent do environmental influences determine the expression and severity of the disorder?

An affirmative answer to the first question began to emerge 10 years ago with the Goodwin et al. (1973) study on adopted-out sons of alcoholics, although the results were initially greeted with skepticism. Impressive evidence now indicates that heritable factors influence susceptibility to alcoholism as well as to a number of physiological and biochemical responses to alcohol consumption. Furthermore, research has documented that environmental and hereditary factors interact to produce the alcoholic state in some individuals.

Heritability of Alcoholism— Early Studies

The notion that alcoholism runs in families dates back centuries. Reports before 1900 on the familial nature of alcoholism were primarily anecdotal. In the past 80 years, however, more than 100 studies have examined the familial incidence of alcoholism (Goodwin 1971). In reviewing 39 studies conducted over the past four decades, Cotton (1979) summarized statistics on the families of 6,251 alcoholics and 4,083 nonalcoholics. The most striking finding was that, regardless of the nature of the nonalcoholic comparison population, alcoholics were more likely than nonalcoholics to have an alcoholic father, mother, sibling, or distant relative. On the average, almost one-third of any sample of alcoholics had at least one parent with an alcoholism problem. Alcoholic patients were six times more likely than nonpsychiatric patients and two times more likely than psychiatric pa-

tients to report parental alcoholism. Thus, the high rate of parental alcoholism appeared to be not a general characteristic of a disturbed population but a specific correlate of alcoholism. Alcoholism was more prevalent in male relatives, in the families of female alcoholics, and in near relatives than in distant relatives.

The results of family studies do not unequivocally support genetic transmission. A family shares a common environment as well as common genes, and familial patterns of alcoholism can be explained just as readily by behavioral and sociocultural theories as by genetic ones. Although evidence in support of environmental theories is tenuous, such factors cannot be ignored. It is notable that, in the family studies summarized by Cotton (1979), 45 to 80 percent of the alcoholic subjects did not have an alcoholic parent or other relative. This suggests that in many individuals environmental factors influence the expression and severity of the disorder.

To separate the influence of heredity from that of environment, studies are needed comparing special populations in which the influence of either heredity or environment differs (e.g., comparing rates of alcoholism among identical versus fraternal twins or between half-siblings, or comparing such rates among adopted children of alcoholics versus those who are raised by their biological parents). All of these special population groups have been used in alcohol-related research. A number of studies have compared identical and fraternal twins with regard to alcohol-drinking practices, alcohol metabolism, and the effect of alcohol on brain waves as measured by electroencephalogram (EEG). None specifically investigated clinically defined alcoholism, however.

In 1972, Schuckit and coworkers reported on a study of alcoholism in half-siblings. The 69 probands (alcoholic subjects who brought their families into the study) were hospitalized patients who had half-siblings and had been diagnosed as having primary alcoholism, i.e.,

additional 42 men and 50 women whose biological mothers had been reported for alcohol abuse were included as well. The subjects were compared with four control groups selected from the same population on the basis of age, sex, age at adoption, occupational category of the adoptive parents, and ages of the biological and adoptive parents when the child was born.

Male adoptees whose fathers were reported as having severe alcoholism showed a 20 percent incidence of alcohol abuse compared with 6 percent in the controls. In the adopted men whose biological mothers had been reported for alcohol abuse, 33 percent were reported for alcohol abuse as compared with 19 percent in the control group—however, the difference is not statistically significant. On the whole, these findings again confirm the data of Goodwin and coworkers (1973) concerning a genetic influence on alcoholism or alcohol abuse in the sons of male alcoholics.

Adopted women whose biological parents were reported for alcohol abuse, as well as adopted women with non-alcohol-abusing biological parents, had low rates of alcohol abuse. Because of this outcome, a conclusion about the presence or lack of genetic influence on alcohol abuse in women could not be made.

Researchers at the University of Iowa (Cadoret and Gath 1978; Cadoret et al. 1980) have contributed two reports on alcoholism in American adoptees. The first study focused on male and female adoptees who had been separated at birth from biological parents who showed evidence of psychiatric or behavior disturbance. Biological parents of the control group adoptees were not mentally disturbed. The experimental and control groups were matched on age, sex, time spent in foster care, and age of biological mother at birth of her child. Data were collected on 84 of 173 adult adoptees by interview, either with the adoptive parents or with both adoptee and adoptive parents. The remainder of the identified adoptees (51 percent) either could not be located or refused to be interviewed.

Two or more social or medical complications of excessive alcohol use or hospitalization for detoxification constituted a diagnosis of alcoholism (Feighner et al. 1972). The alcoholism rate was low in both the experimental and control groups. Three adoptees with primary alcoholism were associated with 6 alcoholic biological parents versus 1 alcoholic adoptee from 78 biological parents without alcoholism. Seven adoptees had sec-

ondary alcoholism (alcoholism occurring with and preceded by adult psychiatric disorders), but none of them had alcoholic biological parents. Adoptee alcoholism was found to be unrelated to psychiatric diagnoses in biological parents or to a variety of psychosocial and environmental variables in postnatal life. It was, however, significantly correlated with childhood conduct disorders in the adoptees and with alcoholism and alcohol abuse in the biological parents.

In the second Iowa study, only male adoptees were studied, but the sample was enlarged to include adoptees whose second-degree relatives (e.g., grandparents, aunts, uncles, cousins) had psychiatric or behavior disturbances. A matched control group was also recruited. Out of the 167 male adoptees 18 years and older thus identified, data were collected on 92 men (55 percent) by interview with the adoptee, the adoptive parent, or both. The studies were marred by high dropout rates; as measured by available information on participants and nonparticipants, however, sample bias in the two studies was estimated to be minimal. Of the 92 male adoptees, 23 were alcoholic and 22 had experienced childhood conduct disorders. Evidence for alcoholism was present in 15 first and second-degree relatives. Statistically significant associations existed between adoptee alcoholism and an alcoholic family (biological) background and between childhood disorder and the development of alcoholism in adulthood. None of the environmental factors, including psychiatric or alcohol problems in the adoptive family as well as its socioeconomic status, predicted alcoholism.

Both studies confirm the conclusions of Goodwin and coworkers "that genetic factors importantly affect alcoholism development." But both failed to demonstrate an independent effect of environmental factors in the development of alcoholism. Also, the association of childhood conduct disorders with alcoholism in adult life is in accord with previous reports suggesting that childhood conduct disorder and hyperactivity may predict subsequent alcoholism (Schuckit et al. 1972; Goodwin et al. 1975).

Environmental-Genetic Influence on Alcoholism

The adoption studies described here clearly demonstrate that genetic predisposition is an

important determinant of alcoholism in some individuals. What then is the contribution of environmental influences to the expression of alcoholism in other individuals? Do these influences affect severity of the disorder in the genetically susceptible individual?

In other words, are subtypes of alcoholism and alcohol abuse each affected to different extents by genetic and environmental variables? To study these questions, researchers from Washington University in St. Louis and from Sweden collaborated to analyze further the population of adoptees studied by Bohman (1978). This research team first examined adopted men with alcohol abuse problems (Cloninger et al. 1981). Out of 1,125 adopted men in the Stockholm adoption study reported by Bohman, 862 subjects were selected whose paternity was known and who were adopted before the age of 3 by nonrelatives.

These adoptees were first divided into groups according to severity of alcohol abuse. Mild abuse was defined as one registration for abuse (by the excise board) without treatment. Moderate abusers had two or three registrations without treatment, whereas severe abusers had at least four registrations and compulsory treatment or psychiatric hospitalization for alcoholism. The characteristics of the biological parents (e.g., severity of alcohol abuse, occupational status) and of postnatal environment (e.g., occupational status of adoptive father, age at adoptive placement) were analyzed to find which factors would best distinguish one group from another. The discriminant characteristics of each group were then applied to the total population to measure the extent to which these characteristics contributed to the number of individuals with alcohol problems.

As a result of these statistical analyses, two heritable subtypes of alcoholism were identified, which the authors named Type I (or milieu-limited) and Type II (or male-limited). Type I alcohol abuse occurred in 13 percent of the adopted men. Both their biological fathers and mothers typically exhibited mild alcohol abuse requiring no treatment, and postnatal environment determined both the frequency and the severity of alcohol abuse in the susceptible sons. The sons' alcohol abuse was usually mild or isolated, but could be severe, depending upon the nature of the environmental provocation. With such provocation, the relative risk of developing alcohol abuse in congenitally predisposed individuals was increased twofold; without it, the risk was the same as that in the general population.

Type II alcohol abuse occurred in 4 percent of the adopted men. It represented, therefore, about 25 percent of the alcohol abusers in the study. Their biological fathers, but not their mothers, had severe alcoholism requiring extensive treatment. Postnatal environment had no effect on the expected numbers of abusers but may have influenced severity. Alcohol abuse in the susceptible sons was usually recurrent and moderate, although sometimes it was severe. An estimated ninefold increase in risk of developing alcohol abuse or alcoholism was evident in the affected individuals, regardless of postnatal environment. The characteristics of this type of alcohol abuse/alcoholism are similar to those described in the Danish adoption studies (Goodwin et al. 1973).

It is notable that an average of 60 percent of the men in all three categories of alcohol abuse had no family history of abuse. Their alcohol abuse may have been caused by other disorders, such as psychiatric or behavioral. Alternatively, they could represent, in part, a group whose parents were genetically susceptible to alcohol abuse but lacked the necessary environmental provocation to become abusers. This issue remains unresolved.

Bohman et al. (1981) studied adopted women, using a similar statistical analysis. From the 1,199 adoptees originally reported by Bohman (1978), 913 satisfied the entry criteria. Because only 31 women (3.3 percent) out of this sample were alcohol abusers, they were not grouped by severity of abuse. A fourfold increase in alcohol abuse occurred in the adopted women if their biological mothers were alcohol abusers. A relationship between alcohol abuse in the biological father and that in the daughter was less clear-cut. It appeared, therefore, that in daughters maternal alcohol abuse was a more significant predictor of abuse than was paternal alcohol abuse or alcoholism. Both biological parents of alcohol-abusing female adoptees were mild alcohol abusers who usually did not require extensive treatment. When young, the alcohol-abusing female adoptees were frequently placed in rural homes in which adoptive fathers had low occupational status; the women generally had had contact with their biological mothers before final placement. Alcohol abuse in the adoptive home did not modify the risk of alcohol abuse in the daughters. As with the study on male adoptees, many women came from families with no history of alcohol abuse.

The most remarkable finding of this study was the strong component of mother-and-

daughter transmission in the adoptee daughters. But the influence may not be purely genetic, since alcohol abuse by the pregnant mother may have affected the growing fetus (the latter would, of course, be true for mother-daughter or mother-son transmission). In the adopted women, both the severity of alcohol abuse and the characteristics of their biological parents resembled those of the Type I male alcohol abusers. The influence of postnatal environment on frequency and severity of abuse could not be conclusively established, however, because of the limitations of the sample population or the failure to identify the relevant variables. Both of these unresolved issues will require further investigation. Nevertheless, the study demonstrated for the first time the existence of a genetic or congenital determinant for alcohol abuse in women.

The demonstration that sociocultural influences are critically important in the majority of genetically predisposed individuals suggests that changes in behavior and social attitudes by and toward individuals at high risk can alter both the course and the prevalence of alcohol abuse and alcoholism. Potential alcohol abusers could be identified through sensitive and accurate physiological, biochemical, and behavioral predictors of alcoholic vulnerability. These recent discoveries are significant for future treatment and prevention strategies as well as for future research on this important health problem. To this end, prospective and longitudinal studies with children of alcoholics, several of which are in progress, offer a promising approach (Schuckit 1980; Gabrielli et al. 1982; Elmasian et al. 1982).

Genetic Factors in Physiological Responses to Alcohol

A number of biological responses to alcohol potentially have pathophysiological significance for alcohol abuse, including

- alcohol craving or alcohol-seeking behavior
- sensitivity of the central nervous system (CNS) to alcohol
- sensitivity of other body systems (systemic sensitivity) to alcohol
- rate of alcohol metabolism or elimination

- rate of acquisition of tolerance to alcohol
- rate of development and severity of physical dependence on alcohol
- susceptibility to the medical complications of chronic alcohol consumption.

Studies in humans and in experimental animals have shown a tremendous degree of individual variation in their biological responses to alcohol. A considerable body of evidence now shows that much of this variation is genetic in origin (McClearn and Erwin 1982). The findings in humans and in experimental animals will be described separately as they use different experimental approaches and design.

Human Studies

Because genes cannot be manipulated in humans, studies have been limited to the comparison of fraternal and identical twins and of different ethnic groups. Identical twins have identical sets of genes, whereas fraternal twins do not. By comparing variability or, conversely, similarity in the expression of a trait within sets of identical and fraternal twins, researchers can determine the heritability or the importance of genes in the expression of that trait. Twin studies are not perfect, however, since the environment of identical twins tends to be more similar than that of fraternal twins. Hence, the extent to which genetic factors affect that trait cannot be established with absolute certainty. A similar limitation pertains to the comparison of different ethnic populations: They differ socioculturally. Accordingly, all candidate genetic traits predisposing to alcohol abuse should be studied in experimental animals so that environmental influences can be experimentally controlled.

Several twin studies have been performed on drinking behavior (Kaij 1960; Partanen et al. 1966; Jonsson and Nilsson 1968; Loehlin 1972; Kaprio et al. 1978). General agreement prevails that genetic factors influence the quantity and frequency of drinking as well as such symptoms of heavy drinking as hangovers and loss of control. Identical twins are more alike than fraternal twins in their drinking behavior, but the environment may play a considerable role.

Twin studies have also been used to demonstrate that neurophysiological sensitivity to alcohol and alcohol elimination rate are genetically controlled. Propping (1977) showed

that alcohol's effects on brain-wave function are nearly identical in identical twins, whereas they are disparate in fraternal twins. Alcohol elimination rate varies by a factor of 2 to 3 among individuals, even after adjustment for body weight (Bennion and Li 1976). Several studies have compared identical and fraternal twins to determine how much genetic factors contribute to this variation (Vesell et al. 1971; Kopun and Propping 1977). Genetic factors account for about 40-50 percent of the variation in alcohol elimination rate. Daily alcohol intake, smoking, diet, and drug use are some of the environmental factors that account for the remainder.

A potential biochemical explanation for genetic variation in human alcohol elimination rate has recently been discovered. Alcohol is eliminated from the body primarily through metabolism. The enzymes or protein catalysts responsible for this process are alcohol dehydrogenase and aldehyde dehydrogenase. The first enzyme oxidizes alcohol to acetaldehyde and the second enzyme oxidizes acetaldehyde to acetate. Acetate then enters the general metabolic pathway for sugars and fats and is oxidized further to carbon dioxide and water (Li 1977). The amount and properties of alcohol dehydrogenase in the liver determine the rate of alcohol metabolism--and there are many genetically disparate forms of this enzyme in human livers, varying among individuals. The individual molecular forms of human liver alcohol dehydrogenase have now been isolated, and many of them differ markedly in their catalytic properties (Bosron and Li 1981). Since the combination of forms is determined genetically, the variation in elimination rate among individuals can be readily accounted for by the large differences in the genetically determined properties of the different enzyme forms. No experiments have yet tried to correlate the specific enzyme makeup of individuals with their alcohol metabolism rates. However, it has been reported that Chinese and Japanese individuals, on the average, metabolize alcohol 30 percent faster than Caucasians do (Hanna 1978). More than 90 percent of Orientals possess the so-called atypical alcohol dehydrogenase genetic trait (Bosron and Li 1981). The atypical alcohol dehydrogenase enzyme forms are much more active than the forms present in Caucasians.

Some individuals, particularly Orientals, respond to alcohol with an "alcohol flush," a systemic reaction consisting of facial flushing and rapid heart rate and, in severe cases, nausea, vomiting, and low blood pressure. For

example, approximately half of the Japanese population exhibit the reaction, which has been linked to elevated blood acetaldehyde levels after alcohol intake (Mizoi et al. 1979). Alcohol flush was once thought to be the consequence of an overproduction of acetaldehyde (Stamatoyannopoulos et al. 1975). More recent research, however, indicates that the reaction may result from the absence of a particular form of aldehyde dehydrogenase, thereby affecting the oxidation of acetaldehyde (Inoue et al. 1980; Agarwal et al. 1981; Teng 1981). Indirect evidence suggests that acetaldehyde can cause the release of histamine and perhaps other substances affecting blood vessels; hence, the flush reaction.

Finally, studies of twins have shown that some individuals may be genetically susceptible to certain medical complications of prolonged alcohol abuse, namely liver cirrhosis and psychosis (Hrubec and Omenn 1981). Also, some people may be susceptible to developing the Wernicke-Korsakoff syndrome due to an inherited metabolic defect involving one of the thiamine (vitamin B₁)-requiring enzymes (Blass and Gibson 1977). Studies to confirm these important observations are clearly needed.

Animal Studies

Animals, like humans, vary in their responses to alcohol. Genetic factors are responsible for some of the differences, including such responses to alcohol as the acquisition of tolerance and physical dependence. An important feature of animal experimentation, impossible with humans, is the ability to manipulate the genetic background of the animals through inbreeding, selective breeding, and other measures. It has been possible to develop lines or strains of animals with high or low responsiveness to the various actions of alcohol. Such animals can then be used to study the biochemical and physiological mechanisms underlying genetic variability and to discern how these developed traits and mechanisms affect alcohol-drinking behavior. Conversely, breeders can develop animals that prefer or disdain alcohol, and researchers can test their responsiveness to the various actions of alcohol. Such animal studies can suggest hypotheses and experimental approaches in human research to learn what predisposes individuals genetically to aberrant drinking behavior. Research with animal models thus provides a cornerstone of future biological knowledge

about the nature of genetic susceptibility to alcoholism.

Inbred strains of mice differ in their preference for alcohol consumption when food, water, and alcohol are made freely available to them (McClearn and Rodgers 1959). Inbred mouse strains also differ in "sleep time," that is, the time required to recover from the hypnotic effects of alcohol (Kakihana et al. 1966), and in alcohol metabolic rate (Sheppard et al. 1970). Inbred strains are developed by mating successive generations of close relatives. After about 20 generations of sibling matings, the animals within each inbred strain are almost genetically uniform and show the trait readily. The strains constitute *prima facie* evidence of genetic influence on that trait.

A genetic influence on alcohol-drinking behavior, CNS sensitivity to alcohol, and alcohol metabolic rate has also been demonstrated in selectively bred lines of rats and mice. Selective breeding is the process by which animals showing a chosen characteristic are mated in successive generations in order to segregate the genes responsible for that trait into one line of animals. Thus, if a quantitative trait is under genetic control, separate strains can be bred, one in the high and the other in the low direction. Rats that differ in alcohol metabolic rate have also been raised through selective breeding (Thurman 1980). Lines of rats have now been bred selectively to differ in voluntary alcohol consumption: the alcohol-preferring AA and P lines and the alcohol-nonpreferring ANA and NP lines (Eriksson 1968; Li et al. 1979). Lines of mice and rats that differ in CNS sensitivity to the sedative-hypnotic effects of alcohol have also been developed: the long-sleep (LS) and short-sleep (SS) mouse lines (McClearn and Kakihana 1973) and the most-affected (MA) and least-affected (LA) lines of rats (Riley et al. 1977). Such selectively bred animals are particularly useful for studying the underlying mechanisms that contribute to these genetic differences. For example, the difference in sleep time between the LS and SS mice is related to a genetically determined difference in the brain that is both region-specific and drug-specific (Sorensen et al. 1981). New and exciting findings are likely to emerge from further studies with these animals that will improve researchers' understanding of the differential CNS sensitivity to alcohol in humans.

Various inbred strains of mice as well as the selectively bred LS and SS lines of mice differ in how rapidly they develop tolerance after

the chronic administration of alcohol (Grieve et al. 1979; Tabakoff et al. 1980). Inbred mouse strains also differ in the severity of withdrawal symptoms after physical dependence has been established. Goldstein (1973) and Goldstein and Kakihana (1974) bred mouse lines with those reactions. Thus, both the rate of tolerance development and the severity of physical dependence caused by chronic alcohol ingestion are genetically determined in mice and possibly in humans.

Potential Mechanisms of Genetic Influence

Alcoholism and alcohol abuse lie at one extreme of a continuum of drinking behavior whose opposite end represents abstinence. Drinking behavior as a trait, therefore, is a continuous variable, as are height and weight. Such traits are usually determined by many sets of genes that sometimes tend to appear together or to be correlated. When different traits can be influenced by common sets of genes, a phenomenon called pleiotropy, true genetic correlation has occurred. The association may come about because one trait influences the expression of the other. For example, preliminary reports from Japan indicate that, compared with the normal population, alcoholic populations contain fewer individuals with deficiencies in the mitochondrial enzyme aldehyde dehydrogenase that oxidizes acetaldehyde (Harada et al. 1982; Yoshihara et al. 1982). When this enzyme is absent, alcohol consumption leads to high blood acetaldehyde levels and the unpleasant alcohol flush reaction. Thus, an individual with the enzyme deficiency is likely to avoid alcohol and the unpleasant reaction it brings.

Animal studies have revealed other potential mechanisms of genetic influence. Mice that consume large amounts of alcohol voluntarily are more likely than others to acquire tolerance rapidly (Erwin et al. 1980). Alcohol-preferring lines of rats recover more rapidly from the sedative-hypnotic effects of alcohol than do alcohol-nonpreferring ones (Lumeng et al. 1982). These studies suggest an experimentally verifiable hypothesis: An ability to adapt quickly to alcohol (tolerance) may predispose individuals to patterns of heavy drinking.

Other mechanisms of alcohol preference are likely to be discovered, each of which may contribute in some way to the trait of high voluntary alcohol consumption. Animal exper-

iments can reveal such mechanisms of alcohol consumption (NIAAA 1981). This research approach may ultimately lead to an understanding of the nature of genetic heterogeneity of alcohol abuse and alcoholism in humans, the physiological and biochemical mechanisms underlying this problem behavior, and how environmental factors interact with these inherited pathways.

Summary

Great strides have been made in recent years in researchers' knowledge and understanding of the genetic factors contributing to alcohol abuse and alcoholism. It has been firmly established that heredity plays a role in determining individual differences in susceptibility to the disorder. Genetic influence is identifiable in at least 35 to 40 percent of alcoholics and alcohol abusers, and it affects both men and women. People with family histories involving parental alcohol abuse face increased risk. Furthermore, many types of alcohol abuse may exist, each with its own genetic predisposition interacting with a particular environment.

Genetic factors also affect individual differences in response to alcohol, such as drinking behavior, sensitivity of the brain and other body systems to alcohol, alcohol elimination rate, the acquisition of tolerance, and the development of physical dependencies. Much of this knowledge has been obtained from experiments in laboratory animals specifically developed for the purpose of alcoholism-related research.

Collectively, the gains in research in the past decade predict an exciting era of new discoveries in the next. Future research should focus on improving methodology for detecting subtypes of alcohol abuse and on developing behavioral, physiological, and biochemical predictors of genetic vulnerability. In this regard, ongoing and future work in experimental animals can provide invaluable clues for human investigation. Existing animal models and those yet to be developed can be used not only to identify genetic correlates of alcohol-seeking behavior, but also to elucidate the biological nature of the process. When this stage of knowledge is reached, the genetic subtypes of alcohol abuse and alcoholism will be precisely defined as pharmacogenetic disorders of behavior by the nature of their underlying pathology.

References

- Agarwal, D.P.; Harada, S.; and Goedde, H.W. Racial differences in biological sensitivity to ethanol: The role of alcohol dehydrogenase and aldehyde dehydrogenase isoenzymes. Alcoholism: Clinical and Experimental Research 5:12-16, 1981.
- Bennion, L.J., and Li, T.-K. Alcohol metabolism in American Indians and whites. New England Journal of Medicine 294:9-13, 1976.
- Blass, J.P., and Gibson, G.E. Abnormality of a thiamine-requiring enzyme in patients with Wernicke-Korsakoff syndrome. New England Journal of Medicine 297:1367-1370, 1977.
- Bohman, M. Some genetic aspects of alcoholism and criminality: A population of adoptees. Archives of General Psychiatry 35:269-276, 1978.
- Bohman, M.; Sigvardsson, S.; and Cloninger, C.R. Maternal inheritance of alcohol abuse: Cross-fostering analysis of adopted women. Archives of General Psychiatry 38:965-969, 1981.
- Bosron, W.F., and Li, T.-K. Genetic determinants of alcohol and aldehyde dehydrogenases and alcohol metabolism. Seminars in Liver Disease 1:179-188, 1981.
- Cadore, R.J., and Gath, A. Inheritance of alcoholism in adoptees. British Journal of Psychiatry 132:252-258, 1978.
- Cadore, R.J.; Cain, C.A.; and Grove, W.M. Development of alcoholism in adoptees raised apart from alcoholic biologic relatives. Archives of General Psychiatry 37: 561-563, 1980.
- Cloninger, C.R.; Bohman, M.; and Sigvardsson, S. Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. Archives of General Psychiatry 38:861-868, 1981.
- Cotton, N.S. The familial incidence of alcoholism. Journal of Studies on Alcohol 40: 89-116, 1979.
- Elmasian, R.; Neville, H.; Woods, D.; Schuckit, M.; and Bloom, F. Event-related brain potentials are different in individuals at high risk and low risk for developing alcoholism. Proceedings of the National Academy of Sciences 79:7900-7903, 1982.
- Eriksson, K. Genetic selection for voluntary alcohol consumption in the albino rat. Science 159: 739-741, 1968.
- Erwin, V.G.; McClearn, G.E.; and Kuse, A.R. Interrelationships of alcohol consumption, actions of alcohol and biochemical traits. Pharmacology, Biochemistry and Behavior 13(Supplement 1): 297-302, 1980.
- Feighner, J.; Robins, E.; Guze, S.; Woodruff, R.; Winokur, G.; and Munoz, R. Diagnostic criteria for use in psychiatric research. Archives of Gen-

- eral Psychiatry 26:57-63, 1972.
- Gabrielli, W.F.; Mednick, S.A.; Volavka, J.; Pollock, V.E.; Schulsinger, F.; and Itil, T.M. Electroencephalograms in children of alcoholic fathers. Psychophysiology 19:404-407, 1982.
- Goldstein, D.B. Inherited differences in intensity of alcohol withdrawal reactions in mice. Nature 245:154-156, 1973.
- Goldstein, D.B., and Kakihana, R. Alcohol withdrawal reactions and reserpine effects in inbred strains of mice. Life Science 15: 415-425, 1974.
- Goodwin, D.W. Is alcoholism hereditary? A review and critique. Archives of General Psychiatry 25:545-549, 1971.
- Goodwin, D.W.; Schulsinger, F.; Hermansen, L.; Guze, S.B.; and Winokur, G. Alcohol problems in adoptees raised apart from alcoholic biological parents. Archives of General Psychiatry 28:238-243, 1973.
- Goodwin, D.W.; Schulsinger, F.; Hermansen, L.; Guze, S.B.; and Winokur, G. Alcoholism and the hyperactive child syndrome. Journal of Nervous and Mental Disease 160:349-353, 1975.
- Goodwin, D.W.; Schulsinger, F.; Knop, J.; Mednick, S.; and Guze, S.B. Alcoholism and depression in adopted-out daughters of alcoholics. Archives of General Psychiatry 34:751-755, 1977.
- Goodwin, D.W.; Schulsinger, F.; Moller, N.; Hermansen, L.; Winokur, G.; and Guze, S.B. Drinking problems in adopted and nonadopted sons of alcoholics. Archives of General Psychiatry 31:164-169, 1974.
- Grieve, S.J.; Griffiths, P.J.; and Littleton, J.M. Genetic influences on the rate of development of ethanol tolerance and the ethanol physical withdrawal syndrome in mice. Drug and Alcohol Dependence 4:77-86, 1979.
- Hanna, J.M. Metabolic response of Chinese, Japanese and Europeans to alcohol. Alcoholism: Clinical and Experimental Research 2:89-92, 1978.
- Harada, S.; Takagi, S.; Agarwal, D.P.; and Goedde, H.W. Ethanol and aldehyde metabolism in alcoholics from Japan (abstract). Alcoholism: Clinical and Experimental Research 6:298, 1982.
- Hrubec, Z., and Omenn, G.S. Evidence of genetic predisposition to alcoholic cirrhosis and psychosis: Twin concordances for alcoholism and its biological end points by zygosity among male veterans. Alcoholism: Clinical and Experimental Research 5:207-215, 1981.
- Inoue, K.; Fukunaga, M.; and Yamasawa, K. Correlation between human erythrocyte aldehyde dehydrogenase activity and sensitivity to alcohol. Pharmacology, Biochemistry and Behavior 13:295-297, 1980.
- Jonsson, E., and Nilsson, T. Alcohol konsumption hos monozygota och dizygota tvillingpar. Nordisk Hygienisk Tidskrift 49:21-25, 1968.
- Kaij, L. Alcoholism in Twins. Studies on the Etiology and Sequels of Abuse of Alcohol. Stockholm: Almqvist and Wiksell, 1960.
- Kakihana, R.; Brown, D.R.; McClearn, G.E.; and Tabershaw, I.R. Brain sensitivity to alcohol in inbred mouse strains. Science 154:1574-1575, 1966.
- Kaprio, J.; Sarna, S.; Koskenvuo, M.; and Rantasalo, I. The Finnish Twin Registry: Baseline Characteristics. Section II. History of symptoms and illnesses, use of drugs, physical characteristics, smoking, alcohol and physical activity. Kansanterveystieteen Julkaisu M 37, Helsinki, 1978.
- Kopun, M., and Propping, P. The kinetics of ethanol absorption and elimination in twins and supplementary repetitive experiments in singleton subjects. European Journal of Clinical Pharmacology 11:337-344, 1977.
- Li, T.-K. Enzymology of human alcohol metabolism. Advances in Enzymology 46:427-483, 1977.
- Li, T.-K.; Lumeng, L.; McBride, W.J.; and Waller, M.B. Progress toward a voluntary oral consumption model of alcoholism. Drug and Alcohol Dependence 4:45-60, 1979.
- Loehlin, J.C. An analysis of alcohol-related questionnaire items from the National Merit Twin Study. In: Seixas, F.A.; Omenn, G.S.; Burk, E.D.; and Eggleston, S., eds. Vol. 197, Nature and nurture in alcoholism. Annals of the New York Academy of Science 197:117-120, 1972.
- Lumeng, L.; Waller, M.B.; McBride, W.J.; and Li, T.-K. Different sensitivities to ethanol in alcohol-preferring and nonpreferring rats. Pharmacology, Biochemistry and Behavior 16:125-130, 1982.
- McClearn, G.E., and Erwin, V. Mechanisms of genetic influence on alcohol-related behaviors. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM)82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 263-289.
- McClearn, G.E., and Kakihana, R. Selective breeding for ethanol sensitivity in mice. Behavioral Genetics (abstract) 3:409-410, 1973.
- McClearn, G.E., and Rodgers, D.A. Differences in alcohol preference among inbred strains of mice. Quarterly Journal of Studies on Alcohol 20:691-695, 1959.
- Mizoi, Y.; Ijiri, J.; Tatsuno, Y.; Kijima, T.; Fujiwara, S.; and Adachi, J. Relationship between facial flushing and blood acetaldehyde levels after ethanol intake. Pharmacology, Biochemistry and Behavior 10:303-311, 1979.
- National Institute on Alcohol Abuse and Alcoholism. Development of Animal Models as Pharmacogenetic Tools. NIAAA Research Monograph No. 6. McClearn, G.E.; Deitrich, R.A.; and Erwin, V.G., eds. DHHS Pub. No. (ADM)81-1133. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- Partanen, J.K.; Bruun, K.; and Markkanen, T. Inheritance of Drinking Behavior. A Study on Intelligence, Personality and Use of Alcohol in Adult Twins. Finnish Foundation for Alcohol Studies Pub. No. 14. Helsinki: Finnish Foundation for Alcohol Studies, 1966.
- Propping, P. Genetic control of ethanol action on the central nervous system. Human Genetics 35:309-334, 1977.

- Riley, E.P.; Worsham, E.D.; Lester, D.; and Freed, E.X. Selective breeding of rats for differences in reactivity to alcohol: An approach to an animal model of alcoholism. II. Behavioral measures. Journal of Studies on Alcohol 38: 1705-1717, 1977.
- Roe, A. The adult adjustment of children of alcoholic parents raised in foster homes. Quarterly Journal of Studies on Alcohol 5:378-393, 1944.
- Schuckit, M.A. Biological markers: Metabolism and acute reactions to alcohol in sons of alcoholics. Pharmacology, Biochemistry and Behavior 13(Supp. 1): 9-16, 1980.
- Schuckit, M.A.; Goodwin, D.A.; and Winokur, G. A study of alcoholism in half siblings. American Journal of Psychiatry 128:1132-1136, 1972.
- Sheppard, J.R.; Albersheim, P.; and McClearn, G.E. Aldehyde dehydrogenase and ethanol preference in mice. Journal of Biological Chemistry 245:2876-2882, 1970.
- Sorensen, S.; Dunwiddie, T.; McClearn, G.; Freedman, R.; and Hoffer, B. Ethanol-induced depressions in cerebellar and hippocampal neurons of mice selectively bred for differences in ethanol sensitivity: An electrophysiological study. Pharmacology, Biochemistry and Behavior 14: 227-234, 1981.
- Stamatoyannopoulos, G.; Chen, S.-H.; and Fukui, M. Liver alcohol dehydrogenase in Japanese: High population frequency of atypical form and its possible role in alcohol sensitivity. American Journal of Human Genetics 27:789-796, 1975.
- Tabakoff, B.; Ritzmann, R.; Raju, T.S.; and Deitrich, R.A. Characterization of acute and chronic tolerance in mice selected for inherent differences in sensitivity to ethanol. Alcoholism: Clinical and Experimental Research 4:70-73, 1980.
- Teng, Y.-S. Human liver aldehyde dehydrogenase in Chinese and Asiatic Indians: Gene deletion and its possible implications in alcohol metabolism. Biochemical Genetics 19:107-114, 1981.
- Thurman, R.G. Ethanol elimination rate is inherited in the rat. In: Thurman, R.G., ed. Alcohol and Aldehyde Metabolizing Systems - IV. Advances in Experimental Medicine and Biology Vol. 132. New York: Plenum Press, 1980. pp. 655-661.
- Vesell, E.S.; Page, J.G.; and Passananti, G.T. Genetic and environmental factors affecting ethanol metabolism in man. Clinical Pharmacology and Therapeutics 12:192-201, 1971.
- Yoshihara, H.; Sato, N.; Kamada, T.; and Abe, H. Low K_m ALDH isozyme and alcoholic liver injury (abstract). Alcoholism: Clinical and Experimental Research 6:319, 1982.

Chapter III

Psychobiological Effects of Alcohol

Alcohol acts on virtually every cell of the body, but the central nervous system is the target most affected. Alcohol's influence is profound--from the personality disarray created by chronic drinking to the distorted judgment of the drunken driver. Current research is converging on explanations for some of these effects and their underlying biological mechanisms, but a comprehensive understanding of an individual's response to alcohol is still elusive.

Because no unitary explanation of alcohol's effect on human behavior seems adequate, researchers continue to explore all levels of brain function and behavior. Accordingly, this chapter presents selected research findings at four broad levels--cognition and neuropsychological effects, neuropathological illness, electrophysiology, and neural and biochemical effects. Finally, a brief overview of theories that may explain the appeal of alcohol and the causes of alcohol addiction are discussed.

Cognitive and Neurophysiological Effects

Driving-Related Cognitive Skills

Our society has acknowledged the profound and often harmful behavioral effects of alcohol, especially as it involves drinking and driving. Driving is a complex task requiring such skills as psychomotor coordination, reaction time, memory, perception, judgment, information processing, and conscious control of behavior. Not surprisingly, many researchers have chosen to investigate alcohol's cognitive effects by measuring how it impairs various driving-related skills. Such studies can yield practical applications while also revealing more basic psychological mechanisms.

Clayton (1980) found that driving skills are impaired at blood alcohol concentrations (.04-.05 percent) one might observe after drinking

only two to three alcoholic beverages on an empty stomach. In addition, he found that the same blood alcohol level decreased a driver's ability to divide his attention among several stimuli while maintaining acceptable performance. Clayton suggested that the resulting diminished performance could be caused by a decreased ability to process information.

Driving a car is a learned skill that requires good judgment based on experience and practice. McNamee et al. (1980) examined how alcohol affects experience and judgment in estimating the velocity of a moving object--an essential task for drivers. Initially, with low doses of alcohol, the subjects actually improved their estimates. But after continuing to drink (resulting in a blood alcohol concentration above .10 percent), their performance declined dramatically. This biphasic phenomenon, of initial stimulation or excitation of the central nervous system with low doses followed by depression as the dose is increased, will be encountered again later in this chapter.

In a study closely related to velocity estimates, Collins and Chiles (1980) investigated how alcohol affects tracking skills in airline pilots. The results confirmed McNamee's study and showed, in addition, that the pilot's errors were greatest and most frequent during evening hours. Thus, it appears that biological rhythms may influence alcohol's effects on judgment. The researchers also found that many hours of abstinence are required to eliminate all such alcohol-induced errors.

Many people believe they can override or control alcohol's negative effects on behavior. Young and Pihl (1980) evaluated the degree to which volunteer subjects could control their speech and behavior and prevent visible symptoms of intoxication. Although the subjects varied greatly in their suppression of symptoms, none was completely successful.

Fatigue impairs cognitive performance in much the same way that alcohol does, leading some researchers to examine the relationship between the two. Ryder et al. (NHTSA 1981)

reported that alcohol increased the cognitive impairments produced by fatigue as determined by driving performance. Caffeine is thought by many to counteract the effects of both alcohol and fatigue. Moskowitz and Burns (1981), using a driving simulator, evaluated caffeine as a possible antidote to alcohol in tracking, searching, and information processing. They found that caffeine could partially counteract the behavioral effects of alcohol. However, since the effects were marginal, these findings should discourage the folk belief that coffee is an effective antidote to alcohol, especially when driving. In a study of another often-used combination, Tong et al. (1980) found that both tobacco and alcohol individually diminished auditory vigilance (ability to detect sound stimuli); taken together, they led to even greater impairments.

Using an animal research design that is likely to reflect human reactions, Moody et al. (1980) demonstrated that alcohol significantly slowed monkeys' reaction time to sound signals. Recordings of electrical activity in the brain's auditory pathways showed that alcohol had not impeded the signal from the ear to the brain. Rather, the impairment seemed to be caused by slow information processing in the brain's "higher" centers.

Cognitive Functioning in Social Drinkers

Since this topic was reviewed in previous reports to Congress (USDHHS 1981; Parsons and Leber 1982), MacVane et al. (1982) have also found that performance on some neuropsychological tests correlates with social drinking patterns. Five studies have found some statistical relationships between alcohol use and decreased cognitive performance in social drinkers (Parker 1982). However, as Parsons and Fabian (1982) have pointed out, such relationships were not consistent for the same cognitive tests in each of these studies. Furthermore, in the MacVane study, although the 58 heavy social drinkers drank three times as often and twice as much as did the 48 light-moderate drinkers, the performance of the two groups on the cognitive tests was not significantly different.

Methodologic issues may partially explain the lack of a dose-response relationship between consumption and cognitive ability (Parker 1982). However, variables other than alcohol use, as yet unknown, may also be involved (Parsons and Fabian 1982). Individuals with poor cognitive function, for example,

may drink more than those with high functioning; the former, due to poor verbal skills, may provide less valid drinking histories. Heavy drinkers may be more anxious and depressed perhaps due to underlying problems or the recency of drinking. Finally, individuals may have to consume a minimal amount of alcohol (threshold) before it has detrimental effects.

As more sensitive techniques for assessing neuropsychological functioning have been applied to individuals with differing social drinking patterns and socioeconomic circumstances, it has become increasingly clear that many questions must be answered. Specifically, what are the limits of normal functioning? At what point should an individual be considered impaired due to alcohol use? Is alcohol use a causative or an exacerbating factor, a complication of or simply coincidental to such measured impairments? The social use of alcohol is sufficiently widespread in our culture, and the degree of behavioral impairment attributed to social drinking is difficult to measure. Therefore, long-term studies of many individuals with varied drinking patterns will be needed to clarify the cognitive consequences of social drinking.

Alcohol and Aging

On average, older persons perform less well on standard psychological tests than younger ones with equivalent patterns of alcohol consumption (Carlen et al. 1981b). However, people of the same chronologic age may age quite differently physiologically.

Because young alcoholics resemble old people in their performance on neuropsychological tests, some researchers have hypothesized that alcohol consumption may accelerate the normal process of aging (Ryan 1982). However, electrophysiologic data, particularly brain evoked potentials (electrical activity in response to sensory stimulation), suggest that the organic brain disease of alcoholics is caused by an underlying mechanism different from premature aging (Porjesz and Begleiter 1982).

Whether or not the two processes share a common cause, alcohol consumption may interact with aging in several ways (Freund and Butters 1982). Changes in their ability to metabolize alcohol and in their central nervous system function may make the elderly increasingly vulnerable to the deleterious effect of alcohol (Wood and Armbricht 1982). In addition, alcohol may interfere with normal

regenerative processes of the central nervous system (West et al. 1982).

Alcoholics Versus Nonalcoholics

All of the previously discussed human studies examined alcohol's effects on the behavior of individuals from the population at large. But the population includes both alcoholics and nonalcoholics, who may respond to alcohol differently. Recent research has attempted to measure alcohol's cognitive and neuropsychologic effects on alcoholics compared with nonalcoholics. Further, it has examined both the effects of acute (short-term) doses of alcohol and the impact of chronic drinking.

Cognitive Effects of Alcoholism

O'Leary et al. (1980) found that alcoholics performed significantly worse than did nonalcoholics when tested for their ability to note and distinguish slight differences between closely related objects. In a study that could explain the results obtained by O'Leary and his associates, Rubin (1980) found that, compared with nonalcoholics, alcoholics exhibit various autonomic nervous system difficulties, including poor physiological regulation of eye pupil size and the rate at which pupils respond to visual stimuli. Such poor regulation could hamper alcoholics in distinguishing between closely related objects.

Several studies have suggested that while alcoholism does impair cognitive skills, alcoholics regain such skills quite rapidly after they stop drinking. Chaney et al. (1980) confirmed the earlier studies, adding that younger alcoholics regain their verbal skills after detoxification more rapidly than do older alcoholics.

Ryan (1980) found that, at the beginning of a testing session, alcoholics and nonalcoholics performed equally well in a short-term memory test. But, when asked to recall words after a 3-hour interval, the alcoholics remembered significantly fewer words than did the nonalcoholics, possibly as a result of more mental fatigue.

Neuropsychological Performance in Alcoholics

Brain abnormalities underlie cognitive impairment in alcoholics (Wilkinson 1982). In the

largest long-term study of computer-assisted tomography (brain scan) changes in alcoholics, all brain scan measurements differed significantly between alcoholics and controls. None of the alcoholics had clinically overt signs of brain damage, but all showed the syndrome of alcohol dependence (Ron et al. 1982).

Alcoholics over 43 years of age differed from their controls more markedly than did the younger alcoholics. Brain scan measurements were significantly correlated with age, but not with such measures of alcoholism as length of drinking history, measures of amount consumed, or scores on a "social decline scale."

In the older subjects, abnormalities seen by brain scans were least severe in those who had abstained longest before scanning. In addition, it was noted that recovery proceeded at a faster rate in younger alcoholics compared with older alcoholics. In followup tests, from 30 weeks to 3 years after initial examination, the largely abstinent patients showed significant improvement over time in brain scans, while the continuing drinkers showed little change. The length of abstinence was correlated with the degree of improvement.

The work of Ron and his colleagues (1982) suggests that neuropsychologic deficits may be reversible. However, Adams et al. (1980) were unable to demonstrate improved neuropsychological performance in patients in their late thirties from an alcoholism treatment program tested 3 weeks after their last drink and again 1 year later. Posttreatment alcohol consumption by 63 percent of these patients may have influenced this study, however, as Eckardt et al. (1980) noted that abstainers perform better than those who resume alcohol consumption. These results emphasize that total abstinence is desirable if alcohol-related cognitive deficits are to be reversed. Reviews of existing literature noted, however, that many studies fail to assess alcohol intake accurately during so-called periods of abstinence (Orrego et al. 1979; Parsons and Leber 1982).

Nutrition and Alcoholism

Because alcohol is a concentrated source of energy (about 200 calories per ounce), alcoholics often find their caloric needs largely satisfied by the alcohol they consume but develop deficiencies of vital nutritional elements. Indeed, most medical textbooks recommend restoration of a proper diet with vitamin supplementation as a first-order treatment of alcoholism. In particular, thia-

mine (vitamin B₁) deficiency may result in the damage to brain cells (Blass 1980), and alcohol itself may interfere with thiamine's absorption from the gastrointestinal tract (Hoyumpa 1980).

A genetic enzyme abnormality may largely determine which individuals are most likely to become thiamine deficient during periods of poor nutrition (Leigh et al. 1981). Shaw and his associates (1981) reported impaired learning in thiamine-deficient rats, but not in rats fed a balanced diet or an alcohol-containing liquid diet. Thus, it appeared that thiamine deficiency alone can cause neuropsychological damage.

Other evidence (Walker et al. 1981), however, suggests that, despite adequate diet, chronic alcohol consumption is in itself neurotoxic and causes learning deficits as well as long-lasting brain damage. Clearly, both nutrition and alcohol contribute to neurotoxicity, and their relative roles in alcoholic brain injury remain to be resolved.

Neuropathologic Illnesses

Chronic Organic Brain Syndromes

Chronic consumption of harmful quantities of alcohol may lead to two clinically and neuropathologically distinguishable chronic organic brain syndromes: alcoholic dementia and alcohol amnesic (memory loss) syndrome (also called Korsakoff's psychosis) (Wilkinson and Carlen 1981; Lishman 1981).

Chronic organic brain syndromes due to alcoholism are the second most common of the known causes of dementia in adults (approximately 10 percent) next to Alzheimer's disease (40-60 percent) (Wells 1979). Despite emerging technological advances, a large proportion of dementing illnesses can be diagnosed with certainty only by examining the microscopic structure of the brain at autopsy. For example, alcoholic dementia is characterized pathologically by destruction of cerebral cortical neurons, particularly those in the frontal lobes.

The clinical picture of alcoholic dementia may be indistinguishable from that of Alzheimer's disease. Global intellectual decline, seen in both, is characterized by deficits in abstracting ability and problem solving, dysphasia (deficit in verbal expression), and apraxia (diminished skill in motor tasks).

Electroencephalographic (EEG) abnormalities, cerebral and cerebellar tissue loss, and the enlargement of the fluid spaces in the interior of the brain are also similar in the two disorders. But the overall course of the diseases is potentially different. In alcoholic dementia, cognitive impairment is reversible to some degree with abstinence, although cognitive decline usually occurs with continued drinking (Cutting 1978). Potential reversibility clearly differentiates alcoholic dementia from Alzheimer's disease, which is invariably progressive. Gottfries (1980) suggests that alcoholic dementia may be differentiable from Alzheimer's disease on the basis of abnormal acetylcholine neurotransmission in the latter. This may be difficult to demonstrate, however, due to the many areas of neurochemical overlap among Alzheimer's disease, alcoholic dementia, and aging.

Patients with Korsakoff's psychosis (see USDHHS 1981; Butters 1982), unlike those with alcoholic dementia, suffer severe and often persistent memory impairment while other intellectual functions remain relatively intact. There are marked deficits in acquiring new learning and amnesia for more recent events. Unlike early-stage Alzheimer patients with superficially similar memory deficits, Korsakoff patients can recall rules for organizing information and can recall previously acquired knowledge (Weingartner et al. 1983). At autopsy, Korsakoff patients have characteristic lesions that resemble those described in patients who have died from acute thiamine deficiency (Wernicke's encephalopathy). Those lesions, whose precise relationship to the Korsakoff memory deficits is still controversial (Butters 1982), are near brain pathways that in animal studies have been implicated in learning, memory, drive, and reward. Mair et al. (1982) have found reductions in neurotransmitters, which are correlated with the performance of Korsakoff patients on learning tasks. However, it is unclear whether such neurochemical changes are significantly different from those seen in age-matched healthy controls or in individuals with dementia due to other causes.

Harper (1979) diagnosed 51 cases with the pathological findings of Wernicke's encephalopathy (thiamine-related degeneration of neurons in the brain) or approximately 2 percent of the brains examined from diseased alcoholics at autopsy in a 4-year study. These findings were not appreciably different from those of similar prior studies (Cravioto et al. 1961; Victor et al. 1971). In only 7 of Harper's

51 cases had the clinical diagnosis of Wernicke's encephalopathy or Korsakoff's psychosis been considered during life. The majority of affected patients were known alcoholics; 10 had died suddenly and unexpectedly, apparently as a result of hemorrhages in the part of the brain that controls the heart rate and respiration.

It is disturbing that Wernicke's encephalopathy is so greatly underdiagnosed because it is treatable and preventable with adequate thiamine supplementation. Harper (1979) and Lishman (1981) suggest that the present clinical appearance of Wernicke encephalopathy and Korsakoff syndrome may have changed somewhat from classical descriptions because many alcoholics may now routinely receive thiamine treatment before the clinical signs are obvious. Such treatment, while insufficient to prevent the disease, masks the classical syndrome, thereby making a traditional diagnosis difficult. Thus, clinical symptoms may develop after repeated episodes of barely detectable brain degeneration in which the usual signs of mental confusion, ataxia (loss of body coordination), ophthalmoplegia (eye paralysis), nystagmus (abnormal eye movements), and memory loss are absent or not readily recognized (Kearsley and Musso 1980).

In alcoholics, Alling and Bostrom (1980) have found damage to myelin, the covering sheaths that enhance electrical conduction in some nerve cells. The damage occurred in a part of the brain almost always affected in classical Korsakoff's psychosis. These randomly selected alcoholics never had Wernicke encephalopathy or Korsakoff syndrome in life and did not show the typical pathologic changes of this disorder under microscopic examination at autopsy.

Alcoholic dementia and Korsakoff's psychosis probably represent ends of a continuum, with the majority of patients seen in clinical practice falling somewhere in between and having some characteristics of both (Parsons and Leber 1982; Butters 1982; Harper 1982). It is not uncommon to find the symptoms of alcoholic dementia (cortical atrophy, enlarged fluid space in the interior of the brain, and moderate intellectual decline) in patients with Korsakoff's psychosis. Conversely, those with alcoholic dementia probably have some memory loss and frequently have microscopic findings that are characteristic of Korsakoff's psychosis. Both nutritional deficits and alcohol neurotoxicity may contribute to either chronic organic brain syndrome.

Relationship Between Alcohol Abuse and Psychiatric Illnesses

Drinking in sufficient amounts to cause serious medical, social, domestic, vocational, or legal problems may reflect, occur together with, or lead to various psychiatric illnesses (Martin 1981). Alcohol use itself may produce various forms of psychopathology as a direct result of its pharmacologic actions (Behar and Winokur 1979), through psychosocial consequences of its chronic use (Goodwin and Guze 1979), or by alteration of psychological equilibrium during alcohol withdrawal (Hamm et al. 1979). Depression, anxiety, hysteria, sociopathy, drug abuse, anorexia nervosa, and childhood hyperactivity are more common in alcoholics and in their family members than in the general population (Morrison and Stewart 1971; Overall et al. 1973; Goodwin et al. 1975; Tarter et al. 1977; Andreasen and Winokur 1979; Eckert et al. 1979; Goodwin and Guze 1979; Munjack and Moss 1981; Wood et al. 1983).

Whether psychopathology is the result of, or the cause of, alcoholism is sometimes difficult to determine. There are, however, definite clinical implications for each alternative (Andreasen and Winokur 1979; Hamm et al. 1979; Weissman and Myers 1980; Linnoila and Martin 1983). For example, if alcoholism predates psychiatric symptoms, abstinence from alcohol is indicated before beginning other treatments (Schuckit 1982). If the alcoholism follows psychiatric disease, it is important to diagnose the psychiatric illness accurately so that treatment may be tailored to the patient's needs (Linnoila and Martin 1983).

The considerable overlap between alcoholism and the affective disorders (mania and depression) has become an important area of research. Alcoholism and depression are the two behavior-associated illnesses with the highest rates of suicide (Tsuang 1977). Recent evidence suggests possible common abnormalities in metabolism of the neurotransmitter serotonin in alcoholism, aggression, suicide, and depression (Ballenger et al. 1979; Rosenthal et al. 1980; Brown et al. 1982). Such neurochemical similarities between alcoholism and depression suggest that there may be a common underlying mechanism. Tricyclic antidepressants and lithium—medications for depression and mania—may offer significant therapeutic benefit to alcoholics who are depressed (Coppen 1980).

Indirect Alcohol-Induced Neuropathology

Liver disease associated with alcoholism may result in behavioral deficits ranging from subtle neuropsychologic abnormalities (Smith and Smith 1977; Rehnstrom et al. 1977) to serious brain degeneration, delirium, and coma (Schenker et al. 1980). In addition, head trauma and inadequate oxygen supply to the brain, frequently sustained during intoxication, can become a recurrent theme in the life of an alcoholic, with cumulative and adverse neurologic effects (McLeod 1982).

Alcohol inhibits the respiratory center of the brain and renders the individual more susceptible to obstructive sleep apnea (inability to breathe normally during sleep), an increasingly recognized condition that causes hypoxemia (reduced blood oxygen content) during sleep (Guilleminault et al. 1978). Even mild hypoxemia may adversely affect brain function and lead to dementia, particularly in individuals who have chronic obstructive lung disease, as is often the case in alcoholics (Blass 1980).

Hillbom and Kaste (1982) have reported that both occasional alcohol intoxication and regular heavy drinking carry an increased risk of stroke due to subarachnoid hemorrhage, but the mechanism for this is not yet clear. In addition, alcohol withdrawal may be accompanied by serious metabolic disturbances that can impair cerebral function (Linnoila and Martin 1983).

The common use of other psychoactive drugs by alcoholics may also contribute to behavioral deficits (Linnoila and Martin 1983). Depression, which frequently accompanies harmful alcohol use (Schuckit 1982), can also impair cognition considerably (Weingartner et al. 1981). Cognitive impairment may often precede alcoholism, however. The high prevalence of attention disorders in young male alcoholics (Wood et al. 1983), for example, suggests that part of the cognitive impairment attributed to alcohol-related brain damage was probably present before the use of alcohol (Parsons and Leber 1982).

Electrophysiology

Increasingly over the past half century, electrophysiological recordings have been expanding researchers' knowledge of the nervous system. Until about 25 years ago, such recordings were limited to macroelectrodes, large

probes that recorded only gross brain activities of thousands or even millions of nerve cells. The recordings, called electroencephalograms (EEGs), could indicate the results of either spontaneous brain activity or the brain's responses to a stimulus such as a light flash (evoked potentials). More recent technological advances allow microrecordings from single nerve cells. In today's research, both macro- and microrecordings of alcohol's effects augment one another to contribute to an understanding that neither alone can provide.

In general, the electrophysiological effects of alcohol are complex and vary at different sites within the brain. Most alcohol research with these techniques has attempted to determine functional central nervous system differences between alcoholics and nonalcoholics and, sometimes, to quantify the acute effects of alcohol.

EEG Studies

Recent studies have compared EEGs accompanying the loss of consciousness resulting from alcohol ingestion, normal sleep, and barbiturates (Zornetzer et al. 1982). Although the loss of consciousness caused by most barbiturates and by alcohol superficially resembles normal sleep, the conditions are quite different electrophysiologically.

Surprisingly little detailed information exists on how acute doses of alcohol affect the EEG. Several recent studies have investigated the relation between alcohol's effects on the cortical (outer brain layer) EEG and its effects on EEGs from deep brain structures. Grupp (1981) reported that alcohol altered the electrical activity of both the hippocampus (a deep brain structure associated with memory and emotion) and the cerebral cortex (involved in high-level information processing). Partly on the basis of such studies, Klemm and Sherry (1981) proposed a brain model of intoxication. They theorized that alcohol "scrambles" brain messages, thus disrupting normal communication within the central nervous system. Because Klemm and Sherry rely largely on supporting evidence recorded from the cerebellum (a part of the brain associated with posture, movement, and balance), their model may not account for the pervasive effects of intoxication on other brain structures.

Evoked Response Studies

Electrophysiological responses to sensory stimuli reveal a great deal about the brain.

Because responses to individual stimuli are often lost in a sea of electrical "noise," however, researchers often resort to a technique called the average evoked response (AER). In essence, a readable AER depends on a computer's averaging a large number of the brain's electrical responses to repetitive sensory stimuli. Individual responses that would be imperceptible without computer assistance become magnified and visible when averaged together.

Recently, several researchers have turned to the AER to investigate the brain's response to sensory stimuli under various conditions of alcohol use. Studies showed that alcohol disrupted (1) the normal AERs to visual stimuli (Hetzler et al. 1980); (2) the normal patterns of attention to visual stimuli (NHTSA 1980); and (3) the normal AERs to auditory stimuli (Pfefferbaum et al. 1980), which were correlated with slowed reaction times to sound signals. Such disrupted electrophysiological responses to perceptual stimuli could explain alcohol's adverse effects on driving.

Many investigators have attempted to detect whether AERs in alcoholics differ from those in nonalcoholics. A frequently cited study demonstrated that, compared with non-alcoholics, alcoholics produce higher amplitude AERs when presented with light flashes of low intensity, even though these alcoholics had been abstinent from alcohol for 1 week or more (Buchsbaum and Ludwig 1980). When the flash intensity was increased, alcoholics' responses increased more than those of normal subjects. Thus, the alcoholics' AERs were somewhat greater than normals' at low intensities, but much greater at high intensities. Buchsbaum and Ludwig (1980) noted that alcoholics, in increasing their AERs at higher light intensities, are more sensitive to higher level stimuli. They speculated that alcoholics may be neurophysiologically vulnerable to intense sensory inputs either by predisposition or as a result of the toxic effects of alcohol, and that perhaps they use alcohol to diminish their augmented sensitivity.

One provocative question is whether differences seen in the AER are consequent to, or predispose for, alcoholism. Recent research indicates that there may be inherited differences in the brain's electrical activity. Elmasian et al. (1982) observed that AERs from groups of individuals with and without a family history of alcoholism were markedly different. These differences were apparent whether or not the individuals had consumed alcohol. Electrophysiological methods may, therefore,

prove to be valuable in future research, especially in identifying specific indicators or markers of alcohol susceptibility.

Neural and Biochemical Effects

Behavior and thought, learning and memory, sensations and movements, all of which are affected by drinking alcohol, find their biological basis in the nervous system. The individual units of this system are neurons (nerve cells).

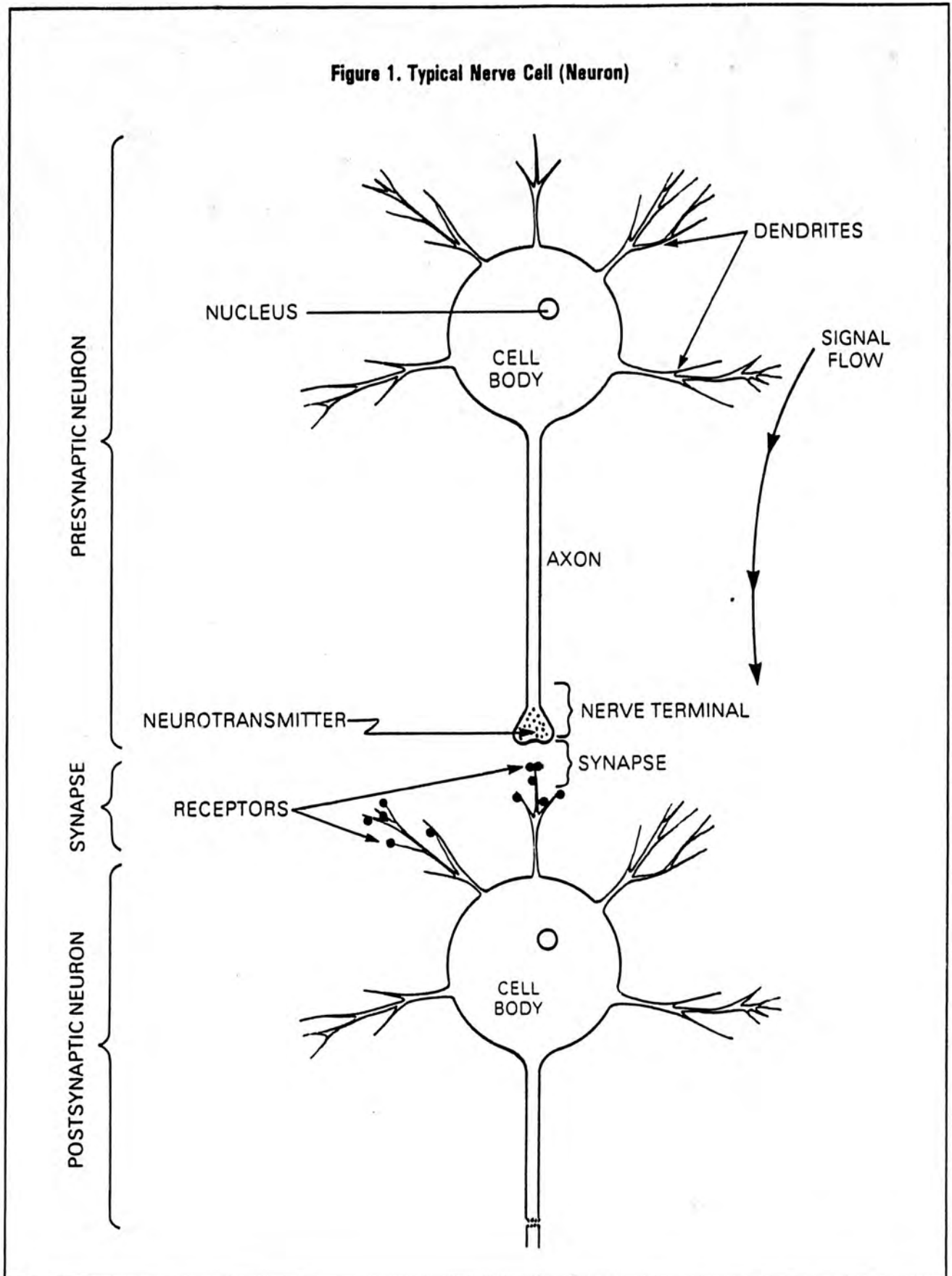
Neurons are now known to be extraordinarily subtle electrochemical processors. New knowledge of neurons, accompanied by new measurement techniques, has encouraged some optimism that a deeper examination of alcohol's effects on neurons could reveal how alcohol affects the nervous system and, in turn, how the nervous system affects behavior. Before discussing alcohol's effects on neurons, it is necessary to understand what a neuron is and how it works.

Neurons are, in computer parlance, logic elements. They conduct information and, based on a combination of electrical and chemical signals, communicate with other neurons, with muscles, and with the body's endocrine system hormones.

A typical neuron (shown highly simplified in figure 1) usually receives chemical inputs, through its dendrites, from other neurons. It changes the chemical signal into an electrical signal, which travels to the other end of the neuron (the nerve terminal or output end) where neurotransmitter chemicals, which continue to carry the signal, are released across a space called the synapse to adjacent neurons. The process is usually repeated many times to create signal paths throughout the nervous system. Essentially, the transmission between neurons is chemical, while the transmission within neurons is electrical.

If we probe into the nature of the electrical signal that is carried along the neuron, we find that it is formed by the movement of sodium and potassium ions (charged particles) flowing across the thin semipermeable membrane enclosing each nerve cell and crossing back again. When the flow of these ions reaches threshold conditions, an impulse is propagated down the neuron to its destination, the nerve terminal. Instruments can detect these short electrical pulses as the electrical signal travels through each neuron. Alcohol could affect the propagation of electrical pulses by making the membrane more or less permeable

Figure 1. Typical Nerve Cell (Neuron)



to the charged particles. After a pulse is propagated, the neuron must be recharged before it can carry another electrical impulse. This process is regulated by an enzyme that moves ions that entered the neuron back outside. Alcohol may also interfere with this ion "pump" mechanism, which maintains the electrical stability of each neuron.

When the electrical impulses reach the nerve terminal, calcium ions, acting as gatekeepers of the synaptic membrane, enter the neuron and allow tiny packets of neurotransmitter chemicals to be released across the synapse. On the adjacent neuron, receptors exquisitely matched to the neurotransmitter chemical characteristics will recognize the chemical message and unlock the membrane of the receiving neuron to allow the signal to continue. Continuation of the signal, inside the receiving neuron, depends on increased flow of sodium and potassium ions to reach the threshold conditions for generating an electrical impulse and allowing the whole process to repeat. (Depending on the neurotransmitter chemical, however, the receiving neuron may be inhibited from firing, rather than being fired. In this manner, the nervous system can turn off, as well as turn on, the flow of information.) This activity can occur hundreds of times a second and in billions of neurons in the brain. The result is thoughts, feelings, learning, or the link between a red light and muscles flexing to step on the brake. Any interference with the neurotransmission process could affect that link.

Electrical Conduction

In the late 1940s, it was found that sodium and potassium ions were responsible for the electrical conduction in the neuron. Researchers interested in alcohol's effect on the brain hypothesized that alcohol could impede the movement of ions necessary to produce and conduct the electrical impulse. Early studies on the squid's giant axon confirmed the hypothesis, but only at alcohol levels four to five times the lethal concentration (Gallego 1948). Therefore, many researchers concluded that alcohol's ionic effects could not explain why or how drinking alcohol could affect the nervous system. The hypothesis seemed to lead to a dead end. With time, new technology and instrumentation have allowed a more detailed view of the brain and individual neurons. After examining and recording the electrochemical activity of neurons throughout the brains of various mammals, it was found that a neuron

is not simply a standard element repeated billions of times in the brain. Rather, a wide diversity exists among neurons—in speed of conduction, size, number of inputs, and chemical makeup; therefore, the case for alcohol's effect on neurons' electrical activity was reopened. It was realized that a differential sensitivity to alcohol might result because of differences between neurons.

Recently, researchers (Carlen et al. 1982; Shefner et al. 1982) have demonstrated that alcohol diminishes electrical activity in parts of the brain of mice and rats. In particular, neurons in the hippocampus and locus ceruleus, deep inside the brain, are affected by alcohol concentrations below those that might be expected in a person after several cocktails.

The differential sensitivity of various brain areas to alcohol seems to be genetically influenced. In a line of mice (LS) bred for sensitivity to the effects of alcohol, the hippocampus was less sensitive than another brain area, the cerebellum. These two brain areas were equally sensitive, however, to the effects of alcohol in another line of mice (SS), bred for resistance to alcohol's effects (Sorenson et al. 1981; Basile et al. 1982). Comparisons between strains also clearly demonstrated a genetically influenced differential response to alcohol. Neurons in the cerebellum of the mice resistant to the disruptive effect of alcohol were less affected electrophysiologically by alcohol (Sorenson et al. 1980, 1981; Basile et al. 1982) than were those from the sensitive mice.

The nature of the effects of alcohol on electrical activity led Carlen and his coworkers (1982) to postulate that alcohol altered, in a complex way, the movement of potassium ions across the neuronal membrane.

Another neuronal component related to the electrical activity of the cells is the enzyme sodium-potassium-ATPase. This enzyme, the ion "pump," is responsible for maintaining the proper concentrations of sodium and potassium ions on either side of the neuronal membrane. Alcohol alone in nonlethal amounts does not affect the enzyme activity. When combined with the neurotransmitter chemical norepinephrine, however, brain alcohol concentrations that could be achieved by beverage consumption can inhibit the enzyme. Such inhibition would be expected to affect the conduction of electrical impulses (Rangaraj and Kalant 1979). Thus, alcohol's effects on the locus ceruleus, which has norepinephrine-containing neurons, could be related to inhibition of the enzyme.

As mice and rats become more tolerant to the sedative effect of alcohol, the sodium-potassium-ATPase becomes more resistant to alcohol's inhibitory effects (Levental and Tabakoff 1980; Rangaraj and Kalant 1982). Such a change suggests that alcohol tolerance may, in part, be based on neurochemical changes in the brain. Clearly, more information is required to establish unequivocally the link between alcohol, electrical activity in the brain, and behavioral responses to alcohol.

Neurotransmission

Chemical messages carried across the synapses—the tiny spaces between adjacent neurons—are critical to brain function. Any interference with the release of neurotransmitters, their recognition by receptors, or the development of the new signal in the adjacent neuron will affect the flow of information in the brain. Without chemical transmission, brain function stops. Therefore, various researchers have examined the chemical flow at the synapse to determine the potential effects of alcohol on neuronal activity. Measurement of neurotransmitter release and metabolism represents an alternative to direct measurement of electrical activity, since the rate of neurotransmitter release and synthesis usually reflects the electrical activity of neurons.

The release and recognition of chemical neurotransmitters between neurons propagates messages through the brain. Neurochemicals are allowed to pass through the nerve cell membrane by a mechanism that is dependent on the movement of calcium ions into the neuron. Could alcohol interfere with this process? Friedman et al. (1980) found that alcohol stimulated the initial rate of calcium accumulation in a special preparation of neuronal membranes deprived of calcium. In another study on mice (Harris and Hood 1980), alcohol inhibited the flow of calcium into a neuronal membrane preparation that was enriched with calcium. Furthermore, this latter effect was detectable only under conditions that mimic the electrical signals' arrival at the synapse. In effect, when there was a message and no calcium ions, alcohol quickly mobilized a new calcium cadre. With an excess of calcium ions, however, alcohol inhibited the calcium stimulated release of neurotransmitter.

Recent studies (Stokes and Harris 1982) have found that the inhibitory effects of alcohol on calcium uptake depend on the part of the brain that is examined. Samples from rat

striatum and cerebellum (both associated with movement) were more sensitive to alcohol's effects than were samples from the cortex and brain stem. These results confirm again that, even at the chemical level, different brain areas seem to respond according to their own programs.

Perhaps more important, neuronal preparations from mice chronically fed alcohol resisted alcohol's effects on calcium uptake, regardless of what the initial alcohol effects were. This evidence suggests a possible direct link between behavioral tolerance of alcohol and the microenvironment of the neuron.

The aforementioned studies measured alcohol's effects on the uptake of calcium—that is, the movement of calcium ions into the neuron. Calcium is also stored within cells near the membrane in a kind of chemically adhesive process called binding, which is also affected by alcohol. Low levels of alcohol increased the binding of calcium, whereas higher levels were not as effective (Michaelis and Myers 1979). As with calcium uptake, alcohol had little effect on calcium binding when added to neuronal preparations from rats fed a chronic diet containing alcohol. Thus, this study also indicates a link between behavioral and biochemical tolerance. It is, however, premature to conclude that behavioral tolerance is the direct result of alcohol's effects on calcium—whether related to calcium uptake or binding.

Evidence linking neurotransmitters to behavior led alcohol researchers to investigate three neurotransmitter systems: norepinephrine, dopamine, and GABA (gamma-aminobutyric acid). They found that alcohol affects the release of the neurotransmitters as well as their recognition by receptors at the post-synaptic membrane (Bacoupoulos et al. 1978; Ticku and Burch 1980; Rabin et al. 1980).

When alcohol is acutely given to an animal, the amount of norepinephrine released depends on the level of alcohol in the brain. Moreover, the process is biphasic: It reverses with larger amounts of alcohol (Hunt and Majchrowicz 1974). That is, a small amount of alcohol leads to an increased release of norepinephrine, while higher doses actually depress the release of norepinephrine. Since release of a neurotransmitter reflects nerve activity, this suggests that low doses of alcohol have an excitant or disinhibiting effect, while high doses have an inhibiting effect on neurons containing norepinephrine. These effects on norepinephrine release may, in turn, be related to the behavioral changes produced by alcohol. Sim-

ilar results were obtained when the effects of alcohol on dopamine were examined in rats (Hunt 1981). In mice, however, the reaction is reversed (Kilianmaa and Tabakoff 1983). Low doses of alcohol lead to low release of dopamine, whereas higher doses lead to greater release. The causes of these different reactions to acute infusions of alcohol in closely related species are not yet understood; they may, however, be related to different organization of the nervous system of these species. In both species, the effect of chronic doses of alcohol is consistent, leading to increased release of norepinephrine and decreased release of dopamine (Hunt and Majchrowicz 1974; Pohorecky 1974).

Over the years, laboratory animals have been bred with specific characteristics in mind. One strain of mice has been developed for its relatively high resistance to the behavioral effects of alcohol. These mice are also more resistant to the effects of alcohol on dopamine release (Kilianmaa and Tabakoff 1983). This finding suggests that some animals may be genetically more tolerant of alcohol than others, and that differences in behavior may be related to differences in biochemistry.

The preceding research collectively shows that alcohol's effects on norepinephrine and dopamine release, as well as on movement of calcium ions, vary with dosage, species, genetic strain, and brain area.

The discussion thus far has focused on the neurotransmitters at the transmitting neuron. Across the synapse, the receiving neuron with its receptors must recognize the neurotransmitters. Much of the research at the receptor end has been devoted to GABA, a neurotransmitter that inhibits firing in neurons and is of particular interest to alcohol researchers. By inhibiting electrical impulses, it controls the tendency to convulsions, which are caused by uncontrolled electrochemical activity in the brain. Because the symptoms of alcohol withdrawal include convulsions, GABA is a prime suspect as a cause of these symptoms.

In fact, research strongly implicates GABA receptors in alcohol withdrawal symptoms. In mice, an acute dose of alcohol results in an increase in the number of GABA receptor sites (Ticku and Burch 1980), suggesting lessened neural activity. This finding is consistent with alcohol's depressive effects. Chronic administration of alcohol results, conversely, in a decrease in the number of GABA receptor sites—a finding that could explain convulsions in withdrawals. Indeed, in rats, the decreased number of receptor sites was most pronounced

at the time of the most severe withdrawal symptoms.

Clinical results demonstrate that benzodiazepines such as diazepam (Valium[®]) and chlordiazepoxide (Librium[®]) control the major symptoms (especially convulsions) of alcohol withdrawal. Specific receptors in the nervous system for the benzodiazepines are related to GABA receptors. Part of this relationship appears to be a common effect on the flow of chloride ions across the nerve membrane. It appears that alcohol disturbs the natural linkage between GABA and diazepam. The strongest evidence for this effect comes from studies in which the receptors were removed from their normal membrane environment (Ticku and Burch 1980).

Recent studies have demonstrated that chronic feeding of alcohol to animals affects the receptors for other neurochemical messengers, including the endogenous opiates (Hoffman et al. 1982), acetylcholine (Tabakoff et al. 1979), norepinephrine (Rabin et al. 1980), and dopamine (Barbaccia et al. 1982).

After the receptors have recognized the neurotransmitter message, a new signal is generated within the receiving neuron. A chemical (cyclic adenosinemonophosphate or cAMP) sometimes called the second messenger carries the message from the receptors to the interior of the receiving neuron. Recent research indicates that after withdrawal of animals from chronic alcohol treatment, the release of the second messenger is inhibited (Tabakoff and Hoffman 1979; Luthin et al. 1983).

As the number of identified neurotransmitters increases (some researchers expect it to go from the current 40 to as many as 200 or even more), and with growth in understanding how they work, neurotransmission is likely to help explain the nature of alcohol's biological effects on the central nervous system.

The Membrane

The transmission of information between nerve cells is dependent upon the function of receptors, enzymes, and ion gates. All of these components are proteins located in and on the neuronal membrane. Changes in the physical state of these membranes could have adverse effects on the ability of the specialized proteins to function normally. A number of researchers are studying this problem and in particular are examining the lipids in membranes, which are the biochemically important elements in maintaining the structural integ-

ity of the membrane. Results have clearly shown that membrane viscosity is decreased by acute treatment with levels of alcohol that can be attained through consumption of alcoholic beverages (Chin and Goldstein 1977). After chronic treatment of mice with alcohol, however, it was found that subsequent acute doses of alcohol would no longer change the viscosity of neuronal membranes (Chin and Goldstein 1977). This suggests that behavioral tolerance to alcohol may, in part, be associated with a tolerance of membrane lipids to alcohol-induced decrease in viscosity.

Goldstein et al. (1982) compared alcohol's effects on neuronal membranes from two different genetic strains of mice: LS mice (long sleep, with greater sensitivity to alcohol) and SS mice (short sleep, with less sensitivity to alcohol). They found that neuronal membranes taken from LS mice were more sensitive to alcohol than were membranes taken from SS mice. Another study (Lyon et al. 1981) showed that alcohol's well-known sedative effect is correlated with its effects on neuronal membranes. In turn, these membrane effects would be expected to influence the function of the specialized proteins that reside in the membrane and are critical for proper neuronal transmission.

Tolerance and Dependence

Alcohol produces tolerance in chronic drinkers—that is, the subjective effects of alcohol decrease with continued exposure. It may take a chronic drinker a pint of whisky to obtain the same effect that an occasional drinker gets with a cocktail or two. Physical dependence—the need to continue to drink alcohol to prevent withdrawal symptoms—may be related to tolerance, as it develops only after chronic ingestion of alcohol.

At one time, it was thought that tolerance and dependence would develop only after relatively long periods of exposure. A recent study (Gallagher et al. 1982), however, found that tolerance to alcohol's ataxic effects (loss of muscular coordination) developed after only 2 days. The fairly straightforward behavioral effects of tolerance and dependence have yet to be described fully; biological mechanisms underlying these phenomena are currently even more obscure.

Recent studies suggest that learning may play an important role in developing tolerance, and several investigators have proposed a Pavlovian model of alcohol tolerance (Crowell et al. 1981; Melchior and Tabakoff 1981). Ac-

cording to this model, animals learn to associate environmental cues with the pharmacological effect of alcohol, much as Pavlov's famous dog associated a bell with food. In the presence of those cues, an animal may develop a response designed to counteract the effect of alcohol. In essence, the Pavlovian model suggests that animals learn to reduce their response to alcohol in a particular context, which provides the cues for their response. Tolerance is reduced or absent, however, when the animal is subsequently tested in a different environment. Thus, in some cases, the mere exposure of an animal to alcohol is not sufficient to cause tolerance development; the initial environmental cues must be repeated (Crowell et al. 1981).

Neurochemical studies have also suggested that the presence of alcohol, while necessary, is not a sufficient condition for the development of tolerance; neurons containing the neurotransmitters norepinephrine and serotonin seem to play a role in developing tolerance. When those neurons were destroyed, animals tended to retain their initial responses to alcohol (Tabakoff and Ritzmann 1977; Le et al. 1981) or to develop tolerance more slowly. Thus, the activity of specific neuronal systems, in conjunction with alcohol, seems to be quite important for tolerance development. In addition to neurotransmitter systems, certain hormonal systems appear to affect the development of tolerance. Vasopressin (sometimes called antidiuretic hormone) can maintain an already developed tolerance, even in the absence of continued exposure of the organism to alcohol (Hoffman et al. 1978). Vasopressin's ability to maintain tolerance depends on the presence of intact norepinephrine—and/or serotonin-containing neuronal pathways (Le et al. 1982; Hoffman et al. in press). Whether vasopressin plays a role in developing alcohol tolerance under normal conditions is not clear, but in at least one study, a strain of rats lacking this hormone could not develop alcohol tolerance (Pittman et al. 1982).

Changes in neuronal membranes could account for tolerance and physical dependence. Alcohol initially increases membrane fluidity, but membranes from alcohol-tolerant animals are less sensitive to this effect. Also, enzymes in the membrane demonstrate a biochemical tolerance to alcohol (Levental and Tabakoff 1980; Rangaraj and Kalant 1982). Membrane fluidity could affect sodium-potassium-ATPase activity and therefore, indirectly, electrical conduction in the brain. The mem-

brane's biochemical adaptation could be related to behavioral tolerance.

Physical dependence, like tolerance, may result from an adaptation of neuronal membranes during chronic alcohol exposure. This adaptation might be expected to produce inappropriate neuronal activity in the absence of alcohol. If so, when alcohol is withdrawn, the membranes of physically dependent animals might be more viscous than normal. Although initial studies did not reveal such changes in animals' brain cell membranes following chronic alcohol consumption (Chin and Goldstein 1977), more recent studies have confirmed the expectations (Lyon and Goldstein 1983). Another study (Ingram 1982) also cataloged a number of changes in lipid (fat) content of brain cell membranes derived from chronically alcohol-fed animals, which could contribute to changes in fluidity. These adaptive changes could well be responsible for some aspects of physical dependence and tolerance.

Tolerance and dependence are complex phenomena, however, and may result from different but interacting biochemical mechanisms. As tolerance and dependence are studied in more detail, it becomes increasingly clear that they are influenced by various neuronal and hormonal systems.

Theories of Alcohol's Effects

Alcohol as Reinforcer

Since the publication of the Fourth Special Report (USDHHS 1981), a new hypothesis has emerged to explain the mechanism behind alcohol's reinforcing activity. Most earlier studies focused on specific neurochemical and behavioral correlates of alcohol's depressive effects. Although the stimulant effects had been noted in the past (USDHEW 1974, 1978; Hunt and Overstreet 1977; USDHHS 1981), most earlier studies lacked the theoretical focus that has emerged in recent years. Now much of alcohol's appeal is thought to be directly related to its excitatory effects, and many current studies are attempting to verify the relationship (Taberner 1980; Lindman 1982).

Essentially all studies of alcohol as a drug of abuse are based on the premise that humans and animals consume alcohol because of its reinforcing (or rewarding) effects. Recent years have seen considerable interest in the

similarities between alcohol's reinforcing properties and those of other drugs of abuse. But how does reinforcement work? Many researchers have noted the similarity between the reinforcing properties of alcohol and those of opiate drugs (Altshuler 1982; Amit and Brown 1982). They have reasoned, therefore, that drugs (opiate antagonists) that nullify the brain's reaction to opiates may well block similar reactions to alcohol. The research results have been somewhat difficult to interpret. In some cases, the opiate antagonist blocked a specific effect of alcohol, while in others the opiate antagonist was ineffective.

Striking similarities have been reported between the electrophysiological effects of alcohol and opiates, especially opiate peptides naturally occurring in mammals (Siggins and Bloom 1980; Siggins et al. 1982; Hoffman et al. 1982). Furthermore, in rats and mice, chronic alcohol doses caused significant changes in the characteristics of opiate receptors (Hoffman et al. 1982). All of these reports confirm the link between alcohol and the opiate system in the central nervous system. In general, it appears that, while opiate antagonists do not affect alcohol's depressive properties, they can alter some of its stimulant or excitatory properties.

Such findings suggest a possible relationship between alcohol's excitatory effects and the phenomenon known as reinforcement. Drinking studies conducted with almost every laboratory species have provided many demonstrations that alcohol is a reinforcer in animals as it is in humans.

Some researchers contend that the most appropriate way to study alcohol reinforcement in laboratory animals is to study their voluntary oral alcohol consumption. Yet the usefulness of oral alcohol consumption studies in animals for measuring alcohol preference or reinforcement has been a persistent source of disagreement among researchers. Advocates contend that since humans consume alcohol by drinking it, oral studies are most likely to provide meaningful results. Opponents argue that when drugs, including alcohol, are taken orally, animals will choose them based on flavor rather than for their reinforcing effects. The problem of estimating or controlling how flavor affects oral intake studies has plagued researchers for many years, but a recent study (Crawford and Baker 1982) suggests a method to distinguish between the reinforcing effects of flavor and alcohol.

Because drinking studies in animals are often difficult to conduct, a technique called

self-administration was developed to substitute for oral consumption. The technique allows animals to work for alcohol by pressing a lever to obtain intravenous (directly into a vein) or intragastric (into the stomach) alcohol infusions through surgically implanted cannulae (tubes). Early studies (Deneau et al. 1969) demonstrated unequivocally that monkeys would self-administer alcohol intravenously. Altshuler (1982) showed that monkeys would also self-administer alcohol intragastrically for extended time periods. Both investigations showed that many animals would work for alcohol over time periods sufficiently long to develop tolerance, physical dependence, and eventually, alcohol-related toxicity.

Another less direct technique to measure alcohol's reinforcing effects is the intracranial self-stimulation (ICSS) model. Stimulating electrodes are placed in parts of the brain where electrical stimulation presumably produces pleasurable sensations. The animals, usually rats, are able to press a lever to activate the stimulating electrode in their brains. Most animals will press the lever frequently to obtain the apparently pleasurable sensation that follows. This ICSS phenomenon is thought to be associated with many different reinforcers, perhaps through a common pathway or center of reinforcement. Indeed, most addictive drugs seem to affect ICSS. Because alcohol (and other reinforcing drugs) changes the rate at which animals will press the lever during ICSS, a number of researchers (Wise 1980; Amit and Brown 1982) have concluded that the drugs were acting as reinforcers.

A controversy has emerged over the neurochemical mechanism responsible for alcohol's reinforcing properties in the ICSS model. Most researchers agree that alcohol is a reinforcer and that reinforcement is controlled by the brain's reward system (Wise 1980; Amit and Brown 1982). Wise (1980) hypothesized that alcohol affects dopamine's role in information transfer within the reward system. Amit and Brown (1982) suggested that norepinephrine rather than dopamine is affected. In either event, alcohol reinforcement is probably associated with a specific cluster of neurons that may also be the site of the reinforcement produced by several drugs and ICSS. The experimental evidence implicates several neurotransmitters, including dopamine, norepinephrine, and opiate peptides, as the neurochemicals responsible for the reinforcing properties of alcohol.

Alcohol Metabolism and TIQs

Another theory about alcohol and its actions is based on the hypothesis that biologically active chemicals called tetrahydroisoquinolines (TIQs) may be formed during alcohol metabolism (Davis and Walsh 1970). Interest arose in these compounds because some of the TIQs are structurally related to morphine (an opiate) and, therefore, could involve a common addictive mechanism.

About 6 years ago, Myers and Melchior (1977) proposed a provocative hypothesis relating TIQs and alcoholism. The hypothesis was based on the observation that infusions of a TIQ, tetrahydropapaveroline (THP), into a brain cavity changed an animal's drinking behavior from rejecting alcohol to choosing moderate doses. It was speculated that excessive drinking by human alcoholics could be caused by metabolic abnormalities that result in the formation of TIQs. They reasoned that because drinking creates TIQs, and TIQs stimulate drinking, the positive feedback loop could explain uncontrolled drinking by alcoholics. That conjecture generated both support and disagreement, which continue unabated to this day. Several research groups (Sinclair et al. 1982; Tuomisto et al. 1982) attempted to replicate the initial studies with, at best, only mixed results. Myers et al. (1982), however, reported recently that TIQ infusions produced outcomes in monkeys identical to those in rats, and that THP infusions produced increased alcohol drinking by monkeys. However, these infusion studies use TIQ that is synthesized in the laboratory. While it is theoretically possible that TIQ is formed in the brain, a great deal of controversy remains as to whether TIQs are in fact formed in the brain, or if the concentrations of these chemical compounds are increased following alcohol ingestion. A number of investigators have searched for these compounds using increasingly sophisticated and sensitive techniques, with only limited success (Bloom et al. 1982).

A less controversial hypothesis relates endogenous (naturally produced in the brain) opiate mechanisms to alcohol's behavioral effects. These studies have investigated possible pharmacological relationships between TIQs, endogenous opiate compounds, and the reinforcing properties of alcohol. Most have reported that the reinforcing properties of alcohol and opiates could be related to TIQ mechanisms.

In future studies, many researchers will probably turn their attention to the biphasic effects of alcohol and may reexamine many assumptions about its effects on behavior. Additional research about alcohol reinforcement, preference, and voluntary consumption is likely to illuminate the role of the TIQs, opiate mechanisms, and alcohol-induced stimulation. These data may provide an understanding of the underlying biological mechanisms that contribute to alcohol abuse.

Summary

Alcohol affects every level of the nervous system—from the neurochemicals within single cells to the macrofunctions governing thought processes and behavior. Research on cognition and brain responses to various stimuli continues to expand the inventory of alcohol's effects on humans. Driving-related studies have shown that alcohol impairs judgment, attention, information processing, and reaction time. Alcohol also alters the brain's responses to visual and auditory stimuli. Chronic drinking affects memory and the ability to detect small differences and creates such autonomic difficulties as lack of proper eye pupil control. Abstinence can reverse alcohol's cognitive effects more easily in young drinkers than in older ones.

Social drinking appears to result in some cognitive impairment, but methodologic issues cloud the picture. Alcoholism, on the other hand, definitely impairs cognitive performance. Late stages of alcoholism may also result in organic brain damage—alcoholic de-

mentia and Korsakoff's psychosis.

Alcohol may be the indirect cause of other neuropathology. Liver damage, head trauma, malnutrition, and breathing impairment during sleep, resulting from alcoholism, can lead to a range of adverse effects from behavioral deficits to serious brain damage.

In an effort to understand the neuropsychological effects of alcohol, researchers have examined the effects of alcohol on structures and neurochemicals in various regions of the brain. Chronic drinking seems to cause several biochemical adaptations in nerve cells that could partially explain why alcoholics become tolerant to alcohol and why they suffer withdrawal symptoms after sudden abstinence.

In a search to explain why people drink alcohol to excess, researchers have proposed several theories. One theory suggests that alcohol's appeal stems from its initial excitatory effects. Animal research tends to support the theory, as animals will work to receive infusions of alcohol—thus meeting one criterion of reinforcement.

Another provocative preliminary hypothesis is that tetrahydroisoquinolines, or TIQs, may play a role in habitual heavy drinking. TIQs are formed when alcohol is metabolized. In some animal experiments, when these chemicals are injected into the brains of previously abstinent animals preference for alcohol is increased.

Empirical and theoretical progress over the past decade offers encouragement that research will continue to acquire fundamental knowledge essential to understanding how humans react to alcohol and what makes them drink excessively.

References

- Adams, K.M.; Grant, I.; and Reed, R. Neuropsychology in alcoholic men in their late thirties: One-year follow-up. *American Journal of Psychiatry* 137:928-931, 1980.
- Alling, C., and Bostrom, K. Demyelination of the mamillary bodies in alcoholism. *Acta Neuropathologica (Berlin)* 50:77-80, 1980.
- Altshuler, H.L. Ethanol reinforcement: Self-administration and discriminative stimulus parameters. In: Saito, S., and Yanagita, T., eds. *Learning and Memory: Drugs as Reinforcers*. Proceedings of symposiums on the Pharmacology of Learning and Memory and on Drugs as Reinforcers (1981). Amsterdam: Excerpta Medica, 1982. pp. 240-256.
- Amit, Z., and Brown, Z.W. Actions of drugs of abuse on brain reward systems: A reconsideration with specific attention to alcohol. *Pharmacology, Biochemistry and Behavior* 17:233-238, 1982.
- Andreasen, N.C., and Winokur, G. Secondary depression: Familial, clinical, and research perspective. *American Journal of Psychiatry* 136: 62-66, 1979.
- Bacopoulos, N.G.; Bhatanger, R.K.; and van Orden, L.S. The effects of subhypnotic doses of ethanol on regional catecholamine turnover. *Journal of Pharmacology and Experimental Therapeutics* 204:1-10, 1978.
- Ballenger, J.C.; Goodwin, F.K.; Major, L.F.; and Brown, G.L. Alcohol and central serotonin metabolism in man. *Archives of General Psychiatry* 36:224-227, 1979.
- Barbaccia, M.L.; Bosio, A.; Lucchi, L.; Spano, P.F.; and Trabucchi, M. Neuronal mechanisms regulating ethanol effects on the dopaminergic sys-

- tem. *Life Sciences* 30:2163-2170, 1982.
- Basile, A.S.; Dunwiddie, T.V.; and Hoffer, B.J. Ethanol effects on Purkinje cell spontaneous activity in the *in vitro* cerebellum of mice differentially sensitive to ethanol (abstract). *Federation Proceedings* 41:1465 (A.6983), 1982.
- Behar, D., and Winokur, G. Research in alcoholism and depression: A two-way street under construction. In: Pickens, R.W., and Heston, L.L., eds. *Psychiatric Factors in Drug Abuse*. New York: Grune & Stratton, 1979. pp. 125-152.
- Blass, J.P. Metabolic dementia. In: Amaducci et al., eds. *Aging of the Brain and Dementia*. Vol. 13. *Aging*. New York: Raven Press, 1980. pp. 261-270.
- Bloom, S.; Barchas, J.; Sandler, M.; and Usdin, E., eds. *Beta-Carbolines and Tetrahydroisoquinolines*. *Progress in Clinical and Biological Research*, Vol. 90. New York: Alan R. Liss, 1982.
- Brown, G.L.; Ebert, M.H.; Goyer, P.F.; Jimerson, D.C.; Klein, W.J.; Bunney, W.E.; and Goodwin, F.K. Aggression, suicide, and serotonin: Relationship to CSF amine metabolites. *American Journal of Psychiatry* 139:741-746, 1982.
- Buchsbaum, M.S., and Ludwig, A.M. Effects of sensory input and alcohol administration on visual evoked potentials in normal subjects and alcoholics. In: Begleiter, H., ed. *Biological Effects of Alcohol: Advances in Experimental Medicine and Biology*. Vol. 126. New York: Plenum Press, 1980. pp. 561-571.
- Butters, N. The Wernicke-Korsakoff syndrome. In: National Institute on Alcohol Abuse and Alcoholism. *Biomedical Processes and Consequences of Alcohol Use*. Alcohol and Health Monograph No. 2. DHHS Pub. No. (ADM) 82-1191. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 257-287.
- Carlen, P.L.; Gurevich, N.; and Durand, D. Ethanol in low doses augments calcium-mediated mechanisms measured intracellularly in hippocampal neurons. *Science* 215:306-309, 1982.
- Carlen, P.L.; Lee, M.A.; Jacob, M.; and Livshits, O. Parkinsonism provoked by alcoholism. *Annals of Neurology* 9:84-86, 1981a.
- Carlen, P.L.; Wilkinson, D.A.; Wortzman, G.; Holgate, R.; Cordingley, J.; Lee, M.A.; Huszar, L.; Moddel, G.; Singh, R.; Kiraly, L.; and Rankin, J.G. Cerebral atrophy and functional deficits in alcoholics without clinically apparent liver disease. *Neurology (New York)* 31:377-385, 1981b.
- Chaney, E.F.; O'Leary, M.R.; Fehrenbach, P.A.; and Donovan, D. Cognitive deficit in middle-aged alcoholics. *Drug and Alcohol Dependence* 6(4):219-226, 1980.
- Chin, J.H., and Goldstein, D.B. Effects of low concentrations of ethanol on the fluidity of spin-labeled erythrocyte and brain membranes. *Molecular Pharmacology* 13:435-441, 1977.
- Clayton, A.B. Effects of alcohol on driving skills. In: Sandler, M., ed. *Psychopharmacology of Alcohol*. New York: Raven Press, 1980. pp. 73-78.
- Collins, W.E., and Chiles, W.D. Laboratory performance during acute alcohol intoxication and hangover. *Human Factors* 22:445-462, 1980.
- Coppen, A. Lithium in the treatment of alcoholism. In: Sandler, M., ed. *Psychopharmacology of Alcohol*. New York: Raven Press, 1980. pp. 209-214.
- Cravioto, H.; Korein, J.; and Silberman, J. Wernicke's encephalopathy. A clinical and pathological study of 28 autopsied cases. *Archives of Neurology* 4:510-519, 1961.
- Crawford, D., and Baker, T.B. Alcohol dependence and taste-mediated learning in the rat. *Pharmacology, Biochemistry and Behavior* 16:253-261, 1982.
- Crowell, C.R.; Hinson, R.E.; and Siegel, S. The role of conditioned drug responses in tolerance to the hypothermic effects of ethanol. *Psychopharmacology* 73:51-54, 1981.
- Cutting, J. The relationship between Korsakov's syndrome and "alcoholic dementia." *British Journal of Psychiatry* 132:240-251, 1978.
- Davis, V.E., and Walsh, M.J. Alcohol, amines and alkaloids: A possible biochemical basis of alcohol addiction. *Science* 167:1005-1007, 1970.
- Deneau, G.; Yanagita, T.; and Seevers, M.H. Self-administration of psychoactive substances by the monkey: A measure of psychological dependence. *Psychopharmacologia* 16:30-48, 1969.
- Eckardt, M.J.; Parker, E.S.; Pautler, C.P.; Noble, E.P.; and Gottschalk, L.A. Neuropsychological consequences of posttreatment drinking behavior in male alcoholics. *Psychiatry Research* 2:135-147, 1980.
- Eckert, E.D.; Goldberg, S.C.; Halmi, K.A.; Casper, R.C.; and Davis, J.M. Alcoholism in anorexia nervosa. In: Pickens, R.W., and Heston, L.L., eds. *Psychiatric Factors in Drug Abuse*. New York: Grune & Stratton, 1979. pp. 267-283.
- Elmasian, R.; Neville, H.; Woods, D.; Schuckit, M.; and Bloom, F. Event-related brain potentials are different in individuals at high and low risk for developing alcoholism. *Proceedings of the National Academy of Sciences* 79:7900-7903, 1982.
- Freund, G., and Butters, N. Alcohol and aging: Challenges for the future. *Alcoholism: Clinical and Experimental Research* 6:1-2, 1982.
- Friedman, M.B.; Erickson, C.K.; and Leslie, S.W. Effects of acute and chronic ethanol administration on whole mouse brain synaptosomal calcium influx. *Biochemical Pharmacology* 29:1903-1908, 1980.
- Gallagher, E.J.; Parsons, L.M.; and Goldstein, D.B. The rapid onset of tolerance to ataxic effects of ethanol in mice. *Psychopharmacology* 78:67-70, 1982.
- Gallego, A. On the effect of ethyl alcohol upon frog nerve. *Journal of Cellular Comparative Physiology* 31:97-106, 1948.
- Goldstein, D.B.; Chin, J.H.; and Lyon, R.C. Ethanol disordering of spin-labeled mouse brain membranes: Correlation with genetically determined ethanol sensitivity of mice. *Proceedings of the National Academy of Sciences* 79:4231-4233, 1982.
- Goodwin, D.W., and Guze, S.B. *Psychiatric Diagnosis*. 2d ed. New York: Oxford University Press, 1979. pp. 118-144.

- Goodwin, D.W.; Schulsinger, F.; Hermansen, L.; Guse, S.B.; and Winokur, G. Alcoholism and the hyperactive child syndrome. Journal of Nervous and Mental Diseases 160:349-353, 1975.
- Gottfried, C.G. Activity of monoamine oxidase and brain levels of monoamines in alcoholics. In: Richeter, D., ed. Addiction and Brain Damage. Baltimore: University Park Press, 1980. pp. 228-239.
- Grupp, L.A. The influence of some neurotransmitter agonists and antagonists on the response of hippocampal units and the cortical EEG to ethanol in the awake rat. Psychopharmacology 75:327-334, 1981.
- Guilleminault, C.; van den Hoed, J.; and Mitler, M.M. Clinical overview of the sleep apnea syndromes. In: Guilleminault, C., and Dement, W.C., eds. Sleep Apnea Syndromes. Kroc Research Series, Vol. 11. New York: A.R. Liss, 1978. pp. 1-12.
- Hamm, J.E.; Major, L.F.; and Brown, G.L. The quantitative measurement of depression and anxiety in male alcoholics. American Journal of Psychiatry 136:580-582, 1979.
- Harper, C. Wernicke's encephalopathy: A more common disease than realised: A neuropathological study of 51 cases. Journal of Neurology, Neurosurgery and Psychiatry 42:226-231, 1979.
- Harper, C. Neuropathology of brain damage caused by alcohol. Medical Journal of Australia 2:277-282, 1982.
- Harris, R.A., and Hood, W.F. Inhibition of synaptosomal calcium uptake by ethanol. Journal of Pharmacology and Experimental Therapeutics 213:562-568, 1980.
- Hetzler, B.E.; Heilbronner, R.L.; Griffin, J.; and Griffin, G. Effects of ethanol on visual cortex and superior colliculus evoked potentials in rats (abstract). Drug and Alcohol Dependence 6:88-89, 1980.
- Hillborn, M., and Kaste, M. Alcohol intoxication: A risk factor for primary subarachnoid hemorrhage. Neurology (New York) 32:706-711, 1982.
- Hoffman, P.L.; Melchior, C.L.; and Tabakoff, B. Vasopressin maintenance of ethanol tolerance requires intact brain noradrenergic systems. Life Sciences, in press.
- Hoffman, P.L.; Ritzmann, R.F.; Walter, R.; and Tabakoff, B. Arginine vasopressin maintains ethanol tolerance. Nature 276:614-616, 1978.
- Hoffman, P.L.; Urwyler, S.; and Tabakoff, B. Alterations in opiate receptor function after chronic ethanol exposure. Journal of Pharmacology and Experimental Therapeutics 222:182-189, 1982.
- Hoyumpa, A.M., Jr. Mechanisms of thiamin deficiency in chronic alcoholism. American Journal of Clinical Nutrition 33:2750-2761, 1980.
- Hunt, G.P., and Overstreet, D.H. Evidence for parallel development of tolerance to the hyperactivating and disordinating effects of ethanol. Psychopharmacology 55:75-81, 1977.
- Hunt, W.A. Neurotransmitter function in the basal ganglia after acute and chronic ethanol treatment. Federation Proceedings 40:2077-2081, 1981.
- Hunt, W.A., and Majchrowicz, E. Alterations in the turnover of brain norepinephrine and dopamine in alcohol-dependent rats. Journal of Neurochemistry 23:549-552, 1974.
- Ingram, L.O. Effect of alcohol on membranes. In: National Institute on Alcohol Abuse and Alcoholism. Biomedical Processes and Consequences of Alcohol Use. Alcohol and Health Monograph No. 2. USDHHS Pub. No. (ADM)82-1191. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 3-27.
- Kearsley, J.H., and Musso, A.F. Hypothermia and coma in the Wernicke-Korsakoff syndrome. Medical Journal of Australia 2:504-506, 1980.
- Kilianmaa, K., and Tabakoff, B. Neurochemical correlates to tolerance and the biphasic behavioral effects of ethanol. Pharmacology, Biochemistry and Behavior 18 (Supplement 1): 383-388, 1983.
- Klemm, W.R., and Sherry, C.J. Messages in the brain: "Scrambling" by alcohol (abstract). Alcoholism: Clinical and Experimental Research 5:158, 1981.
- Le, A.D.; Kalant, H.; and Khanna, J.M. Interaction between des-Glycinamide⁹-[Arg⁸] vasopressin and serotonin on ethanol tolerance. European Journal of Pharmacology 80:337-345, 1982.
- Le, A.D.; Khanna, J.M.; Kalant, H.; and LeBlanc, A.E. The effect of lesions in the dorsal, median and magnus raphe nuclei on the development of tolerance to ethanol. Journal of Pharmacology and Experimental Therapeutics 218:525-529, 1981.
- Leigh, D.; McBurney, A.; and McIlwain, H. Erythrocyte transketolase activity in the Wernicke-Korsakoff syndrome. British Journal of Psychiatry 139:153-156, 1981.
- Levental, M., and Tabakoff, B. Sodium-potassium-activated adenosine triphosphatase activity as a measure of neuronal membrane characteristics in ethanol-tolerant animals. Journal of Pharmacology and Experimental Therapeutics 212:315-319, 1980.
- Lindman, R. Social and solitary drinking: Effects on consumption and mood in male social drinkers. Physiology and Behavior 28:1093-1095, 1982.
- Linnoila, M., and Martin, P.R. Benzodiazepines and alcoholism. In: Trimble, M.R., ed. Benzodiazepines Divided. New York: John Wiley & Sons, 1982. pp. 291-308.
- Lishman, W.A. Cerebral disorder in alcoholism: Syndromes of impairment. Brain 104:1-20, 1981.
- Lyon, R.C., and Goldstein, D.B. Changes in synaptic membrane order associated with chronic ethanol treatment in mice. Molecular Pharmacology 23:86-91, 1983.
- Lyon, R.C.; McComb, J.A.; Schreurs, J.; and Goldstein, D.B. A relationship between alcohol intoxication and the disordering of brain membranes by a series of short-chain alcohols. Journal of Pharmacology and Experimental Therapeutics 318:669-675, 1981.
- MacVane, J.; Butters, N.; Montgomery, K.; and Farber, J. Cognitive functioning in men social drinkers. A replication study. Journal of Studies

- on Alcohol 43:81-95, 1982.
- Mair, R.G.; McEntee, W.J.; and Zatorre, R.J. Brain, NE and DA activity; correlation with specific behavioral deficits in Korsakoff's psychosis. Neurology (New York) 32:A66-67, 1982.
- Martin, P.R. The human genetics of alcoholism. Substance and Alcohol Actions/Misuse 2:389-406, 1981.
- McLeod, J.G. Alcohol, nutrition, and the nervous system. Medical Journal of Australia 2:273-274, 1982.
- McNamee, J.E.; Tong, J.E.; and Piggins, D.J. Effects of alcohol on velocity perception: I. Stimulus velocity and change in performance over time. Perceptual and Motor Skills 51:779-785, 1980.
- Melchior, C.L. and Tabakoff, B. Modification of environmentally cued tolerance to ethanol in mice. Journal of Pharmacology and Experimental Therapeutics 219:175-180, 1981.
- Michaelis, E.K., and Myers, S.L. Calcium binding to brain synaptosomes: Effects of chronic ethanol intake. Biochemical Pharmacology 28:2081-2087, 1979.
- Moody, D.B.; Winger, G.; Woods, J.H.; and Stebbins, W.C. Effect of ethanol and of noise on reaction time in the monkey: Variation with stimulus level. Psychopharmacology 69:45-51, 1980.
- Morrison, J.R., and Stewart, M.A. A family study of the hyperactive child syndrome. Biological Psychiatry 3:189-195, 1971.
- Moskowitz, H., and Burns, M. Effects of alcohol and caffeine, alone and in combination, on skills performance. In: Goldberg, L., ed. Alcohol, Drugs and Traffic Safety. Vol. 3. Stockholm: Almqvist and Wiskell International, 1981. pp. 969-983.
- Munjack, D.J., and Moss, H.B. Affective disorder and alcoholism in families of agoraphobics. Archives of General Psychiatry 38:869-871, 1981.
- Myers, R.D., and Melchior, C.L. Alcohol drinking: Abnormal intake caused by tetrahydropapaveroline (THP) in the brain. Science 196:554-556, 1977.
- Myers, R.D.; McCaleb, M.L.; and Ruwe, W.D. Alcohol drinking induced in the monkey by tetrahydropapaveroline (THP) infused into the cerebral ventricle. Pharmacology, Biochemistry and Behavior 16:995-1000, 1982.
- National Highway Traffic Safety Administration. The Effects of Alcohol on the Driver's Visual Information Processing, by Ziedman, K.; Moskowitz, H.; and Niemann, R.A. DOT HS-805 748. Washington, D.C.: NHTSA, 1980. 131 pp. NTIS No. PB81-172751.
- National Highway Traffic Safety Administration. Effects of Fatigue and Alcohol on Highway Safety, by Ryder, J.M.; Malin, S.A.; and Kinsley, C.H. DOT HS-805 854. Washington, D.C.: NHTSA, 1981. 114 pp. NTIS No. PB81-204117.
- O'Leary, M.R.; Clasyn, D.A.; and Fauria, T. The group embedded figures test: A measure of cognitive style or cognitive impairment. Journal of Personality Assessment 44:532-537, 1980.
- Orrego, H.; Blendis, L.M.; Blake, J.E.; Kapur, B.M.; and Israel, Y. Reliability of assessment of alcohol intake based on personal interviews in a liver clinic. Lancet 2:1354-1356, 1979.
- Overall, J.E.; Brown, D.; Williams, J.D.; and Neill, L.T. Drug treatment of anxiety and depression in detoxified alcoholic patients. Archives of General Psychiatry 29:218-221, 1973.
- Parker, E.S. Comments on "Cognitive functioning in men social drinkers. A replication study." Journal of Studies on Alcohol 43:170-177, 1982.
- Parsons, O.A., and Fabian, M.S. Comments on "Cognitive functioning in men social drinkers. A replication study." Journal of Studies on Alcohol 43:178-182, 1982.
- Parsons, O.A., and Leber, W.R. Alcohol, cognitive dysfunction, and brain damage. In: National Institute on Alcohol Abuse and Alcoholism. Biomedical Processes and Consequences of Alcohol Use. Alcohol and Health Monograph No. 2. DHHS Pub. No. (ADM)82-1191. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 213-253.
- Pfefferbaum, A.; Horvath, T.B.; Roth, W.T.; Clifford, S.T.; and Kopell, B.S. Acute and chronic effects of ethanol on event-related potentials. In: Begleiter, H., ed. Biological Effects of Alcohol. Advances in Experimental Medicine and Biology. Vol. 126. New York: Plenum Press, 1980. pp. 625-639.
- Pittman, Q.J.; Rogers, J.; and Bloom, F.E. Arginine vasopressin deficient Brattleboro rats fail to develop tolerance to the hypothermic effects of ethanol. Regulatory Peptides 4:33-41, 1982.
- Pohorecky, L.A. Effects of ethanol on central and peripheral noradrenergic neurons. Journal of Pharmacology and Experimental Therapeutics 189:380-391, 1974.
- Porjesz, B., and Begleiter, H. Evoked brain potential deficits in alcoholism and aging. Alcoholism: Clinical and Experimental Research 6(1):53-63, 1982.
- Rabin, R.A.; Wolfe, B.B.; Dibner, M.D.; Zahniser, N.R.; Melchior, C.L.; and Molinoff, P.B. Effects of ethanol administration and withdrawal on neurotransmitter receptor systems in C57 mice. Journal of Pharmacology and Experimental Therapeutics 213:419-496, 1980.
- Rangaraj, N., and Kalant, H. Interaction of ethanol and catecholamines on rat brain ($\text{Na}^+ + \text{K}^+$)-ATPase. Canadian Journal of Physiology and Pharmacology 57:1098-1166, 1979.
- Rangaraj, N., and Kalant, H. Effect of chronic ethanol treatment on temperature dependence and on norepinephrine sensitization of rat brain ($\text{Na}^+ + \text{K}^+$)-adenosine triphosphatase. Journal of Pharmacology and Experimental Therapeutics 223:536-539, 1982.
- Rhenstrom, S.; Simert, G.; Hansson, J.A.; Johnson, G.; and Vang, J. Chronic hepatic encephalopathy. A psychometrical study. Scandinavian Journal of Gastroenterology 12:305-311, 1977.
- Ron, M.A.; Acker, W.; Shaw, G.K.; and Lishman, W.A. Computerized tomography of the brain in

- chronic alcoholism. A survey and follow-up study. *Brain* 105:497-514, 1982.
- Rosenthal, N.E.; Davenport, Y.; Cowdry, R.W.; Webster, M.H.; and Goodwin, F.K. Monoamine metabolites in cerebrospinal fluid of depressive subgroups. *Psychiatry Research* 2:113-119, 1980.
- Rubin, L.S. Pupillometric studies of alcoholism. *International Journal of Neurosciences* 11:301-308, 1980.
- Ryan, C. Learning and memory deficits in alcoholics. *Journal of Studies on Alcohol* 41:437-447, 1980.
- Ryan, C. Alcoholism and premature aging: A neuropsychological perspective. *Alcoholism: Clinical and Experimental Research* 6:22-30, 1982.
- Schenker, S.; Henderson, G.I.; Hoyumpa, A.M., Jr.; and McCandless, D.W. Hepatic and Wernicke's encephalopathies: Current concepts of pathogenesis. *American Journal of Clinical Nutrition* 33:2719-2726, 1980.
- Schuckit, M.A. Prevalence of affective disorder in a sample of young men. *American Journal of Psychiatry* 139:1431-1436, 1982.
- Shaw, S.; Gorkin, B.D.; and Lieber, C.S. Effects of chronic alcohol feeding on thiamin status: Biochemical and neurological correlates. *American Journal of Clinical Nutrition* 34:856-860, 1981.
- Shefner, S.A.; Chiu, T.H.; and Anderson, E.G. Intracellular measurement of ethanol effects on rat locus caeruleus neurons in a brain slice preparation. *Society for Neuroscience Abstracts* 8:651(A.185.3), 1982.
- Siggins, G.R., and Bloom, F.E. Alcohol-related electrophysiology. *Pharmacology, Biochemistry and Behavior* 13(Supplement 1):203-211, 1980.
- Siggins, G.R.; Berger, T.; French, E.D.; Shier, T.; and Bloom, F.E. Ethanol, salsolinol and tetrahydropapaveroline alter the discharge of neurons in several brain regions: Comparison to opioid effects. In: *Beta-Carbolines and Tetrahydroisoquinolines: Proceedings of a Workshop Held at the Salk Institute (1981). Progress in Clinical and Biological Research*. Vol. 90. New York: Liss, 1982. pp. 275-285.
- Sinclair, J.D.; Rusi, M.; Airaksinen, M.; and Altshuler, H.L. Relating TIQ's, opiates and ethanol. In: Bloom, F.; Barchas, J.; Sandler, M.; and Usdin, E., eds. *Beta-Carbolines and Tetrahydroisoquinolines. Progress in Clinical and Biological Research*. Vol. 90. New York: Liss, 1982. pp. 365-376.
- Smith, H.H., and Smith, L.S. WAIS functioning of cirrhotic and non-cirrhotic alcoholics. *Journal of Clinical Psychology* 33:309-313, 1977.
- Sorenson, S.; Dunwiddie, T.; McClearn, G.; Freedman, R.; and Hoffer, B. Ethanol-induced depression in cerebellar and hippocampal neurons in mice selectively bred for differences in ethanol sensitivity: An electrophysiological study. *Pharmacology, Biochemistry and Behavior* 14:227-234, 1981.
- Sorenson, S.; Palmer, M.; Dunwiddie, T.; and Hoffer, B. Electrophysiological correlates of ethanol-induced sedation in differentially sensitive lines of mice. *Science* 210:1143-1145, 1980.
- Stokes, J.A., and Harris, R.A. Alcohols and synaptic calcium transport. *Molecular Pharmacology* 22:99-104, 1982.
- Tabakoff, B.; Luthin, G.L.; Saito, T.; and Hoffman, P.L. Effects of ethanol on receptor adenylate cyclase coupling (abstract). *Federation Proceedings* 42(4):902, 1983.
- Tabakoff, B.; Munoz-Marcus, M.; and Fields, J.Z. Chronic ethanol feeding produces an increase in muscarinic cholinergic receptors in mouse brain. *Life Sciences* 25:2173-2180, 1979.
- Tabakoff, B., and Hoffman, P.L. Development of functional dependence on ethanol in dopaminergic systems. *Journal of Pharmacology and Experimental Therapeutics* 208:216-222, 1979.
- Tabakoff, B., and Ritzmann, R.F. The effects of 6-hydroxydopamine on tolerance to and dependence on ethanol. *Journal of Pharmacology and Experimental Therapeutics* 203:319-331, 1977.
- Taberner, P.V. Sex differences in the effects of low doses of ethanol on human reaction time. *Psychopharmacology* 70:283-286, 1980.
- Tarter, R.E.; McBride, H.; Buonpane, N.; and Schneider, D.U. Differentiation of alcoholics: Childhood history of minimal brain dysfunction, family history, and drinking pattern. *Archives of General Psychiatry* 34:761-768, 1977.
- Ticku, M.K., and Burch, T. Alterations in gamma-aminobutyric acid receptor sensitivity following acute and chronic ethanol treatments. *Journal of Neurochemistry* 34:417-423, 1980.
- Tong, J.E.; Henderson, P.R.; and Chipperfield, B.G.A. Effects of ethanol and tobacco on auditory vigilance performance. *Addictive Behavior (Oxford)* 5:153-158, 1980.
- Tsuang, M.T. Genetic factors in suicide. *Diseases of the Nervous System* 38:498-501, 1977.
- Tuomisto, L.; Airaksinen, M.M.; Peura, P.; and Erikson, C.J.P. Alcohol drinking in the rat: Increases following intracerebroventricular treatment with tetrahydro-Beta-carbolines. *Pharmacology, Biochemistry and Behavior* 17:831-836, 1982.
- U.S. Department of Health and Human Services. *Fourth Special Report to the U.S. Congress on Alcohol and Health*. DeLuca, J.R., ed. DHHS Pub. No. (ADM)81-1080. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- U.S. Department of Health, Education, and Welfare. *Second Special Report to the U.S. Congress on Alcohol and Health*. Chafetz, M.E., ed. DHEW Pub. No. (ADM)75-212. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1974.
- U.S. Department of Health, Education, and Welfare. *Third Special Report to the U.S. Congress on Alcohol and Health*. Noble, E.P., ed. DHEW Pub. No. (ADM)79-832. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1978.
- Victor, M.; Adams, R.D.; and Collins, G.H. *The Wernicke-Korsakoff Syndrome*. Philadelphia: F.A. Davis, 1971.
- Walker, D.W.; Hunter, B.E.; and Abraham, W.C. Neuroanatomical and functional deficits subsequent to chronic ethanol administration in animals. *Alcoholism: Clinical and Experimental*

- Research 5:267-282, 1981.
- Weingartner, H.; Cohen, R.M.; Murphy, D.L.; Martello, J.; and Gerdt, C. Cognitive processes in depression. Archives of General Psychiatry 38:42-47, 1981.
- Weingartner, H.; Grafman, J.; Boutelle, W.; Kaye, W.; and Martin, P.R. Forms of memory failure. Science 221:372-373, 1980.
- Weissman, M.M., and Myers, J.K. Clinical depression in alcoholism. American Journal of Psychiatry 137:372-373, 1980.
- Wells, C.E. Diagnosis of dementia. Psychosomatics 20:517-522, 1979.
- West, J.R.; Lind, M.D.; Demuth, R.M.; Parker, E.S.; Alkana, R.L.; Cassell, M.; and Black, A.C. Lesion-induced sprouting in the rat dentate gyrus is inhibited by repeated ethanol administration. Science 218:808-810, 1982.
- Wilkinson, D.A. Examination of alcoholics by computed tomographic (CT) scans: A critical review. Alcoholism: Clinical and Experimental Research 6:31-45, 1982.
- Wilkinson, D.A., and Carlen, P.L. Chronic organic brain syndromes associated with alcoholism: Neuropsychological and other aspects. In: Israel, Y.; Glaser, F.B.; Kalant, H.; Popham, R.E.; Schmidt, W.; and Smart, R.G., eds. Research Advances in Alcohol and Drug Problems. Vol. 6. New York: Plenum Press, 1981. pp. 107-145.
- Wise, R.A. Actions of drugs of abuse on brain reward systems. Pharmacology, Biochemistry and Behavior 13(Suppl 1):213-223, 1980.
- Wood, D.; Wender, P.H.; and Reimherr, F.W. The prevalence of attention deficit disorder, residual type, or minimal brain dysfunction, in a population of male alcoholic patients. American Journal of Psychiatry 140:95-98, 1983.
- Wood, W.G., and Armbricht, H.J. Behavioral effects of ethanol in animals: Age differences and age changes. Alcoholism: Clinical and Experimental Research 6:3-12, 1982.
- Young, J.A., and Pihl, R.O. Self-control of the effects of alcohol intoxication. Journal of Studies on Alcohol 41:567-571, 1980.
- Zornetzer, S.F.; Walker, D.W.; Hunter, B.E.; and Abraham, W.C. Neurophysiological changes produced by alcohol. In: National Institute on Alcohol Abuse and Alcoholism. Biomedical Processes and Consequences of Alcohol Use. Alcohol and Health Monograph No. 2. DHHS Pub. No. (ADM)82-1191. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 95-128.

Chapter IV

Medical Consequences of Alcohol

For most of us, alcohol conjures up pleasant social occasions--wine with a good meal, beer at a ball game, or cocktails during a party of friends. Rarely do we consider the harmful effects of alcohol. Even many experienced drinkers who know every vineyard, vintage year, distillery, and brewery are relatively unaware of the basic medical and biological consequences of drinking.

This chapter follows the trail of adverse effects of alcohol throughout the body and its impact on various organ systems.

Alcohol and the Digestive System

The Mouth

The application of dilute alcohol and a number of alcoholic beverages to the tongue and mucous membranes of the cheek stimulates the flow of saliva (Martin and Pangborn 1971). However, after a short time, the secretion rate of saliva is inhibited for 60 to 90 minutes before returning to normal (Winsor and Strongin 1933). Enlargement of the parotid (a specific salivary gland) is common in alcoholics (approximately 12 percent), but it is uncertain whether it occurs as a result of alcohol use or is due to the protein deficiency frequently seen in alcoholics (Bode 1981).

Inflammation of lips and soft tissue of the mouth, common in alcohol abusers, is thought to occur as a result of nutritional deficiency, particularly of the various B vitamins and iron (Larato 1972).

Cancers of the mouth, tongue, pharynx, and esophagus occur more often in alcoholics than in nonalcoholics. Wynder (1975) has suggested that a synergistic effect between alcohol and tobacco may be responsible. Other factors that may contribute to such carcinogenesis include deficiencies of vitamin A, vitamin B, and zinc that may occur in the alcoholic (Wynder and Chan 1970). Sporn et al. (1976) found that vitamin A deficient animals are

more susceptible to cancers of the skin, mouth, and esophagus than are nondeficient animals.

The Esophagus

The occurrence of nonmalignant esophageal disease such as esophagitis has been associated with chronic alcohol abuse (Shirazi and Platz 1978). In normal human volunteers, alcohol has been shown to interfere with acid clearing mechanisms and to irritate the mucous membranes (Mayer et al. 1978). Mucosal tissue tends to be abnormal in alcoholics (Zeus 1970). Moreover, there is an increased risk of Barrett's esophagitis (a premalignant lesion) in alcoholics when compared with nonalcoholics (Martini and Wienbeck 1974). Esophageal varicose veins, which can develop in cirrhotic patients, can be eroded, resulting in bleeding into the gastrointestinal tract (Chaput et al. 1974).

The Stomach

The effect of alcohol on gastric emptying largely depends on the concentration of alcohol in the stomach (Cooke 1972). Gastric emptying is slowed when alcohol concentrations are greater than 6 percent (Pirola and Davis 1970).

While it has no effect on the muscle that empties the stomach, alcohol has been shown to produce a diminution in the sphincter muscle response to acidity in the duodenum (Phosawasdi et al. 1979).

Chronic consumption of alcohol causes a diminution of gastric acid secretion in humans (Cooke 1972). Furthermore, alcohol increases the formation of prostaglandins, which are known to inhibit acid secretion (Main and Whittle 1976). Such an increase may account for the reduced stomach acid observed in individuals who have abused alcohol for 5 or

more years. Acute administration of alcohol into the mouth, stomach, or blood, however, stimulates secretion of gastric juice. The secretion appears to be maximal at alcohol concentrations between 8 and 16 percent, approximately the concentration found in wine (Elwin 1969).

Acid often injures the mucous membranes of the stomach (Gottfried et al. 1976). Thus, drinking can produce gastric hemorrhage. While alcohol alone does not cause gastric bleeding in normal subjects, it significantly increases blood loss in persons with aspirin-derived mucous membranes injury (DeSchepper et al. 1978). Therefore, the aspirin commonly taken by alcohol abusers to alleviate discomfort may aggravate further stomach injury (Robert et al. 1980).

Alcohol abuse as a cause of gastric ulcer disease, however, remains controversial. Although the incidence of peptic ulcer has been reported to be as high as 20.6 percent in patients with alcoholic liver disease, an association between the two disease conditions has never been proven (Bode 1981).

There is conflicting evidence concerning the role of alcohol in stomach cancer, with most studies showing no clear association. However, a limited number of studies have shown a positive correlation between alcohol use and the development of gastric cancer (Bode 1981). Whether alcohol-induced gastritis is a premalignant lesion, particularly if it occurs repeatedly, is an important question for which sufficient evidence is not yet available (Bode 1981).

The Intestine

The intestine is an important site for the absorption of nutrients into the blood stream. The major focus of research in this area has been on the small intestine. This site is exposed to higher concentrations of alcohol than any other organ in the body except the stomach. After either acute or chronic alcohol consumption, the small intestine may demonstrate structural injury.

In humans, a single small dose (1 gm/kg body weight) of alcohol can produce lesions in the duodenum, a part of the small intestine (Gottfried et al. 1976). Additional studies with human volunteers found that the structural changes that occurred upon consumption of alcohol disappeared once alcohol consumption ceased (Rubin et al. 1972). Similarly, in rats,

intestinal lesions occur within 10 minutes of alcohol ingestion, are well developed within an hour, and disappear after 4 to 16 hours (Baraona et al. 1974). This same study found that animals that chronically consumed alcohol did not respond with such lesions.

Decreases in intestinal enzymes, including those that act on milk, are common in experimental animals and man after alcohol ingestion. Thus, milk intolerance may be exaggerated in alcohol abusers (Perlow et al. 1975).

When applied directly to small intestinal membranes, alcohol decreases the sodium-potassium-ATPase activity from 94 percent to 17 percent of control values (Hoyumpa et al. 1977). As a result of this effect, alcohol in excess of 2 percent can inhibit the transport of electrolytes, amino acids, and glucose across the small intestine (Kuo and Shanbour 1978). Thus, both in animals and in humans, alcohol inhibits the transport of essential amino acids (Israel et al. 1968). Although tissue damage resulting from large amounts of alcohol can inhibit amino acid transport, the fact that it is rapidly reversible suggests that a physiochemical change rather than a toxic injury is responsible (Israel et al. 1968).

Vitamin absorption is adversely affected by alcohol. Alcoholics have shown a significant reduction in the absorption of folic acid (a vitamin) into the jejunum, another part of the small bowel (Halsted et al. 1973). However, after 2 weeks of nutritional supplementation, the folic acid absorption of alcoholics improves despite continued use of alcohol. This suggests that, in alcoholics, impaired folic acid absorption may be primarily a result of poor nutrition rather than a direct toxic effect of alcohol. In support of this contention, studies of acute alcohol administration to normal subjects have shown a decreased folic acid absorption in only 20 percent of the subjects studied (Halsted et al. 1967). However, folic acid deficiency is the most common nutritional deficiency present in alcoholics (Leevy et al. 1965). There also is evidence to suggest that liver tissue is less able to take up folic acid from blood after acute alcohol administration to either normal or alcoholic individuals (Lane et al. 1976).

Thiamine (vitamin B₁) is absorbed much like folic acid. The active absorption of thiamine by cells lining the small intestine is not affected by alcohol (Hoyumpa et al. 1974). However, thiamine movement out of the intestinal cell is impaired by alcohol (Hoyumpa et al. 1975). Both human and animal studies suggest that alcohol inhibits the mechanism

for absorption of thiamine at normal levels of the vitamin (Thomson et al. 1970), but has no effect on the diffusion mechanism that comes into play at high thiamine concentrations (Hoyumpa et al. 1974).

Alcohol's effect on iron absorption is unclear. In one human study (Charlton et al. 1964), a single dose of alcohol increased absorption of a dietary form of iron, ferrous chloride, while others have found no effect of either acute or chronic alcohol administration on iron absorption (Murray and Stein 1965). Adding further confusion to this issue are the studies that showed that ferrous iron absorption is inhibited rather than enhanced by alcohol (Tapper et al. 1968).

Although high levels of zinc are commonly found in the urine of alcoholics, it has not been established that alcohol consumption produces zinc deficiency by increased urinary zinc losses (Sullivan 1962). While alcohol is metabolized by a zinc-containing enzyme, zinc deficiency is not known to decrease alcohol metabolism (Wang and Pierson 1975).

The mechanism involved in calcium transport by the small intestine has not been elucidated. An animal study showed that alcohol inhibits calcium absorption (Krawitt 1975), but the mechanism of such inhibition is not entirely clear. In contrast to animal studies, one study of nonalcoholics given short-term alcohol showed no adverse effect on calcium absorption (Verdy and Caron 1973). Others, however, have reported a doubling of fecal calcium in human volunteers after drinking, suggesting a reduced calcium absorption (Krawitt 1975).

Alcohol has multiple effects on lipid metabolism within the small intestine. In general, alcohol increases triglyceride synthesis in intestines and in mucous membranes (Baraona et al. 1975). It also causes a decrease in fatty acid oxidation.

After exposure to alcohol, the intestinal lymph glands produce more cholesterol and triglyceride, an effect seen only when alcohol is present within the intestine (Middleton et al. 1971). In addition, cholesterol is redistributed among the various intestinal lymph lipoproteins. If alcohol is injected rather than drunk, the redistribution does not occur. This suggests that alcohol acts on the small intestine mucosal enzymes in altering the synthesis and transport of cholesterol. Interestingly, chronic alcohol feeding to animals abolishes or inhibits these acute effects of alcohol on intestinal lipid transport (Baraona et al. 1975; Rodgers and O'Brien 1975). Thus,

the relationship of these findings to the clinical condition of alcohol-induced hyperlipidemia (excess lipids in the blood) remains unclear (Baraona et al. 1975).

The effect of alcohol on the motility of different small intestine segments is variable, but the net effect is to shorten the transit time through the intestine. Initially, when intravenous alcohol is given to volunteers, motility increases, particularly in the second portion of the duodenum (Pirola and Davis 1970). This increased motility is characterized by an increase in intestinal pressure and in the number of contractile waves generated. Later, there is a decrease in duodenal activity with a gradual return toward normal. Moreover, alcohol depresses nonpropulsive motility of the colon in both dogs and humans. It should be noted that these changes in small and large intestinal motor activity may contribute, at least in part, to the diarrhea commonly seen following excessive drinking (Martin et al. 1980).

The Pancreas

Excessive alcohol use can cause chronic pancreatitis, in which lesions and functional impairment persist even in the absence of alcohol (Sarles 1974). This contrasts with gallstone-induced acute pancreatitis in which the pancreas returns to normal after each episode.

More than 75 percent of the cases of chronic pancreatitis in the United States occur in alcoholics. Moreover, pathologic changes or alterations in pancreatic function can be detected in half the alcoholics without pancreatitis symptoms (Reber 1978). Steady alcohol consumption over about 6 to 12 years is necessary before pancreatic symptoms occur.

Acute pancreatitis usually does not occur in the nonalcoholic who occasionally drinks excessively (Strum and Spiro 1971). On the other hand, alcoholics develop symptoms of acute pancreatitis, but only after functional and histologic (tissue) changes of chronic pancreatitis are well established. Progressive destruction of the pancreas continues even after the individual stops drinking.

Surprisingly, chronic alcohol administration to dogs stimulates pancreatic secretion, whereas alcohol's acute effect is inhibitory (Brooks and Thomas 1953). This excitatory effect can be abolished by atropine (a drug that blocks some nervous system activities) and does not occur in previously vagotomized (cutting the nerves connecting the brain to the stomach) dogs. Again, the neurochemical

mechanism acting on alcoholic animals is opposite to that seen in nonalcoholic animals. The reason why chronic alcoholism reverses this acute effect of alcohol on the pancreas is not known.

Regardless of the specific mechanism involved, chronic alcohol consumption results in the secretion of a juice more concentrated in protein. Protein precipitates are rich in calcium carbonate and are identical to the small pancreatic stones found in humans with chronic pancreatitis (Nakamura et al. 1972). In both dogs and humans, these "plugs" of precipitated protein obstruct the ducts, blocking secretion. Acetaldehyde is a highly labile and reactive substance that readily forms adducts with proteins. If sufficient amounts of acetaldehyde are present in the pancreas, it may contribute to the formation of pancreatic stones which obstruct the ducts.

The fine structural changes that occur in the pancreas after alcohol ingestion are similar to those found in liver cells (Darlie et al. 1970), and the changes in early human chronic pancreatitis appear to be similar to those observed in animals chronically treated with the hormones gastrin or cholecystokinin.

A relatively recent finding is that a specific genetically controlled cell surface antigen (a substance that activates the immune system) has an increased prevalence in individuals with chronic pancreatitis due to alcoholism. This suggests a possible genetic mechanism predisposing alcoholics to pancreatitis (Fauchet et al. 1979).

An association between alcoholism and cancer of the pancreas has been hypothesized. Burch and Ansari (1968) were the first to make such a suggestion, based on a retrospective study in which 75 percent of the patients with pancreatic cancer also had a history of moderate to heavy alcohol intake. Additional support for this thesis was provided by the report that the risk of pancreatic cancer was twice as great in males who consumed alcohol daily as it was in nondrinkers (Lieber 1977).

Alcohol and the Liver

Until recently, it was unclear whether liver disease associated with alcohol consumption was a direct effect of the alcohol or of the malnutrition that often accompanies alcoholism. The landmark work, primarily of Lieber (1981) and his colleagues, has now established that alcohol is directly toxic to the liver.

Metabolism of Alcohol

Alcohol is not effectively stored in tissues, and little less than 10 percent of an ingested dose can be eliminated through the kidneys, lungs, and skin. For practical purposes, one can assume that the body rids itself of alcohol only by oxidizing it in the liver, the organ containing the bulk of the enzymes required for alcohol oxidation. The liver's complexity and unique role in alcohol oxidation contribute to the many deleterious effects of alcohol within the liver. Unlike other major sources of calories, alcohol cannot be stored either in the liver or in other tissues; it must therefore be metabolized immediately in preference to all other fuels.

Three liver enzyme systems have been shown to promote the oxidation of alcohol to acetaldehyde. They are, in descending order of importance: alcohol dehydrogenase (ADH), microsomal ethanol-oxidizing system (MEOS), and catalase. The acetaldehyde produced is itself toxic, so a second step is subsequently required to convert it to a final nontoxic substance, acetate.

The major pathway for alcohol metabolism involves ADH. Although an increase in ADH activity above that seen normally may not necessarily increase the rate of alcohol oxidation, reductions in ADH activity do diminish alcohol metabolism (Bosron and Li 1980). Thus, low protein diets, which reduce ADH levels, slow the metabolism of alcohol both in rats and in humans (Pekkanen et al. 1978). Consistent with this observation, prolonged fasting reduces the metabolism of alcohol (Bode and Thiele 1975). Moreover, the sex steroid testosterone inhibits ADH activity while estradiol, an estrogen, enhances it (Rachamin et al. 1980).

As noted above, other systems in addition to ADH can oxidize alcohol. Thus, in animal experiments, as much as 30 percent of the total chronically administered alcohol may be oxidized by a non-ADH enzyme pathway in which MEOS plays the major role (Matsuzaki et al. 1981). However, the contribution of this system to alcohol elimination in humans has not been quantitated. The issue in humans is complicated by the presence of many forms of ADH, including one form, the π ADH, which has an activity profile similar to MEOS (Bosron et al. 1979). Nonetheless, following chronic alcohol consumption, the MEOS system is significantly increased in activity (Lieber and DeCarli 1968).

In humans, alcohol consumption accelerates blood alcohol clearance, particularly at high alcohol concentrations. This observation is consistent with the metabolic activity of MEOS (Salaspuro and Lieber 1978) but is also consistent with the activity of α -ADH. MEOS activity generates only heat, while ADH oxidation can produce storable forms of energy. This may account for the observation that alcohol produces a greater increase in oxygen consumption in alcoholics than in normal individuals, and also for the lesser growth in animals that are fed alcohol as compared to their controls (Pirola and Lieber 1972). Such energy wasting could explain, at least in part, the weight loss experienced when alcohol calories are substituted for a balanced diet (Lieber et al. 1965).

Alcohol oxidation always produces acetaldehyde which, in turn, is metabolized to acetate. It is generally accepted that more than 90 percent of the acetaldehyde formed as a consequence of alcohol oxidation is oxidized by aldehyde dehydrogenase (ALDH) to acetate immediately within the liver.

The principal cellular site of acetaldehyde metabolism lies within the mitochondria, which are found in the cytoplasm of cells. Moreover, the major portion of acetaldehyde oxidation within the mitochondria occurs where ALDH is located.

Acetaldehyde, like its precursor alcohol, adversely affects many tissues. It appears to interfere with vitamin B₆ metabolism and promotes the vitamin's degradation (Veitch et al. 1975). Acetaldehyde has been shown to impair the synthesis of proteins in the heart and can inhibit protein secretion by liver cells (Schreiber et al. 1972, 1974; Sorrell et al. 1983). Finally, acetaldehyde can depress the capacity of the liver to oxidize fatty acids (Cederbaum et al. 1975). The alcoholic may therefore be a victim of a vicious cycle; high acetaldehyde levels and chronic alcohol consumption impair the liver's mitochondrial function, which decreases acetaldehyde metabolism, which accelerates the accumulation of acetaldehyde, all leading to liver damage.

Amino Acid and Protein Metabolism

Alcohol disrupts amino acid (the building blocks of proteins) metabolism in the liver, which leads to their excessive accumulation (Krebs et al. 1973). Amino acid abnormalities in the blood appear in patients with alcoholic hepatitis, cirrhosis, and liver insufficiency (Rosen et al. 1977). In an isolated rat liver,

relatively high concentrations of alcohol inhibit the uptake of amino acids (Krebs et al. 1973).

Patients with cirrhosis frequently have an increased plasma level of aromatic amino acids (Ono et al. 1978). These amino acids convert to amines within the gastrointestinal tract and can adversely affect the central nervous system by acting as false neurotransmitters. In this regard, Faraj and co-workers (1976, 1979) found a statistically significant correlation between the level of plasma tyramine (an amino acid product) and the degree of hepatic encephalopathy (liver-caused brain damage).

The level of alpha amino N-butyric acid (AANB) in the blood is increased after chronic alcohol consumption, which has led to suggestions that it may be of use as a biochemical marker or indicator of alcoholism (Shaw et al. 1976). However, the plasma level of AANB may reflect at least two different factors present in chronic alcoholic individuals: Chronic alcohol consumption tends to increase the level of AANB, while dietary protein deficiency tends to decrease it. It is therefore necessary to control for nutritional factors if one is to use the level of AANB in plasma as a biochemical marker of chronic alcohol consumption (Shaw and Lieber 1978).

Alcoholics with cirrhosis and malnutrition have depressed hepatic levels of carnitine, an amino acid needed for oxidation of fatty acids (Rudman et al. 1977). Possibly this is due to an impaired ability of such patients to synthesize it. The blood level of free proline (an amino acid) is higher in some patients with cirrhosis compared with controls (Rojkind and DeLeon 1970) and appears to be proportional to the collagen (the structural protein that accumulates in cirrhosis) content of the livers of such individuals (Kershenobich et al. 1970).

Increased levels of acetaldehyde have been noted in patients with alcohol-induced liver disease, and acetaldehyde, but not alcohol, has been shown to interfere with protein synthesis (Schreiber et al. 1972, 1974; Sorrell et al. 1983). Consistent with this finding is the observation that alcohol does not inhibit protein synthesis by those liver cells that do not oxidize alcohol to form acetaldehyde (Morland et al. 1979). A similar lack of an inhibitory effect occurs in a variety of nonhepatic tissues in which the ADH activity is absent or less than that present in the liver (Perin and Sessa 1975). Thus, the inhibitory effects of alcohol on protein synthesis are dependent, at least in part, on alcohol metabolism to acetaldehyde.

Fatty Acid Metabolism

Fatty infiltration of the liver after alcohol consumption is the most common hepatic abnormality associated with alcohol abuse. The infiltration is due to an increased accumulation of triglycerides in response to alcohol. The origin of the triglycerides depends upon the pattern of alcohol consumption (dose and duration) and the lipid content of the diet. With acute ingestion of a large dose of alcohol, the excess hepatic triglycerides are derived from peripheral fat stores, while after chronic alcohol ingestion, the excess of triglycerides is the result of fat synthesis by the liver itself.

The replacement of dietary triglycerides containing long chain fatty acids with fat consisting of medium chain triglycerides reduces the propensity of alcohol to induce a fatty liver (Lieber and DeCarli 1966). Apparently the greater propensity of medium chain triglycerides to undergo oxidation accounts for this phenomenon (Lieber 1967). Protein deficiency may also contribute to an alcohol-induced fatty liver in some circumstances; however, even with adequate protein in the diet, alcohol may cause the development of fatty liver. It would appear that the main factors leading to the development of alcoholic fatty liver are as follows: Alcohol replaces fatty acids as the preferred fuel for mitochondria. The metabolic consequence is that more lipid (fatty acid) is synthesized and less is burned. In addition, alcohol at high concentrations inhibits the transport of newly synthesized lipids out of the liver cell. The net effect is accumulation of fatty acids.

A consequence of the hepatic accumulation of fatty acid is the excess formation of ketones (Lefevre et al. 1970). This results in alcoholic ketoacidosis (increased levels of ketones and acids in the blood), which is usually associated with a mild hyperglycemia (elevated blood sugar). Susceptibility to alcoholic ketoacidosis varies among individuals. The basis for alcoholic ketoacidosis is still unclear; however, its development may not be related to the amount of alcohol ingested nor to the levels of alcohol in the blood. It is possible that disrupted hormone levels are involved. Blood insulin levels are low during the ketoacidosis episode, while those of growth hormone, epinephrine, glucagon, and cortisol are high. Such a hormone pattern could act to mobilize free fatty acids from peripheral adipose (fat) tissue to enhance fatty acid metabolism

by the liver and contribute to the mild hyperglycemia.

Other Metabolic Effects

Chronic alcohol ingestion leads to proliferation of liver microsomes, fine intracellular structures that are responsible for detoxifying many drugs. Therefore, it is not surprising that an enhanced metabolism of drugs is common in alcoholics. For example, the metabolism of meprobamate, pentobarbital, antipyrine, tolbutamide, warfarin, and diphenylhydantoin is enhanced after heavy alcohol intake and can persist for up to 3 weeks after alcohol intake has been discontinued (Mezey 1979).

Chronic drinking is associated with a raised metabolic rate, which is characterized by increased oxygen consumption, inefficient use of calories, increased heat output, and weight loss (Pirola and Lieber 1972). This hypermetabolic state has been hypothesized to result, at least in part, from increased enzyme activity within the liver (Israel et al. 1973). The increased rate of oxygen consumption can be markedly depressed by surgically removing the thyroid and can be abolished by treatment with propylthiouracil (PTU), a thyroid inhibitor (Orrego et al. 1979). These observations suggest that PTU may be of potential therapeutic value in alcoholic patients with liver disease. Indeed, in a recent study, PTU was reported to exert a beneficial short-term effect on both the magnitude and the rate of improvement in patients with alcoholic hepatitis (Orrego et al. 1979). Moreover, the therapeutic value of PTU was found to be greatest in those patients with the most severe liver disease. There are, however, some conflicting reports. For example, Halle et al. (1982) performed a double-blind controlled trial in patients with severe acute alcoholic hepatitis. No beneficial effects of PTU therapy were observed. The question of the therapeutic usefulness of PTU therefore remains to be answered.

Pathology

There are three specific subtypes of hepatic pathology associated with alcohol use: alcoholic fatty liver, alcoholic hepatitis, and alcoholic cirrhosis. In its early stages, alcoholic liver disease is characterized by accumulation of excess fat in the liver as a result of the mechanisms already described above. Alcoholic hepatitis is characterized by the death and inflammation of individual liver cells. This

more severe form of alcohol-associated hepatic injury may cause death in 10 to 30 percent of the afflicted individuals. Eventually the entire structural and functional capacity of the liver is impaired, resulting in cirrhosis, the most severe and presumably irreversible form of alcohol-induced liver disease.

Most patients with alcoholic fatty liver are virtually asymptomatic; the various laboratory tests appear nearly normal. Importantly, alcoholic fatty liver is a completely reversible lesion that does not necessarily progress to a more severe disease.

The study of patients with alcoholic hepatitis may lead to an understanding of the natural evolution of alcoholic liver disease culminating as cirrhosis. In some populations, particularly in Europe and Japan, cirrhosis develops in alcoholics without an apparent intermediate stage of alcoholic hepatitis (Karasawa et al. 1980). This observation raises the question as to whether alcohol can promote the development of cirrhosis without being preceded by a stage of alcoholic hepatitis. It now appears from both human and animal studies that alcoholic hepatitis may not be necessary for the development of alcoholic cirrhosis (Popper and Lieber 1980).

According to a new theory, the balloon-like swelling characteristic of alcoholic hepatitis develops as a result of the accumulation within the cell of lipid and protein, which normally would have been secreted into the blood. The retention of such proteins presumably results from degenerative effects of alcohol on subcellular structures (microtubules) that are important for the secretory activity of the liver (Matsuda et al. 1979). Thus, the effect of alcohol may be similar to that of drugs which bind to the cellular proteins and produce lesions similar to those seen in human liver cells with prolonged alcohol use (Denk et al. 1979).

The severity of the various alcohol-associated pathologies of the liver correlates poorly with either the duration of drinking or the amount of alcohol consumed.

Not all patients with alcoholic hepatitis develop cirrhosis, and alcoholic hepatitis appears to occur in 29 percent of male and 47 percent of female alcoholics (Wilkinson et al. 1969; Morgan and Sherlock 1977; Gavalier 1982). Since specific histocompatibility antigens (HLA) are more prevalent in patients with cirrhosis than in controls, these genetic factors might determine an alcoholic's susceptibility to develop cirrhosis (Goodwin 1971; Bell and Nordhagen 1980).

Alcohol and Muscle Systems

Heart Muscle

An association between alcoholism and heart disease has been recognized for more than 100 years. Until recently, however, heart disease was thought to result from malnutrition, particularly thiamine deficiency, and not alcohol itself. Now that thiamine deficiency has been eliminated as an important contributing factor, heart disease has been directly linked to alcoholism.

Specifically, cardiomyopathy, a primary disorder of heart muscle, is often found in chronic heavy drinkers. It generally appears in patients with a long history of alcohol consumption, usually somewhat greater than 10 years in duration. The major symptoms of alcohol cardiomyopathy are chronic shortness of breath and signs of congestive failure such as edema, chest pain, fatigue, palpitations, and blood-stained sputum.

Alcoholic cardiomyopathy does not occur suddenly (Demakis et al. 1974). It gradually progresses from a subclinical condition characterized by minimal cardiac findings and nonspecific electrocardiographic abnormalities to overt congestive heart failure. The severity of the illness appears to be related directly to the duration of alcohol abuse. The prospect for recovery is reasonably good, if the individual ceases to drink, but recovery is slow and may require 5 to 6 years (Fuster et al. 1981).

It is generally believed that damage to the cell mitochondria of the heart muscle is the major mechanism responsible for alcoholic cardiomyopathy (Alexander 1967). Electron microscopic studies clearly show degenerative changes of the mitochondria in response to chronic drinking (Alexander 1967; Alexander et al. 1977b). Other subcellular structures also may be abnormal, leading to depression of mitochondrial respiration, liberation of mitochondrial enzymes, and a reduction of energy-providing ATP in the heart (Alexander et al. 1977a).

In contrast, some epidemiological studies have indicated that alcohol in moderate doses may have a health enhancing effect on the risk for coronary heart disease (Klatsky 1980). However, it has also been shown that at mildly intoxicating doses alcohol adversely affects left ventricular function (Ahmed et al. 1973). Larger doses actually impair the ability of the heart muscle to contract. Thus, alcohol depresses the heart muscle function at blood

concentrations found during alcohol intoxication. Gimeno and coworkers (1962) observed such depression in rats at concentrations of alcohol as low as 0.1 percent. Spann and his colleagues (1968) found that heart contractions of left ventricular muscles, obtained from both normal cats and those with chronic heart failure, are slowed by alcohol. As the alcohol dose is increased, the heart muscle contractions continue to be slowed. Nakano and Moore (1972) observed a similar heart muscle velocity/alcohol dose relationship in guinea pigs.

Delgado and his associates (1975) observed that consuming one to two drinks of Scotch whisky affected the heart in normal subjects 30 to 90 minutes following ingestion. Similarly, Ahmed et al. (1973) found that whisky ingested over 2 hours by normal subjects resulted in a reduction of the duration of systole.

The peripheral circulatory and neurohormonal effects of alcohol tend to obscure the unfavorable action of alcohol on the heart. Both acetaldehyde and acetate (metabolites of alcohol) have been shown to have direct effects on the vascular system resulting in vasodilatation (dilated blood vessels) (Liang and Lowenstein 1978). Acetate also increases cardiac output and left ventricular peak pumping rate and reduces the resistance of peripheral blood vessels, all varying with the alcohol dose (Liang and Lowenstein 1978).

Interestingly, alcohol cannot be metabolized by the heart because alcohol dehydrogenase is absent in the heart (Lochner et al. 1969). Acetaldehyde dehydrogenase is present, however, and thus acetaldehyde can be and is metabolized by cardiac muscle (Forsyth et al. 1973). Despite this lack of alcohol metabolism, Regan and his associates (1969) have shown that following alcohol administration to dogs various ions and enzymes usually found only inside cells are released by heart muscle into the blood. This leakage of cellular contents presumably indicates cellular damage, suggesting that alcohol itself is toxic to the heart. Moreover, Swartz and coworkers (1974) have reported that calcium uptake and binding in the heart, which is needed for muscle contraction, is inhibited by alcohol. Furthermore, Williams and his coworkers (1975) have demonstrated a dose-dependent inhibition of the sodium-potassium-ATPase activity of blood vessel membranes isolated from guinea pig hearts, with both alcohol and acetaldehyde. The enzyme plays a critical role in controlling the amounts of sodium and potassium, nec-

essary for muscle contraction, in the cell.

Cardiac arrhythmias (heart beat irregularities), such as ventricular fibrillation and palpitations, are common in individuals with alcohol-related disease and, in particular, during alcohol intoxication (Ettinger et al. 1978; Greenspon et al. 1981). Gould and his coworkers (1969) have suggested that sudden death probably caused by cardiac arrhythmias may be relatively common among young adults who are found, at autopsy, to have large fatty livers, presumably due to alcohol abuse. It is likely that these alcohol-induced cardiac arrhythmias are due to the direct effects of alcohol upon the heart muscle as well as to the adverse effects of acetaldehyde upon the conduction system. As little as 2 ounces of whisky, consumed by individuals with existing organic heart disease, can suppress the heart's ability to generate muscle contractions that pump the blood throughout the body (Newman and Valicenti 1971). Moreover, another study has indicated that alcohol intoxication can result in a complete heart block (absence of heart beat), requiring pacemaker therapy (Leier et al. 1974).

Finally, it appears that patients with existing heart disease are sensitive to adverse myocardial effects of alcohol or acetaldehyde (Conway 1968; Gould et al. 1971).

Skeletal Muscle

Myopathy, the disease of skeletal muscle, has been associated with alcohol abuse for about 120 years. Modern concepts of alcoholic myopathy began, however, in 1955 with the studies of Ekbom and his associates (1964), who first described a syndrome of muscle weakness without evidence of other signs or symptoms in alcoholics. Eventually, a wide spectrum of alcoholic myopathy has come to be recognized, from the completely asymptomatic to the near fatal (Hed et al. 1962).

Subclinical myopathies occur in more than one-third of alcoholics. Some alcoholics will have a history of muscle cramps, weakness, and occasional episodes of dark urine (myoglobinuria) which is thought to be due to the presence of myoglobin released by damaged muscle tissue (Faris et al. 1967).

The development of muscle weakness in any alcohol-abusing individual could imply acute alcoholic myopathy. Such individuals may experience a sudden attack of muscle pain or a rapid progression of a previously diagnosed chronic myopathy. Such attacks can be fatal. Generally, the muscles are exquisitely tender

during attacks, and are centered in one group of muscles, although on occasion the effect can be diffuse. Fortunately, if alcohol use is discontinued, the disease and its symptoms disappear.

Smooth Muscle

Alcohol's effects on smooth muscle have been recognized only recently. Previously, when alcohol was used as an analgesic for pregnant women, the uterine muscles occasionally stopped contracting. This led researchers to speculate that alcohol directly inhibited smooth muscle contractions (Chapman and Williams 1951). This concept was challenged, however, when Fuchs and Wagner (1963) reported that alcohol interfered with the release of oxytocin, a hormone responsible for uterine contractions, and that this inhibition of hormone activity was the major factor responsible for the diminished uterine contractions. However, more recently, several research teams have shown that alcohol in low concentrations does indeed directly depress uterine contractions (Gimeno et al. 1971; Laversen et al. 1973).

Moreover, Fischer et al. (1965), Winship and his associates (1968), Hogan et al. (1972), and Kjellen and Tibbling (1978) have reported that alcohol reduces sphincter (a smooth muscle) pressure in the lower esophagus. Even more recently, Altura and colleagues (1976, 1978) have shown that both alcohol and acetaldehyde consistently enlarge the peripheral blood vessels, by relaxation of the smooth muscle in the vessel walls.

Alcohol and Blood Disorders

Prolonged protein deprivation and deficiency of B vitamins, particularly riboflavin, pyridoxine, and folic acid, play a role in reducing the formation of new blood cells. In animal studies, protein deficiency or the removal of even a single essential amino acid from the diet can produce anemia with reduced blood formation in the bone marrow, which recovers after resuming a normal diet (Ghitis et al. 1963). Thus, protein deprivation probably plays an important role in the production of both the anemia and the abnormal iron levels seen in alcoholics, particularly those with liver disease.

The close relationship of folic acid deficiency to chronic alcoholism has been recognized for years. Consumption of a folate-poor

diet for several months will deplete body stores, and the diet of most alcoholics contains significantly less than the daily minimum requirement of this vitamin (Wu et al. 1975). Therefore, folic acid deficiency is the primary cause of megaloblastic (abnormal red blood cells) anemia in alcoholics (Sullivan and Herbert 1964; Hines and Cowan 1974). Because folic acid deficiency causes ineffective production of red blood cells, it is associated with increased iron turnover in the blood and a delayed red blood cell incorporation of iron. Therefore, folic acid deficiency leads to increased iron absorption and may be responsible for the increased iron stores in some chronic alcoholics. Impaired liver function may also contribute to these effects. Not only is folate intake reduced, but in individuals with alcoholic liver disease, folate may be released from stores in the liver and lost in the urine, therefore further reducing folate stores (Cherrick et al. 1965). Thus, folate deficient alcoholics may need to be treated with folic acid in therapeutic doses much larger than the amount needed to simply maintain normal function.

The symptoms of riboflavin deficiency, which include anemia, underdeveloped bone marrow, and abnormalities in developing blood cells, are identical to those seen in chronic drinkers. Thus, riboflavin deficiency may also contribute to the anemia present in alcoholics.

Vitamin B₆ is ineffectively converted to its active coenzyme form in alcoholics (Lumeng and Li 1974). The coenzyme deficiency appears to cause several clinical problems in alcoholics, including some forms of anemia as well as changes in the bone marrow, peripheral neurological disease, convulsions, and worsening of liver disease (Davis and Smith 1974; Hines 1975).

Finally, it is possible that alcohol itself may inhibit heme (a constituent of hemoglobin) synthesis in red blood cells (Moore 1974; Ibrahim et al. 1979).

Most alcoholics without iron deficiency have enlarged red blood cells (macrocytosis), a recognized but poorly understood disorder (Bingham 1960). Such blood cell dysmorphism may be the result of a variety of underlying mechanisms, including (1) folate deficiency, (2) increased numbers of immature red blood cells, and (3) increased deposition of cholesterol and phospholipids in red cell membranes, associated with liver disease.

Unger and Johnson (1974) were the first to report that, during routine screening of employees of an insurance company, a number of

alcoholics were discovered to have an elevated MCV (mean corpuscular volume, or average blood cell size) in the absence of other changes usually associated with alcoholism, such as folate deficiency, increased numbers of immature red blood cells, or significant liver disorder. Since then, many reports have confirmed their observations (Buffet et al. 1975; Morin and Porte 1976; Davidson and Hamilton 1978; Wright and Ree 1978). An increased MCV has been found in about 90 percent of alcoholics, which suggests that the MCV may be useful as a marker for alcoholism (Unger and Johnson 1974; Buffet et al. 1975; Morin and Porte 1976). Furthermore, in several large population studies, the number of alcoholic drinks taken daily correlated with the MCV (Whitehead et al. 1978; Eschwege et al. 1978). Even with abstinence, little or no change in the MCV occurs for many weeks; usually 2 to 4 months of abstinence are required before the MCV returns to normal (Unger and Johnson 1974).

Leukopenia (a reduced number of white blood cells) is relatively common in alcoholics (Cowan and Hines 1971). Eichner et al. (1972), for example, observed leukopenia in 8 percent of their cases. Other studies found 1.6 percent (Eichner 1973) and 8.5 percent (Liu 1973) of subjects with leukopenia.

Bone marrow smears obtained from such individuals consistently show a decreased number of cells with markedly decreased numbers of mature granulocytes. The leukopenic episodes in alcoholics tend to be transient, lasting 2 to 4 days, and usually followed by rapid recovery and an increased number of white blood cells.

As long ago as 1938, Pickrell noted in rabbits that white blood cells (which kill intruding bacteria) were inhibited from moving into sites of bacterial infections within the skin and lung during alcohol intoxication. He noted also that the white blood cells attacked the bacteria normally. Similar results have been obtained in mice (Louria 1963) and in humans (Brayton et al. 1970). MacGregor and his associates (1974; Gluckman et al. 1977) have shown that alcohol inhibits the movement of human white blood cells. Alcohol consumption for as short as 6 to 8 days can result in such inhibition. The mechanism by which alcohol impairs such white blood cell functions remains unclear, although it has been suggested that increased intracellular concentrations of cyclic AMP (adenosine monophosphate) after consuming alcohol may be partly responsible for this phenomenon.

There is additional evidence of altered immunologic activity of white blood cells in alcoholics. Studies of animals and humans during periods of alcohol ingestion found that the immunologic response of the skin was reduced (Gluckman et al. 1977). Also, many patients with alcoholic hepatitis have an altered immunity to liver antigens (Young et al. 1979). Furthermore, lymphocytes (one type of white blood cell) from patients with alcoholic liver disease may attack the patients' own liver cells (Paronetto and Lieber 1976). In summary, these observations suggest that abnormalities of lymphocytic function occur in alcoholics and that immunologic mechanisms may account for some of the liver damage induced by alcohol even after abstinence (Lue et al. 1979).

The natural tendency for the blood to coagulate and seal a bleeding wound is disrupted in alcoholics, principally as a complication of advanced liver disease. These changes are similar to those occurring in patients with other chronic liver diseases. Most often, the levels of the clotting factors dependent on vitamin K are decreased. Moreover, the clotting compounds themselves may be abnormal in alcoholics (Weinstein and Deykin 1978; Conoso et al. 1979).

Alcohol and Kidney Disease

Alcoholics with liver disease may have enlarged kidneys with increased fat, protein, and water (Van Thiel et al. 1977). Seven to 14 percent of alcoholics, about 20 times the rate of nonalcoholics, suffer tissue loss in their kidneys (Edmondson et al. 1966; Longacre and Popky 1968). In one study, 12 of 20 alcoholics suffered kidney tissue loss during their first attack of acute kidney infection, with the lower urinary tract the source in most cases. These infections occurred in alcoholics without cirrhosis, suggesting that alcohol abuse and not liver disease was the cause. Such a conclusion is warranted because kidney infection does not accompany other types of severe liver disease.

Alcohol and Pulmonary Disease

Chronic obstructive pulmonary disease is common among alcohol-abusing males, especially smokers (Rankin et al. 1969). Other pulmonary problems such as pneumococcal pneumonia are more likely to occur in alcoholics than in the general population. It is

possible that poor dental health, combined with recurrent abnormal respiration (aspiration) during intoxication, may be responsible for the increased prevalence of bacterial lung abscesses in alcoholics. Tuberculosis is a well-recognized health problem in malnourished alcoholics, and impaired connections between the arteries and veins in the lungs of alcoholics, especially those with advanced liver disease, can cause cyanosis (reduced hemoglobin), hyperventilation, and hypoxia.

Alcohol and the Endocrine System

Gonadal and Adrenal Effects

Low levels of androgens (male hormones) are found in alcoholic men. Thus, 70 to 80 percent experience decreased libido and/or impotence (Van Thiel 1980). In addition, 70 to 80 percent of such men show both testicular atrophy and infertility (Van Thiel and Lester 1979a).

Not only do alcoholic men produce fewer androgens, but such men also produce excess estrogens, the female hormones. Fifty percent of alcoholic men with cirrhosis develop feminine pubic hair patterns, and 20 percent develop enlarged mammary glands (Van Thiel and Lester 1979a). These signs of chronic alcoholism, unlike the transient impotence commonly experienced with an acute alcoholic bout, persist even in the absence of intoxication and are due in large measure to alcohol-induced tissue injury.

Until recently, liver disease was considered to be a primary cause of the sexual dysfunction present in alcoholics. During the past 10 years, however, this concept has been severely challenged, and a diametrically opposite view has gained currency (Van Thiel and Lester 1979a). This recent view is the result of evidence showing that sexual dysfunction can be present in alcoholic men with essentially normal livers (Van Thiel et al. 1974b). Plasma levels of testosterone, a male hormone, are reduced in men within hours after drinking enough alcohol (approximately seven drinks) to produce a hangover (Ylikahri et al. 1974). Many of the features of alcohol-induced sexual dysfunction can be produced in experimental animals with normal livers (Van Thiel et al. 1975a, 1979a). Furthermore, alcohol reduces testosterone levels in mice and rats (Van Thiel and Lester 1979a). Thus, the concept that the sexual changes observed in alcoholic men are the result of alcohol per se, rather than the

indirect consequences of alcohol-induced liver disease, has gained considerable credence.

The specific mechanisms by which alcohol adversely affects testicular function are being unraveled slowly. Vitamin A is essential for creating sperm, and alcohol may interfere with vitamin A activation in the testes (Van Thiel et al. 1974a). Alcohol metabolism may shift the balance between essential enzymes in the testes as it does in the liver, thereby limiting the generation of testosterone (Van Thiel and Lester 1976a). Similarly, acetaldehyde, either produced in the testes or entering the testes from the blood as a result of alcohol metabolism outside the testes, may have deleterious effects upon testicular mitochondria, subcellular organelles that are critical for generating steroids (Gavaler et al. 1983). Indeed, the conversion of cholesterol to the steroid pregnenolone, a reaction that occurs in mitochondria, is inhibited when mitochondria are exposed either to alcohol or to acetaldehyde. Several studies have found a reduced capacity to produce steroids in the testes obtained from alcohol-fed rats (Chiao et al. 1981; Cicero et al. 1980).

Not only is testicular testosterone production inhibited as a result of alcohol exposure, but recent studies have demonstrated that alcohol interferes with gonadotropin (a substance that stimulates the gonads) binding to testicular tissue. Furthermore, chronic drinking is associated with a pituitary-endocrine defect for gonadotropin secretion (Van Thiel et al. 1978c). Alcoholics thus have low gonadotropin concentrations in their blood and respond inadequately to stimuli such as luteinizing hormone releasing factor that provoke gonadotropin release (Van Thiel et al. 1978c). Similar inadequate gonadotropin responses can be demonstrated in chronic alcohol-fed rats and after acute alcohol administration to normal rats (Van Thiel et al. 1979a). The cumulative evidence suggests that chronic drinking induces gonadal injury through its direct effects on the gonad, as well as through indirect effects on the hypothalamus (a part of the brain that integrates endocrine activity) and the pituitary gland.

In addition to having reduced gonad performance, alcoholic men also have relatively high levels of estrogens (Van Thiel and Lester 1976b, 1979a). As noted earlier, they show many of the secondary sex characteristics of women. Such feminization results from increases in estrogen-responsive proteins and the hormone neurophysin (Van Thiel et al. 1975b). The increases in the hormone prolactin

lins and endorphins) on the control of pituitary releasing factors. Such opiates appear to inhibit CRF and thereby ACTH release. They therefore might contribute to reduced ACTH release seen during alcohol intoxication and withdrawal (Morley et al. 1980). This hypothesis is consistent with other observations that opiates alter some of alcohol's effects. For example, the opiate antagonist, naloxone, improves the psychomotor impairment and depressed level of consciousness induced by alcohol (Jeffcoate et al. 1979; Jefferys et al. 1980).

The urine of patients with cirrhosis, especially those with edema, contains increased amounts of the steroid aldosterone, which controls the amount of fluid and salt in the body (Bongiovanni and Eisenmenger 1951). Individuals with cirrhosis are likely to have an increased secretion rate and blood levels of aldosterone (Coppage et al. 1962).

Chronic liver disease is associated with a significantly decreased blood flow in the liver, the site where aldosterone is metabolized. As a result, the clearance of aldosterone from the body is reduced and the duration of the activity of the hormone is prolonged (Coppage et al. 1962). It should be noted, however, that the resultant elevated blood levels of aldosterone in cirrhotic individuals reflect not only a decreased metabolic clearance but also an increased secretion rate of the hormone occurring, at least in part, in response to an increased production of angiotensin, which stimulates aldosterone secretion.

The state of the hypothalamic-pituitary-adrenal axis during alcohol withdrawal has been studied extensively in animals. After alcohol is withdrawn from mice, they are likely to be hyperactive, with seizures and increased corticosteroids in the blood (Goldstein 1972). Chronic alcoholics also experience increased corticosteroid levels during alcohol withdrawal (Stokes 1973).

Interestingly, the pituitary reserve of ACTH in alcoholics experiencing withdrawal is frequently reduced (Wright et al. 1975). Alcoholics who abstain from drinking often show persistent abnormalities of the hypothalamic-pituitary-adrenal axis (Oxenkrug 1978).

In addition to altering steroid hormone release by the adrenal gland, alcohol stimulates the secretion of catecholamines, e.g., epinephrine and norepinephrine, and increases the adrenal content of catecholamine synthesizing enzymes (Pohorecky 1974). Studies in rats have suggested that acetaldehyde is principally responsible for these observed changes in

catecholamine secretion in response to alcohol (Walsh and Truitt 1970).

The Thyroid

The most consistent effect of alcohol on the function of the thyroid is to moderately decrease the hormone thyroxine (T_4) levels in the blood and to markedly decrease the hormone triiodothyronine (T_3) in the blood (Israel et al. 1979). It has been suggested that the low T_3 levels after alcohol consumption reflect injury to the liver and reduced ability of the liver to remove iodine from T_4 (Orrego et al. 1979). Despite the considerable data suggesting that alcohol affects the thyroid (and its interactions with the hypothalamus and pituitary) indirectly via the liver, alcohol can directly cause an increase in the thyroid uptake of iodine, which is needed to synthesize the thyroid's hormones (Yadav et al. 1970). Moreover, Wright (1978) and Van Thiel et al. (1979b) have reported a diminished thyroid stimulating hormone (TSH) response to thyrotropin releasing factor (TRH) in alcoholics.

Growth Hormone and Prolactin

Considerable evidence suggests that alcohol blocks the release of growth hormone. Early studies of laboratory animals suggested that alcohol produces degenerative changes in the pituitary (Sanchez-Calvo 1941). More recently, the acute administration of large doses of alcohol to rats has been shown to abolish spontaneous growth hormone secretion (Redmond 1980). Alcoholics with cirrhosis, in contrast, have elevated levels of growth hormone and frequently respond abnormally to stimuli of growth hormone (Van Thiel et al. 1978b).

Two studies (Torro et al. 1973; Earll et al. 1976) have reported that a single dose of alcohol had no effect on prolactin (a hormone associated with milk-stimulation as well as other functions). However, Loosen and Prange (1977) reported reduced prolactin levels, but with minimally impaired prolactin responses to TRH during alcohol withdrawal. Jung and Russfield (1972) reported an increased prevalence of prolactin-secreting cells in the pituitaries of male and female patients who died of alcoholic liver disease. Ylikahri et al. (1976) noted increased prolactin responses to TRH in alcoholics, a finding confirmed by Van Thiel and his coworkers (1978a, b, c, d). Van Thiel and his associates (1978a, b, c, d) also reported that elevated prolactin levels occur in cir-

rhosis, and that cirrhotics have reduced prolactin responses to TRH. In contrast, individuals with fatty liver have reduced prolactin levels and exaggerated prolactin responses to TRH. Preliminary studies in the rat also suggest that alcohol increases prolactin levels in the blood (Gordon and Southren 1977).

Vasopressin and Oxytocin

Alcohol produces diuresis, which seems to occur because the hormone vasopressin, the body's antidiuretic hormone, is inhibited. Indeed, several groups have found reduced vasopressin levels after acute alcohol administration (Linkola et al. 1977; Helderma et al. 1978). Little or no tolerance to the diuretic effects of alcohol or its inhibition of vasopressin release has been observed in alcoholics (Marquis et al. 1975). Alcohol's suppression of vasopressin release appears to originate in the hypothalamus. Thus, stimuli applied directly to the hypothalamus can override the diuretic effects of alcohol. Alcohol may, however, inhibit the electrically evoked discharges of the hypothalamus (Raiha 1960).

More recently, studies showed that vasopressin was suppressed while blood alcohol levels were increasing. After the maximum blood alcohol levels were attained, the vasopressin levels increased (Marquis et al. 1975).

On the basis of bioassay data, alcohol would appear to inhibit the release of the hormone oxytocin, which is the principal uterine-contracting and lactation-stimulating hormone of the pituitary gland (Wagner and Fuchs 1968). No data, however, are available on the actual blood levels of oxytocin during alcohol administration and withdrawal.

Parathormone

Alcohol causes hypocalcemia (reduced blood calcium) in both dogs and rats (Peng et al. 1972). Parathormone, the parathyroid hormone that controls blood calcium levels, does not reverse this hypocalcemic effect of alcohol (Peng and Gitelman 1974). The fall in calcium may be related to the shift of calcium ions out of the blood (Peng et al. 1972). Some studies in normal volunteers and in alcoholics have suggested that alcohol produces an increased urinary excretion of both calcium and magnesium (Kalbfleisch et al. 1963; Jones et al. 1969). Alcohol has been shown to reduce the transport of calcium in the duodenum (Krawitt 1975). The defect in calcium stability found in alcoholics with hypocalcemia, however, is

most likely due to an alcohol-induced decrease in blood magnesium levels (Estep et al. 1969). Magnesium is well known to be important in the regulation of parathormone secretion and in the mediation of the hormone's peripheral action (Rasmussen 1974). Both malabsorption of calcium and a primary dietary deficiency of vitamin D probably are responsible, at least in part, for the hypocalcemia in individuals with advanced alcoholic cirrhosis. Finally, Williams and his coworkers (1978) recently have reported that alcohol results in an increased level of the hormone calcitonin, which may play a role in developing hypocalcemia.

It is of some interest that a decreased density of tibial bone has been reported in rats chronically fed alcohol (Saville and Lieber 1965). Alcoholic patients do have a decreased bone mass and an increased incidence of fractures (Nilsson 1970). Finally, low phosphate in the blood, which also is common in alcoholics, may contribute to the development of the metabolic bone disease seen in alcoholics (Knochel 1977).

The Endocrine Pancreas

Several studies have shown that alcohol increases insulin secretion in response to glucose (Singh and Patel 1976; Shah et al. 1977). Interestingly, alcohol increases the insulin response to other compounds that stimulate secretion as well as to glucose (Kuhl and Anderson 1974).

Glucose intolerance is common in individuals with alcoholic liver disease, particularly those with portal hypertension (Phillips and Safrit 1971; Kreisberg et al. 1972). Insulin and especially glucagon (a hormone that ultimately increases blood glucose) levels are increased in such individuals (Marco et al. 1973). It is thought that the glucose intolerance in such individuals is due primarily to raised levels of glucagon in the blood. However, abnormal glucose tolerance can occur in alcoholics due to chronic pancreatitis as well as to advanced liver disease.

The syndrome of fasting alcoholic hypoglycemia (low blood sugar) develops within 6 to 36 hours after ingestion of alcohol by a previously malnourished or fasting individual. No age group appears to be exempt from this type of alcohol-induced reaction, but children and women seem to be particularly susceptible. Several studies have reported that alcohol is capable of producing severe and conceivably fatal hypoglycemia in susceptible subjects (Marks and Medd 1964; Arky and Fienkel

1966). In addition to children, athletes are at particular risk for the development of severe hypoglycemia following the combination of alcohol ingestion, fasting, or exercise (Arky et al. 1968).

Alcohol and Cancer

Evidence for the strong associations between chronic alcohol consumption and cancers of the mouth, pharynx, larynx, and esophagus has existed for many years (Lieber et al. 1979). In addition to the cancers of the upper respiratory and alimentary tracts, more recent studies indicate that alcohol may play a role in cancers of the liver, pancreas, stomach, large intestine, rectum, breast, and malignant melanoma (Williams and Horm 1977; Rosenberg et al. 1982; Tuyns 1978).

There are no supporting data from animal studies to clearly show that alcohol by itself is carcinogenic. This issue is further clouded by the fact that, either by accident or as a result of the production process, carcinogenic materials were formerly found in various alcoholic beverages. These substances include nitrosamines, polycyclic hydrocarbons, fusel oils, and asbestos fibers (McGlashan et al. 1968; Wehman and Plantholt 1974; Spiegelholder et al. 1979).

While the basis for the relationship between alcohol and cancer is unknown, it is estimated that alcohol by itself, or in combination with substances such as tobacco, is associated with 3 percent of the cancer deaths in the United States (Rothman 1980; Doll and Peto 1981).

The interaction between alcohol consumption and tobacco is particularly striking in terms of cancers of the mouth, pharynx, and larynx, where the total risk is increased in a synergistic manner (Rothman and Keller 1972; Tuyns 1978; McCoy and Wynder 1979; Schottenfeld 1979). As an example, one study indicated that alcohol consumption and heavy smoking produced up to a fifteenfold increase in the risk of oral cancer, compared with the chances of people who neither drank nor smoked. Heavy smoking alone increased the risk by only twofold to threefold (Lemon et al. 1964; Lyon et al. 1976; Phillips 1980).

Despite the absence of a known mechanism of action, it is clear that heavy alcohol consumption is a risk factor for the development of cancer. To account for this action, several

hypotheses have been generated, including: alcohol acting as a solvent to increase the entry of carcinogens into cells (McCoy and Wynder 1979); alcohol-induced enzyme changes leading to the conversion of procarcinogens (noncarcinogenic substances) into active carcinogens (Seitz et al. 1981a,b; Garro et al. 1981); and repeated alcohol-induced cellular damage promoting cancer in the liver (Lieber 1969).

Summary

Chronic alcohol consumption leads to ubiquitous toxic effects in the body, with medical consequences ranging from slight functional impairment to life-threatening disease states. The most common site of injury is the liver, the organ most responsible for ridding the body of alcohol.

In the early stages of alcoholic liver disease, the liver becomes fatty, a condition that may progress to hepatitis. After an extended period of chronic drinking, the liver structure may be affected, followed by a breakdown in its functional capacity. The result is cirrhosis--an often fatal disease.

Because the liver is so essential for metabolism and detoxification, many organs and body systems may be adversely affected indirectly by alcoholic liver disease. Excessive alcohol also may directly injure the gastrointestinal tract, muscles, and pancreas. As alcohol and its metabolites travel through the body in the blood stream, they may affect the major body systems, including the cardiovascular system, the nervous system, and the endocrine system. Furthermore, there is evidence of a strong association between chronic alcohol use and cancer of the stomach, large intestine, pancreas, and liver.

Many of alcohol's deleterious consequences are the result of effects on the endocrine system hormones. For example, fewer androgens are produced in alcoholic men, often leading to a feminization of their appearance, decreased libido, and infertility. Alcoholic women are affected also, frequently with ovarian dysfunction. Alone, and in conjunction with dietary deficiencies frequently associated with alcoholism, alcohol abuse can produce nutrition-related deficits in the body. These deficits result in altered protein metabolism and vitamin-related disorders such as anemia.

References

- Ahmed, S.S.; Levinson, G.E.; and Regan, T.J. Depression of myocardial contractility with low doses of ethanol in normal man. *Circulation* 48: 378-385, 1973.
- Alexander, C.S. Electron microscopic observations in alcoholic heart disease. *British Heart Journal* 29:200-206, 1967.
- Alexander, C.S.; Forsyth, G.W.; and Nagasawa, H.T. Alcoholic cardiomyopathy in mice: Myocardial glycogen, lipids and certain enzymes. *Journal of Molecular and Cellular Cardiology* 9:235-245, 1977a.
- Alexander, C.S.; Sekhri, K.K.; and Nagasawa, H.T. Alcoholic cardiomyopathy in mice: Electron microscopic observations. *Journal of Molecular and Cellular Cardiology* 9:247-254, 1977b.
- Altura, B.M.; Carella, A.; and Altura, B.T. Acetaldehyde on vascular smooth muscle: Possible role in vasodilator action of ethanol. *European Journal of Pharmacology* 52:73-83, 1978.
- Altura, B.M.; Edgar, H.; and Altura, B.T. Differential effects of ethanol and mannitol on contraction of arterial smooth muscle. *Journal of Pharmacology and Experimental Therapeutics* 197(2):352-361, 1976.
- Arky, R.A., and Fienkel, N. Alcohol hypoglycemia. V. Alcohol infusion to test gluconeogenesis in starvation, with special reference to obesity. *New England Journal of Medicine* 274:426-433, 1966.
- Arky, R.A.; Veverbrants, E.; and Abramson, E.A. Irreversible hypoglycemia; a complication of alcohol and insulin. *Journal of the American Medical Association* 206:575-578, 1968.
- Baraona, E.; Pirola, R.C.; and Lieber, C.S. Small intestine damage and changes in cell population produced by ethanol ingestion in the rat. *Gastroenterology* 66:226-234, 1974.
- Baraona, E.; Pirola, R.C.; and Lieber, C.S. Acute and chronic effects of ethanol on intestinal lipid metabolism. *Biochimica et Biophysica Acta* 388: 19-28, 1975.
- Bell, H., and Nordhagen, R. HLA antigens in alcoholics with special reference to alcoholic cirrhosis. *Scandinavian Journal of Gastroenterology* 15:453-456, 1980.
- Bingham, J. The macrocytosis of hepatic disease. II. Thick macrocytes. *Blood* 15(2):244-254, 1960.
- Bode, J.C.H. Alcohol and the gastrointestinal tract. In: Bode, J.C.H., ed. *Systemic Illness and the Gastrointestinal Tract*. Stuttgart: Georg Thieme, 1981.
- Bode, J.C., and Thiele, D. Hemmung des Aethanolabbaus beim Menschen durch Fasten: Reversibilitaet durch Fructose-Infusion [Inhibition of ethanol catabolism in humans through fasting: Reversibility through fructose infusion]. *Deutsche Medizinische Wochenschrift* 100:1849-1851, 1975.
- Bongiovanni, A.M., and Eisenmenger, W.J. Adrenal cortical metabolism in chronic liver disease. *Journal of Clinical Endocrinology and Metabolism* 11:152-172, 1951.
- Bosron, W.F., and Li, T.-K. Alcohol dehydrogenase. In: Jakoby, W.B., ed. *Enzymatic Basis of Detoxification*. Vol. 1. New York: Academic Press, 1980. pp. 231-248.
- Bosron, W.F.; Li, T.-K.; Däfeldecker, W.P.; and Vallee, B.L. Human liver pi-alcohol dehydrogenase: Kinetic and molecular properties. *Biochemistry* 18:1101-1105, 1979.
- Brayton, R.G.; Stokes, P.E.; Schwartz, M.S.; and Louria, D.B. Effect of alcohol and various diseases on leukocyte mobilization, phagocytosis and intracellular bacterial killing. *New England Journal of Medicine* 282:123-128, 1970.
- Brooks, F.P., and Thomas, J.E. The effect of alcohol on canine external pancreatic secretion. *Gastroenterology* 23:36-42, 1953.
- Buffet, C.; Choput, J.-C.; and Albuissou, F. La macrocytose dans l'hepatite alcoolique chronique histologiquement prouvee [Histologically confirmed macrocytosis in chronic alcoholic hepatitis]. *Archives Francaises des Maladies de l'Appareil Digestif* 64:309-315, 1975.
- Burch, G.E., and Ansari, A. Chronic alcoholism and carcinoma of the pancreas: A correlative hypothesis. *Archives of Internal Medicine* 122:273-275, 1968.
- Cederbaum, A.I.; Lieber, C.S.; and Rubin, E. Effect of acetaldehyde on fatty acid oxidation and ketogenesis by hepatic mitochondria. *Archives of Biochemistry and Biophysics* 169:29-41, 1975.
- Chapman, E.R., and Williams, P.T. Intravenous alcohol on an obstetrical analgesia. *American Journal of Obstetrics and Gynecology* 61:676-679, 1951.
- Chaput, J.-C.; Petite, J.-P.; Gueroult, N.; Buffet, C.; Regensberg, M.; and Etienne, J.-P. La fibroscopie d'urgence dans les hemorrhagies digestives des cirrhoses: A propos de 100 cas [Emergency fibroscopy in digestive hemorrhages in cirrhosis: 100 cases]. *Nouvelle Presse Medicale* 3:1227-1230, 1974.
- Charlton, R.W.; Jacobs, P.; Stefel, H.; and Bothwell, J.H. Effect of alcohol on iron absorption. *British Medical Journal* 2:1427-1429, 1964.
- Cherrick, G.R.; Baker, H.; Frank, O.; and Leevy, C.M. Observations on hepatic avidity for folate in Laennec's cirrhosis. *Journal of Laboratory and Clinical Medicine* 66:446-451, 1965.
- Chiao, Y.-B.; Johnston, D.E.; Gavalier, J.S.; and Van Thiel, D.H. Effect of chronic ethanol feeding on testicular content of enzymes required for testosteroneogenesis. *Alcoholism: Clinical and Experimental Research* 5:230-236, 1981.
- Cicero, T.J.; Bell, R.D.; Meyer, E.R.; and Badger, T.M. Ethanol and acetaldehyde directly inhibit testicular steroidogenesis. *Journal of Pharmacology and Experimental Therapeutics* 213:228-233, 1980.
- Cohen, B.I., and Raicht, R.F. Effect of ethanol on

- sterol metabolism in the rat (abstract). Alcoholism: Clinical and Experimental Research 4:212, 1980.
- Conoso, R.T.; Hutton, R.A.; and Deykin, D. The hemostatic defect of chronic liver disease. Kinetic studies using ^{75}Se -selenomethionine. Gastroenterology 76:540-547, 1979.
- Conway, N. Hemodynamic effects of ethyl alcohol in coronary heart disease. American Heart Journal 76:581-582, 1968.
- Cooke, A.R. Ethanol and gastric function. Gastroenterology 62:501-502, 1972.
- Coppage, W.S.; Island, D.P.; Cooner, A.E.; and Liddle, G.W. The metabolism of aldosterone in normal subjects and in patients with hepatic cirrhosis. Journal of Clinical Investigation 41:1672-1680, 1962.
- Cowan, D.H., and Baunach, D.C. Platelet cyclic AMP reduction in alcohol-related thrombocytopathia. Abstracts of the Meeting of the American Society of Hematology, p. 198, 1976.
- Cowan, D.H., and Hines, J.D. Thrombocytopenia of severe alcoholism. Annals of Internal Medicine 74:37-43, 1971.
- Czaja, C., and Kalant, H. The effect of acute alcohol intoxication on adrenal ascorbic acid and cholesterol in the rat. Canadian Journal of Biochemistry 39:327-334, 1961.
- Darle, N.; Ekholm, R.; and Edlund, Y. Ultrastructure of the rat exocrine pancreas after long term intake of ethanol. Gastroenterology 58:62-72, 1970.
- Davidson, R.J.L., and Hamilton, P.J. High mean red cell volume: Its incidence and significance in routine haematology. Journal of Clinical Pathology 31:493-498, 1978.
- Davis, R.E., and Smith, B.K. Pyridoxal and folate deficiency in alcoholics. Medical Journal of Australia 2:357-360, 1974.
- Delgado, C.E.; Fortuin, N.J.; and Ross, R.S. Acute effects of low doses of alcohol on left ventricular function by echocardiography. Circulation 51:535-540, 1975.
- Demakis, J.G.; Proskey, A.; Rahimtoola, S.H.; Jamil, M.; Sutton, G.C.; Rosen, K.M.; Gunnar, R.M.; and Tobin, J.R. The natural course of alcoholic cardiomyopathy. Annals of Internal Medicine 80:293-297, 1974.
- Denk, H.; Franke, W.W.; Eckerstorfer, R.; Schmid, E.; and Kerjaschki, D. Formation and involution of Mallory bodies ("alcoholic hyalin") in murine and human liver revealed by immunofluorescence microscopy with antibodies to prekeratin. Proceedings of the National Academy of Sciences of the United States of America 76:4112-4116, 1979.
- DeSchepper, P.J.; Tjandramaga, T.B.; DeRoo, M.; Verhaest, L.; Daurio, C.; Steelman, S.L.; and Tempero, K.F. Gastrointestinal blood loss after diflunisal and after aspirin: Effect of ethanol. Clinical Pharmacology and Therapeutics 23:669-676, 1978.
- Doll, R., and Peto, R. The causes of cancer: Quantitative estimates of avoidable risks of cancer in the United States today. Journal of the National Cancer Institute 66:1191-1308, 1981.
- Earll, J.M.; Gaunt, K.; Earll, L.A.; and Djuh, Y.Y. Effect of ethyl alcohol on ionic calcium and prolactin in man. Aviation, Space, and Environmental Medicine 47:808-810, 1976.
- Edmondson, H.A.; Reynolds, T.B.; and Jacobson, H.G. Renal papillary necrosis with special reference to chronic alcoholism: A report of 20 cases. Archives of Internal Medicine 118:225-264, 1966.
- Eichner, E.R. The hematologic disorders of alcoholism. American Journal of Medicine 54:621-630, 1973.
- Eichner, E.R.; Buchanan, B.; Smith, J.W.; and Hillman, R.S. Variations in the hematologic and medical status of alcoholics. American Journal of the Medical Sciences 263:35-42, 1972.
- Ekblom, K.; Hed, R.; Kirstein, L.; and Astrom, K.-E. Muscular affections in chronic alcoholism. Archives of Neurology 10:449-458, 1964.
- Ellis, F.W. Effect of ethanol on plasma corticosterone levels. Journal of Pharmacology 153:121-127, 1966.
- Elwin, C.-E. Some factors influencing the stimulatory effect of ethanol on gastric acid secretion during antrum application. Acta Physiologica Scandinavica 75:12-27, 1969.
- Eschwege, E.; Papor, L.; Lellouch, J.; and Claude, J.R. Blood cells and alcohol consumption with special reference to smoking habits. Journal of Clinical Pathology 31:654-658, 1978.
- Estep, H.; Shaw, W.A.; Watlington, C.; Hobe, R.; Holland, W.; and Tucker, S.-G. Hypocalcemia due to hypomagnesemia and reversible parathyroid hormone unresponsiveness. Journal of Clinical Endocrinology 29:842-848, 1969.
- Ettinger, P.O.; Wu, C.F.; De la Cruz, C.; Weisse, A.B.; Ahmed, S.S.; and Regan, T.J. Arrhythmias and the "holiday heart": Alcohol-associated cardiac rhythm disorders. American Heart Journal 95:555-562, 1978.
- Faraj, B.A.; Bowen, P.A.; Isaacs, J.W.; and Rudman, D. Hypertyrminemia in cirrhotic patients. New England Journal of Medicine 294:1360-1364, 1976.
- Faraj, B.A.; Fulenwider, J.T.; Rypins, E.B.; Nordlinger, B.; Ivey, G.L.; Jansen, R.D.; Ali, F.M.; Camp, V.M.; Kutner, M.; Schmidt, F.; and Rudman, D. Tyramine kinetics and metabolism in cirrhosis. Journal of Clinical Investigation 64:413-420, 1979.
- Faris, A.A.; Reyes, M.G.; and Abrams, B.M. Subclinical alcoholic myopathy; Electromyographic and biopsy study. Transactions of the American Neurological Association 92:102-106, 1967.
- Fauchet, R.; Genetet, B.; Gosselin, J.; and Gastrad, J. HLA antigens in chronic alcoholic pancreatitis. Tissue Antigens 13:163-166, 1979.
- Fazekas, I.G. Hydrocortisone content of human blood, and alcohol content of blood and urine, after wine consumption. Quarterly Journal of Studies on Alcohol 27:439-446, 1966.
- Fischer, R.A.; Ellison, G.W.; Thayer, W.R.; Spiro, H.M.; and Glaser, G.H. Esophageal motility in neuromuscular disorders. Annals of Internal Medicine 63:229-248, 1965.

- Forbes, J.C., and Duncan, G.M. The effect of acute alcohol intoxication on the adrenal glands of rats and guinea pigs. Quarterly Journal of Studies on Alcohol 12:355-359, 1951.
- Forsyth, G.W.; Nagasawa, H.T.; and Alexander, C.S. Acetaldehyde metabolism by the rat heart. Proceedings of the Society for Experimental Biology and Medicine 144:498-500, 1973.
- Fuchs, A.-R., and Wagner, G. The effect of ethyl alcohol on the release of oxytocin in rabbits. Acta Endocrinologica 44:593-605, 1963.
- Fuster, V.; Gersh, B.J.; Giuliani, E.R.; Tajik, A.J.; Brandenburg, R.O.; and Frye, R.L. The natural history of idiopathic dilated cardiomyopathy. American Journal of Cardiology 47:525-531, 1981.
- Garro, A.J.; Seitz, H.K.; and Lieber, C.S. Enhancement of dimethylnitrosamine metabolism and activation to a mutagen following chronic ethanol consumption. Cancer Research 41:120-124, 1981.
- Gavaler, J.S. Sex-related differences in ethanol-induced liver disease: Artifactual or real? Alcoholism: Clinical and Experimental Research 6:186-196, 1982.
- Gavaler, J.S.; Perez, H.A.; Estes, L.; and Van Thiel, D.H. Morphologic alterations of rat Leydig cells induced by ethanol. Pharmacology, Biochemistry and Behavior 18(Supplement 1):341-347, 1983.
- Ghitis, J.; Piazeulo, E.; and Vitale, J.J. Cal-Harvard nutrition project. III. The erythroid atrophy of severe protein deficiency in monkeys. American Journal of Clinical Nutrition 12:452-454, 1963.
- Gimeno, A.L.; Gimeno, M.F.; and Webb, J.L. Effects of ethanol on cellular membrane potentials and contractility of isolated rat atrium. American Journal of Physiology 203:194-196, 1962.
- Gimeno, M.F.; Bedners, A.S.; DeVastik, F.J.; and Gimeno, A.L. Effect of ethanol on the motility of isolated rat myometrium. Archives Internationales de Pharmacodynamie et de Therapie 191:213-219, 1971.
- Gluckman, S.J.; Dvorak, V.C.; and MacGregor, R.R. Host defenses during prolonged alcohol consumption in a controlled environment. Archives of Internal Medicine 137:1539-1543, 1977.
- Goldstein, D.B. An animal model for testing effects of drugs on alcohol withdrawal reactions. Journal of Pharmacology and Experimental Therapeutics 183:14-22, 1972.
- Goodwin, D.W. Is alcoholism hereditary? A review and critique. Archives of General Psychiatry 25:545-549, 1971.
- Gordon, G.G.; Olivo, J.; Rafii, F.; and Southren, A.L. Conversion of androgens to estrogens in cirrhosis of the liver. Journal of Clinical Endocrinology and Metabolism 40:1018-1026, 1975.
- Gordon, G.G., and Southren, A.L. Metabolic effects of alcohol on the endocrine system. In: Lieber, C.S., ed. Metabolic Aspects of Alcoholism. Baltimore: University Park Press, 1977. pp. 249-302.
- Gordon, G.G.; Southren, A.L.; and Lieber, C.S. Hypogonadism and feminization in the male: A triple effect of alcohol. Alcoholism: Clinical and Experimental Research 3:210-212, 1979.
- Gottfried, E.B.; Korsten, M.A.; and Lieber, C.S. Gastritis and duodenitis induced by alcohol: An endoscopic and histologic assessment (abstract). Gastroenterology 70:A-32/890, 1976.
- Gould, L.; Shariff, M.; Zahir, M.; and Di Lieto, M. Cardiac hemodynamics in alcoholic patients with chronic liver disease and a presystolic gallop. Journal of Clinical Investigation 48:860-868, 1969.
- Gould, L.; Zahir, M.; DeMartino, A.; and Gomprecht, R.F. Cardiac effects of a cocktail. Journal of the American Medical Association 218:1799-1802, 1971.
- Greenspon, A.J.; Leier, C.B.; and Schaal, S.F. Acute ethanol effects cardiac arrhythmias and conduction in man (abstract). Clinical Research 29:200A, 1981.
- Halle, P.; Pare, P.; Kaptein, E.; Kanel, G.; Redeker, A.G.; and Reynolds, T.B. Double-blind, controlled trial of propylthiouracil in patients with severe acute alcoholic hepatitis. Gastroenterology 82:925-931, 1982.
- Halsted, C.H.; Griggs, R.C.; and Harris, J.W. The effect of alcoholism on the absorption of folic (H^3 PGA) evaluated by plasma levels and urine excretion. Journal of Laboratory and Clinical Medicine 69:116-131, 1967.
- Halsted, C.H.; Robles, E.A.; and Mezey, E. Intestinal malabsorption in folate-deficient alcoholics. Gastroenterology 64:526-532, 1973.
- Hed, R.; Lundmark, C.; Fahlgren, H.; and Orell, S. Acute muscular syndrome in chronic alcoholism. Acta Medica Scandinavica 171:585-599, 1962.
- Helderman, J.H.; Vestal, R.E.; Rowe, J.W.; Tobin, J.; Andres, R.; and Robertson, G. The response of arginine vasopressin to intravenous ethanol and hypertonic saline in man: The impact of aging. Journal of Gerontology 33:39-47, 1978.
- Hines, J.D., and Cowan, D.H. Anemia in alcoholism. In: Dimitrov, N.V., and Nodine, J.H., eds. Drugs and Hematologic Reactions. New York: Grune & Stratton, 1974. pp. 141-153.
- Hion-Jon, V. The influence of alcohol on the endocrine glands. Folia Neuropathologica Estoniana 3:288-301, 1925.
- Hogan, W.J.; Viegas De Andrade, S.R.; and Winship, D.H. Ethanol induced acute esophageal motor dysfunction. Journal of Applied Physiology 32:755-760, 1972.
- Hoyumpa, A.M.; Breen, K.J.; and Schenker, S. Thiamine transport across the rat intestine: II. Effect of ethanol. Journal of Laboratory and Clinical Medicine 86:803-816, 1975.
- Hoyumpa, A.; Middleton, H.; Wilson, F.; and Schenker, S. Dual system of thiamine transport: Characteristics and effect of ethanol (abstract). Gastroenterology 66:A-60/714, 1974.
- Hoyumpa, A.M.; Nichols, S.G.; Wilson, F.A.; and Schenker, S. Effect of ethanol on interstitial (Na,K) ATPase and intestinal thiamine transport in rats. Journal of Laboratory and Clinical Medicine 90:1086-1095, 1977.

- Ibrahim, N.G.; Spieler, P.J.; and Freedman, M.L. Ethanol inhibition of rabbit reticulocyte haem synthesis at the level of delta-aminolaevulinic acid synthetase. British Journal of Haematology 41:235-243, 1979.
- Israel, Y.; Salazar, L.; and Rosenmann, E. Inhibitory effects of alcohol on intestinal amino acid transport in vivo and in vitro. Journal of Nutrition 96:499-504, 1968.
- Israel, Y.; Videla, L.; MacDonald, A.; and Bernstein, J. Metabolic alteration produced in the liver by chronic ethanol administration. Biochemical Journal 134:523-529, 1973.
- Israel, Y.; Walfish, P.G.; Orrego, H.; Blake, J.; and Kalant, H. Thyroid hormones in alcoholic liver disease. Effect of treatment with 6-n-propylthiouracil. Gastroenterology 76:116-122, 1979.
- Jeffcoate, W.J.; Herbert, M.; Cullen, M.H.; Hastings, A.G.; and Walder, C.P. Prevention of effects of alcohol intoxication by naloxone. Lancet 2: 1157-1159, 1979.
- Jefferys, D.B.; Flanagan, R.J.; and Volans, G.N. Reversal of ethanol-induced coma with naloxone (letter). Lancet 1:308-309, 1980.
- Jones, J.E.; Shane, S.R.; Jacob, W.H.; and Flink, E.B. Magnesium balance studies in chronic alcoholism. Annals of the New York Academy of Sciences 162:934-946, 1969.
- Jung, Y., and Russfield, A.B. Prolactin cells in the hypophysis of cirrhotic patients. Archives of Pathology 94:265-269, 1972.
- Kalbfleisch, J.J.; Lindeman, R.D.; Ginn, H.E.; and Smith, W.O. Effects of ethanol administration on urinary excretion of magnesium and other electrolytes in alcoholic and normal subjects. Journal of Clinical Investigation 42:1471-1475, 1963.
- Karasawa, T.; Kushida, T.; Shikata, T.; and Kaneda, H. Morphologic spectrum of liver diseases among chronic alcoholics: A comparison between Tokyo, Japan, and Cincinnati, U.S.A. Acta Pathologica Japonica 30:505-514, 1980.
- Kershenovich, D.; Fierro, F.J.; and Rojkind, M. The relationship between the free pool of proline and collagen content in human liver cirrhosis. Journal of Clinical Investigation 49:2246-2249, 1970.
- Kjellen, G., and Tibbling, L. Influence of body position, dry and water swallows, smoking and alcohol on esophageal acid clearing. Scandinavian Journal of Gastroenterology 13:283-288, 1978.
- Klatsky, A. "The Kaiser-Permanente Experience." Paper presented at the symposium on Alcohol and Cardiovascular Disease of the 20th Annual Conference on Cardiovascular Epidemiology, San Diego, 1980.
- Knochel, J.P. The pathophysiology and clinical characteristics of severe hypophosphatemia. Archives of Internal Medicine 137:203-220, 1977.
- Krawitt, E.L. Ethanol inhibits intestinal calcium transport in rats. Nature 243:88-89, 1973.
- Krawitt, E.L. Effect of ethanol ingestion on duodenal calcium transport. Journal of Laboratory and Clinical Medicine 85:665-671, 1975.
- Krebs, H.A.; Hems, R.; and Lund, P. Accumulation of amino acids by the perfused rat liver in the presence of ethanol. Biochemical Journal 134: 697-705, 1973.
- Kreisberg, R.A.; Owen, W.C.; and Siegal, A.M. Hyperlacticacidemia in man: Ethanol-phenformin synergism. Journal of Clinical Endocrinology and Metabolism 34:29-35, 1972.
- Kuhl, C., and Anderson, O. Glucose- and tolbutamide-mediated insulin response after preinfusion with ethanol. Diabetes 23:821-826, 1974.
- Kuo, Y.-J., and Shanbour, L.L. Effects of ethanol on sodium, 3-O-methyl glucose, and L-alanine transport in the jejunum. American Journal of Digestive Diseases 123:51-56, 1978.
- Lane, F.; Goff, P.; McGuffin, R.; Eichner, E.R.; and Hillman, R.S. Folic acid metabolism in normal, folate deficient and alcoholic man. British Journal of Haematology 34:489-500, 1976.
- Larato, D.C. Oral tissue changes in the chronic alcoholic. Journal of Periodontology 43:772-773, 1972.
- Laversen, N.H.; Raghavan, K.S.; Wilson, K.H.; Fuchs, F.; and Niemann, W.H. Effects of prostaglandin F₂alpha oxytocin, and ethanol on the uterus of the pregnant baboon. American Journal of Obstetrics and Gynecology 115:912-918, 1973.
- Levy, C.M.; Baker, H.; Ten Hove, W.; Frank, O.; and Cherrick, G.R. B-complex vitamins in liver disease on the alcoholic. American Journal of Clinical Nutrition 16:339-346, 1965.
- Lefevre, A.; Adler, H.; and Lieber, C.S. Effect of ethanol on ketone metabolism. Journal of Clinical Investigation 49:1775-1782, 1970.
- Leier, C.V.; Schaal, S.F.; Leighton, R.F.; and Whayne, T.F. Heart block in alcoholic cardiomyopathy. Archives of Internal Medicine 134: 766-768, 1974.
- Lemon, F.R.; Walden, R.T.; and Woods, R.W. Cancer of the lung and mouth in Seventh-Day Adventists. Cancer 17:486-497, 1964.
- Liang, C.S., and Lowenstein, J.M. Metabolic control of the circulation: Effects of acetate and pyruvate. Journal of Clinical Investigation 62 (5):1029-1038, 1978.
- Lieber, C.S. Chronic alcoholic hepatic injury in experimental animals and man: Biochemical pathways and nutritional factors. Federation Proceedings 26:1443-1448, 1967.
- Lieber, C.S. Alcohol and the liver. In: Bittar, E.E., and Bittar, N., eds. The Biological Basis of Medicine. Vol. 5. New York: Academic Press, 1969. pp. 317-344.
- Lieber, C.S. Pathogenesis of alcoholic liver disease: An overview. In: Fisher, M.M., and Rankin, J.G., eds. Alcohol and the Liver. Hepatology: Research and Clinical Issues, Vol. 3. New York: Plenum Press, 1977. pp. 197-255.
- Lieber, C.S. Metabolic effects of ethanol on the liver and other digestive organs. In: Levy, C.M., ed. Alcohol and the GI Tract. Clinics in Gastroenterology, Vol. 10, No. 2. London: W.B. Saunders, 1981. pp. 315-342.
- Lieber, C.S., and DeCarli, L.M. Study of agents for the prevention of the fatty liver produced by prolonged alcohol intake. Gastroenterology 50: 316-322, 1966.

- Lieber, C.S., and DeCarli, L.M. Ethanol oxidation by hepatic microsomes. Adaptive increase after ethanol feeding. *Science* 162:917-918, 1968.
- Lieber, C.S.; Jones, D.P.; and DeCarli, L.M. Effects of prolonged ethanol intake: Production of fatty liver despite adequate diet. *Journal of Clinical Investigation* 44:1009-1021, 1965.
- Lieber, C.S.; Seitz, H.K.; Garro, A.J.; and Worner, T.M. Alcohol-related diseases and carcinogenesis. *Cancer Research* 39:2863-2886, 1979.
- Lieber, C.S., and Spritz, N. Effects of prolonged ethanol intake in man: Role of dietary, adipose, and endogenously synthesized fatty acids in the pathogenesis of the alcoholic fatty liver. *Journal of Clinical Investigation* 45:1400-1411, 1966.
- Linkola, J.; Fyhrquist, F.; and Forsander, O. Effects of ethanol in urinary arginine vasopressin excretion in two rat strains selected for their different ethanol preferences. *Acta Physiologica Scandinavica* 101:126-128, 1977.
- Liu, Y.K. Leukopenia in alcoholics. *American Journal of Medicine* 54:605-610, 1973.
- Lochner, A.; Cowley, R.; and Brink, A.J. Effects of ethanol on metabolism and function of perfused rat heart. *American Heart Journal* 78:770-780, 1969.
- Longacre, A.M., and Popky, G.L. Papillary necrosis in patients with cirrhosis: A study of 102 patients. *Journal of Urology* 99:391-395, 1968.
- Loosen, P.T., and Prange, A.J. Alcohol and anterior pituitary secretion. *Lancet* 2:985, 1977.
- Louria, D.B. Susceptibility to infection during experimental alcohol intoxication. *Transactions of the Association of American Physicians* 76:102-112, 1963.
- Lue, S.L.; Paronetto, F.; and Lieber, C.S. Cytotoxicity of lymphocytes in alcoholic fatty liver: Respective role of lymphocytes and target cells (abstract). *Gastroenterology* 76:1290, 1979.
- Lumeng, L., and Li, T.-K. Vitamin B₆ metabolism in chronic alcohol abuse: Pyridoxal phosphate levels in plasma and the effects of acetaldehyde on pyridoxal phosphate synthesis and degradation in human erythrocytes. *Journal of Clinical Investigation* 53:693-704, 1974.
- Lyon, J.L.; Klauber, M.R.; Gardner, J.W.; and Smart, C.R. Cancer incidence in Mormons and non-Mormons in Utah, 1966-1970. *New England Journal of Medicine* 294:129-133, 1976.
- MacGregor, R.R.; Spagnuolo, P.J.; and Lentnek, A.L. Inhibition of granulocyte adherence by ethanol, prednisone and aspirin, measured with an assay system. *New England Journal of Medicine* 291:642-646, 1974.
- Main, L.H.M., and Whittle, B.J.R. The role of prostaglandins in gastric acid secretion. In: Case, R.J., and Goebell, H., eds. *Stimulus-Secretion Coupling in the Gastrointestinal Tract*. Baltimore: University Park Press, 1976. pp. 147-157.
- Marco, J.; Diego, J.; Villanueva, M.L.; Diaz-Fierros, M.; Valverde, I.; and Segovia, J.M. Elevated plasma glucagon levels in cirrhosis. *New England Journal of Medicine* 289:1107-1111, 1973.
- Marks, V., and Medd, W.E. Alcohol induced hypoglycemia. *British Journal of Psychiatry* 110:228-232, 1964.
- Marquis, C.; Marchetti, J.; Burlet, C.; and Boulande, M. Secretion urinaire et hormone antidiurétique chez des rats soumis à une administration répétée d'éthanol [Urinary secretion and antidiuretic hormone in rats subjected to repeated ethanol administration]. *Comptes Rendus des Seances de la Société de Biologie et de ses Filiales* 169:154-161, 1975.
- Martin, J.L.; Justus, P.G.; and Mathias, J.R. Altered motility of the small intestine in response to ethanol (ETOH): An explanation for the diarrhea associated with the consumption of alcohol (abstract). *Gastroenterology* 78:1218, 1980.
- Martin, S., and Pangborn, R.M. Human parotid secretion in response to ethyl alcohol. *Journal of Diet Research* 50:485-490, 1971.
- Martini, G.A., and Wienbeck, M. Begünstigt Alkohol die Entstehung eines Barrett-Syndroms (Endobrachyoesophagus) [Does alcohol promote the development of Barrett's syndrome (endobrachy-esophagus)]. *Deutsche Medizinische Wochenschrift* 99:434-439, 1974.
- Matsuda, Y.; Baraona, E.; Salaspuuro, M.; and Lieber, C.S. Effects of ethanol on liver microtubules and Golgi apparatus: Possible role in altered hepatic secretion of plasma proteins. *Laboratory Investigation* 41:455-463, 1979.
- Matsuzaki, S.; Gordon, E.; and Lieber, C.S. Increased alcohol dehydrogenase independent ethanol oxidation at high ethanol concentrations in isolated rat hepatocytes: The effect of chronic ethanol feeding. *Journal of Pharmacology and Experimental Therapeutics* 217:133-137, 1981.
- Mayer, E.M.; Grabowski, C.J.; and Fisher, R.S. Effects of graded doses of alcohol upon esophageal motor function. *Gastroenterology* 75:1133-1136, 1978.
- McCoy, G.D., and Wynder, E.L. Etiological and preventive implications in alcohol carcinogenesis. *Cancer Research* 39:2844-2850, 1979.
- McGlashan, N.D.; Walters, C.L.; and McLean, A.E.M. Nitrosamines in African alcoholic spirits and esophageal cancer. *Lancet* 2:1017, 1968.
- Mezey, E. Ethanol: Metabolism and adverse effects. *Viewpoints on Digestive Diseases* 11:215-219, 1979.
- Middleton, W.R.J.; Carter, E.A.; Drummey, G.D.; and Isselbacher, K.J. Effect of oral ethanol administration on intestinal cholesterologenesis in the rat. *Gastroenterology* 60:880-887, 1971.
- Moore, M.R. The effects of ethanol on heme biosynthesis (abstract). In: Thurman, R.G.; Yonetani, T.; Williamson, J.R.; and Chance, B., eds. *Alcohol and Aldehyde Metabolizing Systems*. New York: Academic Press, 1974. p. 543.
- Morgan, M.Y., and Sherlock, S. Sex-related differences among 100 patients with alcoholic liver disease. *British Medical Journal* 1:939-941, 1977.
- Morin, J., and Porte, P. Macrocytose érythrocytaire chez les éthyliques [Erythrocyte macrocytosis in alcoholics] (letter). *Nouvelle Presse Médicale* 5:273, 1976.

- Morland, J.; Bessesen, A.; and Svendsen, L. Incorporation of labelled amino acids into proteins of isolated parenchymal and nonparenchymal rat liver cells in the absence and presence of ethanol. *Biochimica et Biophysica Acta* 561:464-474, 1979.
- Morley, J.E.; Baranetsky, N.G.; Wingert, T.D.; Carlson, H.E.; Hershman, J.M.; Melmed, S.; Levin, S.R.; Jamison, K.R.; Weitzman, R.; Chang, R.S.; and Varner, A.A. Endocrine effects of naloxone-induced opiate receptor blockade. *Journal of Clinical Endocrinology and Metabolism* 50:251-257, 1980.
- Murray, J., and Stein, N. Effect of ethanol on absorption of iron in rats. *Proceedings of the Society for Experimental Biology and Medicine* 120:816-819, 1965.
- Nakamura, K.; Sarles, H.; and Payan, H. Three-dimensional reconstruction of the pancreatic ducts in chronic pancreatitis. *Gastroenterology* 62:942-949, 1972.
- Nakano, J., and Moore, S.E. Effects of different alcohols on the contractile force of the isolated guinea pig myocardium. *European Journal of Pharmacology* 20:266-270, 1972.
- Newman, W.H., and Valicenti, J.F. Ventricular function following acute alcohol administration: A strain-gauge analysis of depressed ventricular dynamics. *American Heart Journal* 81:61-68, 1971.
- Nilsson, B.E. Conditions contributing to fracture of the femoral neck. *Acta Chirurgica Scandinavica* 136:383-384, 1970.
- Noble, E.P.; Kakihana, R.; and Butte, J.C. Corticosterone metabolism in alcohol-adapted mice. In: Roach, M.K.; McLissac, W.M.; and Creaven, P.J., eds. *Biological Aspects of Alcohol*. Advances in Mental Science III. Dallas: University of Texas Press, 1971. pp. 389-417.
- Olive, J.; Gordon, G.G.; Rafii, F.; and Southren, A.L. Estrogen metabolism in hyperthyroidism and in cirrhosis of the liver. *Steroids* 26:47-56, 1975.
- Ono, J.; Hutson, D.G.; and Dombro, R.S. Tryptophan and hepatic coma. *Gastroenterology* 74:196-200, 1978.
- Orrego, H.; Kalant, H.; Israel, Y.; Blake, J.; Medline, A.; Rankin, J.G.; Armstrong, M.; and Kapur, B. Effect of short-term therapy with propylthiouracil in patients with alcoholic liver disease. *Gastroenterology* 76:105-115, 1979.
- Oxenkrug, G.F. Dexamethasone test in alcoholics (letter). *Lancet* 2:795, 1978.
- Paronetto, F., and Lieber, C.S. Cytotoxicity of lymphocytes in experimental alcoholic liver injury in the baboon. *Proceedings of the Society for Experimental Biology and Medicine* 153:495-497, 1976.
- Pekkanen, L.; Eriksson, K.; and Shvonen, M.-L. Dietarily-induced changes in voluntary ethanol consumption and ethanol metabolism in the rat. *British Journal of Nutrition* 40:103-113, 1978.
- Peng, T.C.; Cooper, G.W.; and Munson, P.L. The hypocalcemic effect of ethyl alcohol in rats and dogs. *Endocrinology* 91:586-593, 1972.
- Peng, T.C., and Gitelman, H.J.L. Ethanol-induced hypocalcemia, hypermagnesemia and inhibition of the serum calcium-raising effect of parathyroid hormone in rats. *Endocrinology* 94:608-611, 1974.
- Perin, A., and Sessa, A. In vitro effects of ethanol and acetaldehyde on tissue protein synthesis. In: Lindros, K.O., and Eriksson, C.J.P., eds. *The Role of Acetaldehyde in the Actions of Ethanol*. Helsinki: The Finnish Foundation for Alcohol Studies, 1975. pp. 105-122.
- Perlow, W.; Baraona, E.; and Lieber, C.S. Symptomatic intestinal disaccharidase deficiency in alcoholics. *Gastroenterology* 68:935-942, 1975.
- Phaosawasdi, K.; Tolin, R.; Mayer, E.; and Fisher, R.S. Effects of alcohol on the pyloric sphincter. *Digestive Diseases and Sciences* 24:934-939, 1979.
- Phillips, G.B., and Safrit, H.F. Alcoholic diabetes: Induction of glucose intolerance with alcohol. *Journal of the American Medical Association* 217:1513-1519, 1971.
- Phillips, R.L. Cancer among Seventh-Day Adventists. *Journal of Environmental Pathology and Toxicology* 3:157-169, 1980.
- Pickrell, K.L. The effect of alcoholic intoxication and ether anaesthesia on resistance to pneumococcal infection. *Bulletin of the Johns Hopkins Hospital* 63:238-260, 1938.
- Pirola, R.C., and Davis, A.E. Effect of intravenous alcohol on motility of the duodenum and of the sphincter of Oddi. *Australian Annals of Medicine* 19:24-29, 1970.
- Pirola, R.C., and Lieber, C.S. The energy cost of the metabolism of drugs, including ethanol. *Pharmacology* 7:185-196, 1972.
- Pohorecky, L.A. Effects of ethanol on central and peripheral noradrenergic neurons. *Journal of Pharmacology and Experimental Therapeutics* 189:380-391, 1974.
- Popper, H., and Lieber, C.S. Histogenesis of alcoholic fibrosis and cirrhosis in the baboon. *American Journal of Pathology* 98:695-716, 1980.
- Rachamin, G.; MacDonald, J.A.; Wahid, S.; Clapp, J.J.; Khanna, J.M.; and Israel, Y. Modulation of alcohol dehydrogenase and ethanol metabolism by sex hormones in the spontaneously hypertensive rat. *Biochemical Journal* 186:483-490, 1980.
- Raiha, N. Effect of ethanol on cytological changes induced by salt load in nucleus supraopticus of rat. *Proceedings of the Society for Experimental Biology and Medicine* 103:387-391, 1960.
- Rankin, J.G.; Hale, G.S.; Wilkinson, P.; O'Day, D.M.; Santamaria, J.N.; and Babarczy, G. Relationship between smoking and pulmonary disease in alcoholism. *Medical Journal of Australia* 1:730-733, 1969.
- Rasmussen, H. Parathyroid hormone, calcitonin, and the calciferols. In: Williams, R.H., ed. *Textbook of Endocrinology*, 5th ed. Philadelphia: W.B. Saunders Co., 1974. p. 680-773.
- Reber, H.A. Chronic pancreatitis. In: Sleisenger,

- M.H., and Fordtran, J.S., eds. Gastrointestinal Disease: Pathophysiology, Diagnosis, Management. Philadelphia: W.B. Saunders Co., 1978. pp. 1439-1456.
- Redmond, G.P. Effect of ethanol on endogenous rhythms of growth hormone secretion. Alcoholism: Clinical and Experimental Research 4:50-56, 1980.
- Regan, T.J.; Levinson, G.E.; Oldewurtel, H.A.; Frank, M.J.; Weisse, A.B.; and Moschos, C.B. Ventricular function in noncardiacs with alcoholic fatty liver: Role of ethanol in the production of cardiomyopathy. Journal of Clinical Investigation 48:397-407, 1969.
- Robert, A.; Nezamis, J.E.; Hanchar, A.J.; and Lancaster, C. Aspirin combined with alcohol is ulcerogenic (abstract). Gastroenterology 78(5, part 2):1245, 1980.
- Rodgers, J.B., and O'Brien, R.J. The effect of acute ethanol treatment on lipid-reesterifying enzymes of the rat small bowel. American Journal of Digestive Diseases 20(4):354-358, 1975.
- Rojkind, M., and DeLeon, L.D. Collagen biosynthesis in cirrhotic rat liver slices: A regulatory mechanism. Biochimica et Biophysica Acta 217: 512-522, 1970.
- Rosen, H.M.; Yoshimura, N.; Hodgman, J.M.; and Fischer, J.E. Plasma amino acid patterns in hepatic encephalopathy of differing etiology. Gastroenterology 72:483-487, 1977.
- Rosenberg, L.; Slone, D.; Shapiro, S.; Kaufman, D.W.; Helmrich, S.P.; Miettinen, O.S.; Stolley, P.D.; Levy, M.; Rosenshein, N.B.; Schottenfeld, D.; and Engle, R.L., Jr. Breast cancer and alcoholic-beverage consumption. Lancet 1:267-271, 1982.
- Rothman, K.J. The proportion of cancer attributable to alcohol consumption. Preventive Medicine 9:174-179, 1980.
- Rothman, K., and Keller, A. The effect of joint exposure to alcohol and tobacco on risk of cancer of the mouth and pharynx. Journal of Chronic Diseases 25:711-716, 1972.
- Rubin, E.; Rybak, B.J.; Lindenbaum, J.; Gerson, C.D.; Walker, G.; and Lieber, C.S. Ultrastructural changes in the small intestine induced by ethanol. Gastroenterology 63:801-814, 1972.
- Rudman, D.; Sewell, D.W.; and Ansley, J.D. Deficiency of carnitine in cachectic cirrhotic patients. Journal of Clinical Investigation 60:716-723, 1977.
- Salaspuro, M., and Lieber, C.S. Non-uniformity of blood ethanol elimination: Its exaggeration after chronic consumption. Annals of Clinical Research 10:294-297, 1978.
- Sanchez-Calvo, R. Die Alkoholvergiftung und ihre Folgen fuer das endokrine und das Ernahrungssystem [Alcohol intoxication and its consequences for the endocrine and nutrition system]. Virchows Archiv 308:14-21, 1941.
- Sarles, H. Chronic calcifying pancreatitis-chronic alcoholic pancreatitis. Gastroenterology 66:604-616, 1974.
- Sarles, H. Alcohol and the pancreas. In: Seixas, F.A.; Williams, K.; and Eggleston, S., eds. Medical Consequences of Alcoholism. Annals of the New York Academy of Sciences, Vol. 252, 1975. pp. 171-182.
- Saville, P.D., and Lieber, C.S. Effect of alcohol on growth, bone density and muscle magnesium in the rat. Journal of Nutrition 87:477-484, 1965.
- Schottenfeld, D. Alcohol as a co-factor in the etiology of cancer. Cancer 43:1962-1966, 1979.
- Schreiber, S.S.; Briden, K.; Oratz, M.; and Rothschild, M.A. Ethanol, acetaldehyde, and myocardial protein synthesis. Journal of Clinical Investigation 51:2808-2826, 1972.
- Schreiber, S.S.; Oratz, M.; and Rothschild, M.A. Alcoholic cardiomyopathy. II. The inhibition of cardiac microsomal protein synthesis by acetaldehyde. Journal of Molecular and Cellular Cardiology 6:207-213, 1974.
- Seitz, H.K.; Garro, A.J.; and Lieber, C.S. Enhanced pulmonary and intestinal activation of procarcinogens and mutagens after chronic ethanol consumption in the rat. European Journal of Clinical Investigation 11:33-38, 1981a.
- Seitz, H.K.; Garro, A.J.; and Lieber, C.S. Sex dependent effect of chronic ethanol consumption in rats on hepatic microsome mediated mutagenicity of benzo(a)pyrene. Cancer Letters 13:97-102, 1981b.
- Shah, J.H.; Wongsurawat, N.; and Aran, P.P. Effect of ethanol on stimulus-induced insulin secretion and glucose tolerance: A study of mechanisms. Diabetes 26:271-277, 1977.
- Shaw, S., and Lieber, C.S. Plasma amino acid abnormalities in alcoholics. Gastroenterology 74: 677-682, 1978.
- Shaw, S.; Stimmel, B.; and Lieber, C.S. Plasma alpha amino-n-butyric acid to leucine ratio: An empirical biochemical marker of alcoholism. Science 194:1057-1058, 1976.
- Shirazi, S.S., and Platz, C.E. Effect of alcohol on canine esophageal mucosa. Journal of Surgical Research 25:373-379, 1978.
- Singh, S.P., and Patel, D.G. Effects of ethanol on carbohydrate metabolism. I. Influence on oral glucose tolerance test. Metabolism 25:239-243, 1976.
- Smith, J.J. The effect of alcohol on adrenal ascorbic acid and cholesterol of the rat (abstract). Journal of Clinical Endocrinology 11:792, 1951.
- Sorrell, M.F.; Nauss, J.M.; Donahue, T.M.; and Tuma, D.J. Effects of chronic ethanol administration on hepatic glycoprotein secretion in the rat. Gastroenterology 84:580-586, 1983.
- Southren, A.L.; Gordon, G.G.; Olivo, J.; Rafii, F.; and Rosenthal, W.S. Androgen metabolism in cirrhosis of the liver. Metabolism Clinical and Experimental 22:695-702, 1973.
- Spann, J.F., Jr.; Mason, D.T.; Beiser, G.D.; and Gold, H.K. Actions of ethanol on the contractile state of the normal and failing cat papillary muscle (abstract). Clinical Research 16:249, 1968.
- Spiegelholder, B.; Eisenbrand, G.; and Preussmann, H.

- R. Contamination of beer with trace quantities of N-nitrosodimethylamine. Food and Cosmetics Toxicology 17:29-31, 1979.
- Sporn, M.B.; Dunlop, N.M.; Newton, D.L.; and Smith, J.M. Prevention of chemical carcinogenesis by vitamin A and its synthetic analogs (retinoids). Federation Proceedings 35(6):1332-1338, 1976.
- Stokes, P.E. Adrenocortical activity in alcoholics during chronic drinking. In: Seixas, F.A., and Eggleston, S., eds. Alcoholism and the Central Nervous System. Annals of the New York Academy of Sciences, Vol. 215, 1973. pp. 77-83.
- Strum, W.B., and Spiro, H.M. Chronic pancreatitis. Annals of Internal Medicine 74:264-277, 1971.
- Sullivan, J.F. Effect of alcohol on urinary zinc excretion. Quarterly Journal of Studies on Alcohol 23:216-220, 1962.
- Sullivan, L.W., and Herbert, V. Suppression of hematopoiesis by ethanol. Journal of Clinical Investigation 43:2048-2062, 1964.
- Swartz, M.H.; Repke, D.I.; Katz, A.M.; and Rubin, E. Effects of ethanol on calcium binding and calcium uptake by cardiac microsomes. Biochemical Pharmacology 23:2369-2376, 1974.
- Tabakoff, B.; Jaffe, R.C.; and Ritzmann, R.F. Corticosterone concentrations in mice during ethanol drinking and withdrawal. Journal of Pharmacy and Pharmacology 30: 371-374, 1978.
- Tapper, E.J.; Bushi, S.; Ruppert, R.D.; and Greenberger, N.J. Effect of acute and chronic ethanol treatment on the absorption of iron in rats. American Journal of the Medical Sciences 255: 46-52, 1968.
- Thomson, A.D.; Baker, H.; and Leevy, C.M. Patterns of ³⁵S-thiamine hydrochloride absorption in the malnourished alcoholic patient. Journal of Laboratory and Clinical Medicine 76:34-45, 1970.
- Torro, G.; Kolodny, R.C.; Jacobs, L.S.; and Masters, W.H. Failure of alcohol to alter pituitary and target organ hormone levels (abstract). Clinical Research 21:505, 1973.
- Tuyns, A.J. Alcohol and cancer. Alcohol Health and Research World 2:20-31, 1978.
- Tuyns, A.J. Epidemiology of alcohol and cancer. Cancer Research 39:2840-2843, 1979.
- Tuyns, A.J. Alcohol. In: Schottenfeld, D., and Fraumeni, J.F., Jr., eds. Cancer Epidemiology and Prevention. Philadelphia: W.B. Saunders Co., 1982. pp. 293-303.
- Unger, K.W., and Johnson, D. Red blood cell mean corpuscular volume: A potential indicator of alcohol usage in a working population. American Journal of the Medical Sciences 267:281-289, 1974.
- Van Thiel, D.H. Alcohol and its effect upon endocrine function. Alcoholism: Clinical and Experimental Research 4:44-49, 1980.
- Van Thiel, D.H. Adrenal response to ethanol: A stress response? In: Pohorecky, L.A., and Brick, J., eds. Stress and Alcohol Use. New York: Elsevier Biomedical, 1983a. pp. 23-27.
- Van Thiel, D.H. Ethanol: Its adverse effects upon the hypothalamic-pituitary-gonadal axis. Journal of Laboratory and Clinical Medicine 101: 21-33, 1983b.
- Van Thiel, D.H., and Lester, R. Alcoholism: Its effect on hypothalamic-pituitary-gonadal function. Gastroenterology 71:318-327, 1976a.
- Van Thiel, D.H., and Lester, R. Sex and alcohol: A second peek (editorial). New England Journal of Medicine 295:835-836, 1976b.
- Van Thiel, D.H., and Lester, R. The effect of chronic alcohol abuse on sexual function. Clinics in Endocrinology and Metabolism 8:499-510, 1979a.
- Van Thiel, D.H., and Lester, R. Hypothalamic-pituitary-gonadal dysfunction in patients with alcoholic liver disease. In: Davidson, C.S., ed. Problems in Liver Disease. New York: Stratton Intercontinental Medical Book Corp., 1979b. pp.286-298.
- Van Thiel, D.H.; Gavalier, J.S.; and Lester, R. Ethanol inhibition of vitamin A metabolism in the testes: Possible mechanism for sterility in alcoholics. Science 186:941-942, 1974a.
- Van Thiel, D.H.; Lester, R.; and Sherins, R.J. Hypogonadism in alcoholic liver disease: Evidence for a double defect. Gastroenterology 67:1188-1199, 1974b.
- Van Thiel, D.H.; Gavalier, J.S.; Lester, R.; and Goodman, M.D. Alcohol-induced testicular atrophy: An experimental model for hypogonadism occurring in chronic alcoholic men. Gastroenterology 69:326-332, 1975a.
- Van Thiel, D.H.; Gavalier, J.S.; Lester, R.; Loriaux, D.L.; and Braunstein, G.D. Plasma estrone, prolactin, neurophysin and sex steroid-binding globulin in chronic alcoholic men. Metabolism 24: 1015-1019, 1975b.
- Van Thiel, D.H.; Gavalier, J.S.; Little, J.M.; and Lester, R. Alcohol: Its effect on the kidney. Metabolism 26:857-866, 1977.
- Van Thiel, D.H.; Gavalier, J.S.; Lester, R.; and Sherins, R.J. Alcohol-induced ovarian failure in the rat. Journal of Clinical Investigation 61: 624-632, 1978a.
- Van Thiel, D.H.; Gavalier, J.S.; Wight, C.; Smith, W.I.; and Abuid, J. Thyrotropin-releasing hormone (TRH)-induced growth hormone (hGH) responses in cirrhotic men. Gastroenterology 75: 66-70, 1978b.
- Van Thiel, D.H.; Lester, R.; and Vaitukaitis, J. Evidence for a defect in pituitary secretion of luteinizing hormone in chronic alcoholic men. Journal of Clinical Endocrinology and Metabolism 47:499-507, 1978c.
- Van Thiel, D.H.; McClain, C.J.; Elson, M.K.; and McMillin, M.J. Hyperprolactinemia and thyrotropin-releasing factor responses in men with alcoholic liver disease. Alcoholism: Clinical and Experimental Research 2:344-348, 1978d.
- Van Thiel, D.H.; Gavalier, J.S.; Cobb, C.F.; Sherins, R.J.; and Lester, R. Alcohol induced testicular atrophy in the adult male rat. Endocrinology 105:888-895, 1979a.
- Van Thiel, D.H.; Smith, W.I.; Wight, C.; and Abuid, J. Elevated basal and abnormal thyrotropin-

- releasing hormone-induced thyroid-stimulating hormone secretion in chronic alcoholic men with liver disease. Alcoholism: Clinical and Experimental Research 3:302-308, 1979b.
- Van Thiel, D.H.; Gavalier, J.S.; Slone, F.L.; Cobb, C.F.; Smith, W.L.; Klaus, M.B.; and Lester, R. Is feminization in alcoholic men due in part to portal hypertension: A rat model. Gastroenterology 78:81-91, 1980.
- Veitch, R.L.; Lumeng, L.; and Li, T.-K. Vitamin B₆ metabolism in chronic alcohol abuse: The effect of ethanol oxidation on hepatic pyridoxal 5'-phosphate metabolism. Journal of Clinical Investigation 55:1026-1032, 1975.
- Verdy, M., and Caron, D. Ethanol et absorption du calcium chez l'humain [Ethanol and calcium absorption in man]. Biologie et Gastroenterologie 6:157-160, 1973.
- Wagner, G., and Fuchs, A.-R. Effects of ethanol on uterine activity during suckling in post-partum women. Acta Endocrinologica 58:133-141, 1968.
- Walsh, M.J., and Truitt, E.B. Acetaldehyde mediation in the mechanism of ethanol-induced changes in norepinephrine metabolism. Molecular Pharmacology 6:416-424, 1970.
- Wang, J., and Pierson, R.N. Distribution of zinc in skeletal muscle and liver tissue in normal and dietary controlled alcoholic rats. Journal of Laboratory and Clinical Medicine 85:50-58, 1975.
- Wehman, H.J., and Plantholt, B.A. Asbestos fibrils in beverages. I. Gin. Bulletin of Environmental Contamination and Toxicology 11:267-272, 1974.
- Weinstein, M.J., and Deykin, D. Quantitative abnormality of an alpha-chain molecular weight form in the fibrinogen of cirrhotic patients. British Journal of Haematology 40:617-630, 1978.
- Whitehead, T.P.; Clarke, C.A.; and Whitfield, A.G.W. Biochemical and haematological markers of alcohol intake. Lancet 1:978-981, 1978.
- Wilkinson, P.; Santamaria, J.N.; and Rankin, J.G. Epidemiology of alcoholic cirrhosis. Australian Annals of Medicine 18:222-226, 1969.
- Williams, G.A.; Bowser, E.N.; Hargis, G.K.; Kukreja, S.C.; Shah, J.H.; Vora, N.M.; and Henderson, W.J. Effect of ethanol on parathyroid hormone and calcitonin secretion in man. Proceedings of the Society for Experimental Biology and Medicine 159:187-191, 1978.
- Williams, J.W.; Tada, M.; Katz, A.M.; and Rubin, E. Effects of ethanol and acetaldehyde on the (Na⁺ + K⁺)-activated adenosine triphosphatase activity of cardiac plasma membranes. Biochemical Pharmacology 24:27-32, 1975.
- Williams, R.R., and Horm, J.W. Association of cancer sites with tobacco and alcohol consumption and socioeconomic status of patients: Interview study from the Third National Cancer Survey. Journal of the National Cancer Institute 58:525-547, 1977.
- Winship, D.H.; Caffisch, C.R.; Zboralske, F.F.; and Hogan, W.J. Deterioration of esophageal peristalsis in patients with alcoholic neuropathy. Gastroenterology 55:173-178, 1968.
- Winsor, A.L., and Strongin, E.L. The effect of alcohol on the rate of parotid secretion. Journal of Experimental Psychology 16:589-597, 1933.
- Wright, J.; Merry, J.; Fry, D.; and Marks, V. Pituitary function in chronic alcoholism. In: Gross, M.M., ed. Alcohol Intoxication and Withdrawal. II. Advances in Experimental Medicine and Biology, Vol. 59, 1975. pp. 253-255.
- Wright, J.W. Endocrine effects of alcohol. In: Marks, V., and Wright, H., eds. Metabolic Effects of Alcohol. Clinics in Endocrinology and Metabolism, Vol. 7, No. 2, 1978. pp. 351-367.
- Wright, S.G., and Ree, G.H. Blood in the alcohol stream (letter). Lancet 1:49, 1978.
- Wynder, E.L. Toward the prevention of laryngeal cancer. Laryngoscope 85:1190-1196, 1975.
- Wynder, E.L., and Chan, P.C. The possible role of riboflavin deficiency in epithelial neoplasia. II. Effect on skin tumor development. Cancer 26:1221-1224, 1970.
- Yadav, H.S.; Chaudhuri, B.N.; and Mukherjee, S.K. Effect of ethyl alcohol on thyroidal iodide trapping and renal clearance of ¹³¹I label in rats. Indian Journal of Medical Research 58:1421-1427, 1970.
- Ylikahri, R.H.; Huttunen, M.O.; and Harkonen, M. Effect of alcohol on anterior-pituitary secretion of trophic hormones. Lancet 1:1353, 1976.
- Ylikahri, R.; Huttunen, M.; Harkonen, M.; Seuderling, U.; Onikki, S.; Karonen, S.-L.; and Adlercreutz, H. Low plasma testosterone values in men during hangover. Journal of Steroid Biochemistry 5:655-658, 1974.
- Young, G.P.; Dudley, F.J.; and Van-der-Weyden, M.B. Suppressive effect of alcoholic liver disease sera on lymphocyte transformation. Gut 20:833-839, 1979.
- Zeus, J. Histologische Untersuchungen und distalen Oesophagus zur Pathogenese der Oesophagitis unter besonderer Beruecksichtigung der Beziehung zum Sodbrennen [Histological research on the distal esophagus and pathogenesis of esophagitis, with special consideration of its relationship to heartburn]. Dissertation Erlangen. 1970.

Chapter V

The Effects of Alcohol on Pregnancy Outcome

Clinical reports and experimental studies published in the past 10 years leave little doubt that prenatal alcohol exposure poses a threat to the health of the unborn child. This threat ranges from miscarriage and newborn death, to a cluster of abnormalities called fetal alcohol syndrome (FAS), to subtle behavioral disturbances such as hyperactivity, which may occur in the absence of observable physical abnormalities.

This chapter represents a selective review and status report on current knowledge concerning the effects of alcohol on pregnancy outcome. It is not intended to be exhaustive. A number of recent more detailed reviews have dealt with a full range of issues involving alcohol-related pregnancy complications and birth defects (see, for example, Rosett and Weiner 1981; Rosett et al. 1981; Sokol 1981, 1982; Abel 1982a; Randall 1982). In addition, overviews of fetal alcohol research are contained in the Third and Fourth Special Reports to the U.S. Congress on Alcohol and Health (USDHEW 1978; USDHHS 1981).

The present survey examines current findings about alcohol and pregnancy and needs for future research. The first section begins with a brief historical introduction. This is followed by an examination of data documenting alcohol as a teratogen (a substance that can cause birth defects). Some specific alcohol-related pregnancy complications and birth defects are then examined, with particular emphasis on new information about brain damage and behavioral disorders. The need for further information about time of exposure, patterns of drinking, type of beverage, determination of dose/response relations and thresholds, and host susceptibility are discussed in conjunction with ways of improving pregnancy outcomes by preventing alcohol-related birth defects.

It should be noted that this chapter uses, where appropriate, the terms alcohol-related birth effects as well as alcohol-related birth defects. Birth defects include entities such as organ anomalies. The term birth effects, as

used in this chapter, is more inclusive and contains, in addition, pregnancy complications and outcomes such as small-for-gestation-age weight. These factors usually place the infant at higher risk for an adverse health occurrence, but are not in their own right anomalies.

Historical Perspective

Although even during ancient times alcohol may have been considered a pregnancy risk, by the 1700s a link between alcohol and birth defects was strongly suspected. In 1759, for example, the London College of Physicians petitioned the British Parliament to reinstate taxes on gin so that it would be less available and, therefore, would constitute less of a risk to pregnant women and their infants (Warner and Rosett 1975). Despite reinstatement of these taxes, a steady outpouring of warnings about drinking during pregnancy continued, indicating that the problem did not disappear after the law was changed.

In 1899, a report appeared in England which documented in a novel way many of the adverse effects of alcohol during pregnancy (Sullivan 1899). Six hundred offspring of 120 alcoholic women in a Liverpool jail were compared with offspring born to 28 nondrinking female relatives of these women. Infant mortality and stillbirth rates for the alcoholics' children were 2-1/2 times higher than the rates among offspring of the comparison group. Even more dramatic was the finding that many of those alcoholic women who previously had infants with severe and often fatal complications gave birth to healthy children when forced to become abstinent because of imprisonment. The obvious inference drawn by the author was that alcohol is toxic to the fetus.

Although several relevant studies pointing to fetal damage from prenatal alcohol exposure were performed during the early 20th

century, interest in alcohol and pregnancy notably decreased during Prohibition, and only sporadic reports can be found in the world literature from the succeeding 50 years (Sokol 1982; Abel in press). Modern recognition awaited the publication of two key papers in 1973 by Jones, Smith, and their colleagues. These investigators reported that 11 children of chronically alcoholic mothers were growth-retarded and had craniofacial and cardiac defects, as well as developmental delay (Jones and Smith 1973; Jones et al. 1973). Perhaps, in retrospect, the major contribution of these papers was the coining of the term fetal alcohol syndrome, which refocused interest on important scientific and public policy questions. These reports gave particular impetus because they appeared during a period of increased public and professional concern with maternal/fetal/infant health. If alcohol consumption represented an important and potentially avoidable pregnancy risk, perhaps pregnancy outcomes could be improved when the risk had been recognized.

Fetal Alcohol Syndrome and Alcohol-Related Birth Effects

Numerous cases of fetal alcohol syndrome from all over the world have now been described in the scientific literature, e.g., from Australia, Belgium, Brazil, Canada, Chile, Czechoslovakia, France, Germany, Hungary, Ireland, Italy, Japan, South Africa, Spain, Sweden, Switzerland, and the United States (Abel in press). Because standardized criteria were not used for many of the diagnoses, the Fetal Alcohol Study Group of the Research Society on Alcoholism proposed specific criteria in 1980 (Rosett 1980). These criteria require that at least one feature from each of the following three categories be present for a diagnosis of fetal alcohol syndrome:

- Growth retardation before and/or after birth
- A pattern of abnormal features of the face and head, including small head circumference, small eyes, or evidence of retarded formation of the midfacial area, including a flattened bridge and short length of the nose, and flattening of the vertical groove between the nose and mouth, i.e., the philtrum

- Evidence of central nervous system abnormality, including, for example, abnormal neonatal behavior, mental retardation, or other evidence of abnormal neurobehavioral development

None of these features alone is specifically characteristic of fetal exposure to alcohol. In fact, many other nonspecific abnormalities are seen in conjunction with FAS, as well as in isolation, which makes diagnosis difficult. These include eye and ear defects; heart murmurs, which are often associated with septal defects (openings between the chambers of the heart); genitourinary anomalies, such as undescended testicles; hemangiomas (a type of birthmark); fingerprint and palmar crease abnormalities; and other anomalies, such as hernias. Any of these abnormalities may be seen individually, even without maternal drinking. However, when these conditions are seen in conjunction with a pattern of abnormalities consistent with FAS, the diagnosis of FAS may be made. Alcohol-related birth effects are complications of pregnancy, risk associated pregnancy outcomes (e.g., small-for-gestation-age weight), and birth defects that can be attributed to alcohol use after statistical analysis has corrected for the contribution of other possible factors.

Prevalence

The reported prevalence of FAS varies widely and no firm national data are as yet available. Depending on the location and the population under study, the overall prevalence has varied from 0.4 per 1,000 in Cleveland (Sokol et al. 1980) to 2.1 per 1,000 in Boston (Rosett et al. 1983). In Seattle, where the syndrome was first identified, one study indicated a prevalence of 1.3 per 1,000 (Hanson et al. 1978). Estimates from Europe have ranged from 1.6 per 1,000 in Sweden (Olegard et al. 1979) to 2.9 per 1,000 in France (Dehaene et al. 1977). It appears then that, calculated on the basis of total number of births, the overall prevalence of FAS is in the range of 1 to 3 per 1,000.

Estimates of the frequency of the full fetal alcohol syndrome, calculated on the basis of occurrence only among women who have been identified as problem drinkers or alcohol abusers, are somewhat more consistent and higher than the above figures, ranging from 23 to 29 per 1,000 (Hanson et al. 1978; Sokol et al. 1980; Rosett et al. 1983). Further, as might

be expected, alcohol-related birth effects have been reported considerably more frequently among alcohol abusers. However, the estimates vary widely, ranging from 78 per 1,000 to 690 per 1,000 (Ouellette et al. 1977; Hanson et al. 1978; Sokol et al. 1980). This wide range may be attributed to the differing criteria used for inclusion in this diagnostic category. Thus, although only a limited proportion of the offspring may have enough abnormalities to be diagnosed as FAS, some effects possibly attributable to alcohol appear to occur in relatively high proportions of infants born to women who drink heavily during pregnancy.

In general, about 65 to 70 percent of developmental defects have been noted to be of unknown origin (Wilson 1973). Sokol (1981) has calculated that about 5 percent of all congenital anomalies may be attributable to prenatal alcohol exposure, suggesting that alcohol may account for a significant proportion of previously unexplained anomalies and that it should be considered a major contributor to abnormal fetal development. In terms of mental retardation, which may be considered the most serious and damaging of all alcohol-related birth defects, Clarren and Smith (1978) have stated that maternal alcohol abuse during pregnancy "appears to be the most frequent known teratogenic cause of mental retardation in the Western World."

The high prevalence of alcohol-related birth defects is worth noting not only because of its impact on the affected individual and on society, but also because of its significant economic impact. While difficult to evaluate precisely, one study has estimated a lifetime cost of \$155 million for those born in a given year with FAS and alcohol-related birth defects in New York State alone (Russell 1980).

Problems with Human Fetal Alcohol Studies

The experimental administration of alcohol to pregnant women is clearly unethical. Studies of alcohol and pregnancy in humans are limited by virtue of the usual constraints associated with human subjects to clinical observation and nonmanipulative designs. Clinical reports have been of particular importance in special cases, such as in the birth of twins. For example, a case in which one fraternal twin was more severely affected with FAS than the other (Christoffel and Salafsky 1975) suggests that genetic factors are significant in determining fetal susceptibility to

alcohol's damaging effects. This may partially explain why only one of two women who consume the same amount of alcohol may give birth to a child with FAS.

Observational studies involving many patients rather than single cases, however, can be more difficult to interpret. Recent detailed critical reviews of such studies (Neugut 1981; Sokol 1980b) have identified two general problem areas, the first involving the issue of bias. In many published case reports and studies, the clinician diagnosing alcohol-related birth defects may have been aware of maternal alcohol abuse, leading to the possibility that some of the observed associations may have been affected by such knowledge. However, even when infants have been examined without knowledge of maternal alcohol history, as in the large prospective observational studies funded by the National Institute on Alcohol Abuse and Alcoholism (NIAAA), alcohol-related pregnancy complications and birth effects, including lowered birth weight and congenital abnormalities, have been identified (Ouellette et al. 1977; Kuzma and Sokol 1982).

A second major problem in human fetal alcohol studies is called "confounding." Alcohol is but one of a multitude of possible pregnancy risks (cofactors) which include maternal characteristics, medical disorders, pregnancy complications, abuse of substances other than alcohol, and exposure to environmental contaminants. In observational study designs, complex statistical techniques are used to try to control and adjust for as many factors as possible to support the inference that any observed effect is attributable to alcohol. Many pregnancy risks remain unknown, however, and these statistical techniques are limited, making it almost impossible to adjust for confounding completely. While human studies can document associations between alcohol and adverse pregnancy outcome, they cannot demonstrate causality convincingly (Sokol 1980b). Experimental studies in animals have therefore proven to be valuable. If rigorously designed and performed, animal studies allow greater control and greater certainty in inferring a causative role for alcohol as a teratogen.

Animal Models

Alcohol-related birth defects comparable to those occurring in humans have been reported among mice (Randall and Taylor 1979), rats

(Abel 1979), beagles (Ellis and Pick 1980), sheep (Potter et al. 1980), miniature swine (Dexter et al. 1980), and monkeys (Altshuler and Shippenberg 1981). These studies in animals have documented direct dose/response effects of alcohol on perinatal mortality, infant weight, and soft tissue malformation and have shown that these effects cannot be attributed to other factors, such as undernutrition, often associated with alcohol abuse in humans. For example, Randall and her colleagues (1981), using a study design that equates the nutritional intake of alcohol-exposed and control animals, clearly demonstrated in mice that restricted caloric intake could not account for the teratogenic effects observed. However, nutritional factors, e.g., low protein intake, can interact with alcohol exposure, leading to higher blood alcohol levels and more severe alcohol-related birth defects, as suggested by the work of Wiener and her colleagues (1981). These findings indicate that nutritional factors alone did not account for the observed deficits. The alcohol present in the body rather than nutritional factors is the teratogen.

Other compelling evidence for a direct effect of alcohol as a teratogen comes from in vitro (literally in glass) studies of embryonic tissue. In these experiments, rat embryos have been directly exposed to alcohol (Brown et al. 1979). Compared with unexposed controls, embryos exposed to 0.15 or 0.3 grams of alcohol per 100 ml of culture medium (an intoxicating dose) had decreased crown-rump and head lengths and decreased total cell counts, as well as retarded development, after only 24 hours of exposure. The rat embryo at this stage cannot metabolize alcohol to its primary metabolite, acetaldehyde; thus, this study strongly supports a causative role for alcohol as an agent directly toxic to the fetus but does not rule out the possibility that acetaldehyde also may be a teratogen (Popov et al. 1981).

Additional support for a direct effect of alcohol in inducing aberrant fetal development is provided by a notable study performed in mice. Sulik and her colleagues (1981) administered early in pregnancy two large closely spaced doses of alcohol on a single day, such as might occur in binge drinking. They were able to produce abnormal facial features in the mouse fetus that appear remarkably similar to those observed in human FAS. However, certain methodological issues raise questions as to the immediate applicability of such findings to humans. Nonetheless, this study

both supports the concept of a causative role for alcohol in inducing fetal malformation and raises questions about the critical periods of exposure for specific effects and the ability of fetal tissues to recover from exposure to toxic levels of alcohol early in pregnancy.

Specific Adverse Pregnancy Outcomes

Exposure to intoxicating levels of alcohol over prolonged periods is clearly associated with a range of specific adverse fetal outcomes. Some of these outcomes have been described in detail in the Third and Fourth Special Reports to the U.S. Congress on Alcohol and Health (USDHEW 1978; USDHHS 1981) and therefore will be reviewed only briefly.

Spontaneous Abortion

The risk for spontaneous abortion (miscarriage or early pregnancy loss) is increased approximately twofold in pregnancies complicated by maternal drinking, although perhaps only among the heaviest or most frequent drinking 3 to 5 percent of women (Harlap and Shiono 1980; Kline et al. 1980; Sokol 1980a; Sokol et al. 1980). Pregnant monkeys also tend to abort early in pregnancy following alcohol exposure (Altshuler and Shippenberg 1981), as do rodents, as indicated by an increased rate of resorption, the analog of spontaneous abortion in humans (Chernoff 1977; Randall et al. 1981). With respect to later pregnancy loss, i.e., the stillbirth rate, the data are less clear. A statistically significant increase in the stillbirth rate was detected in an initial study from France for women consuming three or more drinks per day (Kaminski et al. 1978), but not in a subsequent study by this group (Kaminski et al. 1981). Also, no increase in the stillbirth rate was noted in a large study from the Cleveland area (Sokol et al. 1980). Studies in animals are likewise equivocal with respect to alcohol and stillbirths (Ellis and Pick 1980; Abel et al. 1981b).

Physical Congenital Anomalies

Virtually all of the congenital anomalies reported to be alcohol-related birth defects in humans have now been duplicated in animals prenatally exposed to alcohol, although probably only when blood alcohol levels exceeded 0.1 percent--the legal level of intoxication in most States (Randall 1982). Acute intragastric

administration (directly into the stomach) of alcohol to mice on one day of pregnancy did not result in a significant increase in birth defects, compared with two nutritional control groups (Lochry et al. 1982). Thus, it appears that duration as well as level of exposure are important determinants of alcohol-related physical birth defects.

Lowered Birth Weight

Lowered birth weight is the most reliably observed effect attributable to in utero alcohol exposure in humans and animals. Based on a review of more than 300 reported cases of FAS, the average birth weight of such children was 2,000 grams (Abel 1982b), compared with the median birth weight for all infants in the United States of more than 3,300 grams (NCHS 1980). Further, decreased birth weight has been observed even in the absence of full FAS (Ouellette et al. 1977; Sokol et al. 1980). These findings are of concern because lowered birth weight is widely recognized as being associated with significant increases in risk for fetal and infant mortality and long-term abnormalities in neurologic development and intelligence. Lowered birth weight may be the result of preterm delivery (prematurity) or intrauterine growth retardation, or both.

Preterm delivery.--Effects of alcohol on pregnancy duration are unclear. Alcohol-related prematurity was not identified in one study of more than 12,000 pregnancies (Sokol et al. 1980). Two more recent studies, however, suggest that preterm delivery may result from prenatal alcohol exposure. Berkowitz (1981), in a study of 175 mothers of preterm infants and 313 mothers of term infants, found that alcohol consumption prior to the third trimester of pregnancy was associated with preterm delivery. Furthermore, in a more detailed analysis of the same data, Berkowitz and her coworkers (1982) found that heavier alcohol consumption (an average of two or more drinks every day) during pregnancy was associated with an approximately threefold increase in risk for preterm delivery; no increased risk was identified for lighter drinking.

In a recent study from Boston City Hospital of 1,690 births, maternal drinking prior to pregnancy was found to be associated with shortened gestation (Hingson et al. 1982). It is interesting to note that preterm delivery does not occur in pregnant animals that are given alcohol—in fact, parturition may be delayed (Bond 1982).

Intrauterine growth retardation.--In contrast to the data involving prematurity, the evidence linking in utero alcohol exposure and decreased fetal growth is very strong. For example, in their study of more than 12,000 pregnancies, Sokol and his colleagues (1980) found that with alcohol abuse birth weight was decreased by about 190 grams, in the absence of any effect on pregnancy duration. Similarly, in another group of approximately 5,000 women in a different part of the country, Kuzma and Sokol (1982) detected about a 100-gram decrement in birth weight, limited to the most frequently drinking 3 percent of women. Little (1977) noted a statistically significant decrease in infant birth weight of 91 grams for women reporting average alcohol use of 1 ounce (two drinks) per day for the prepregnancy period, and a 160-gram decrement for late pregnancy consumption at that level.

Prenatal growth retardation associated with maternal alcohol consumption has been observed in numerous animal studies (Ellis and Pick 1980; Lee and Leichter 1980). The extent of this retardation appears to be dose-dependent with exposure during the third trimester having a more severe effect on birth weight than did exposure earlier in gestation (Abel 1979; Lochry et al. 1982). The decrease in birth weight can be seen even after a single administration of alcohol shortly before term (Greizerstein and Abel 1979; Lochry et al. 1982). These observations are consistent with studies in humans that indicate that drinking continued through the third trimester is associated with decreased infant weight (Little 1977; Rosett et al. 1978).

Studies in animals are also beginning to help identify mechanisms by which alcohol may decrease fetal growth. For example, animal embryos provide evidence for the direct effects of alcohol and acetaldehyde on fetal growth (Brown et al. 1979; Popov et al. 1981). In addition, alcohol exposure may lead to chronic fetal hypoxia (Abel 1982b), or to hypoglycemia, i.e., low blood glucose. Decreased fetal nutrition resulting from hypoglycemia induced by high blood alcohol levels could contribute to fetal growth retardation (Tanaka et al. 1982). Other studies suggest that alcohol may affect fetal cell growth by interfering with the passage of amino acids across the placenta (Henderson et al. 1981). With inadequate transport of amino acids to the fetus, protein synthesis and growth would be inhibited. Moreover, decreased incorporation of amino acids into protein in alcohol-exposed fetuses has also been reported (Henderson et

al. 1979, 1980). Although alcohol-related decreased absorption of zinc has also been suggested as a contributor to growth retardation, the evidence on this issue is contradictory (e.g., Samson and Diaz 1981; Flynn et al. 1981; Ghishan et al. 1982).

Based on these results, it is reasonable to conclude that alcohol is associated with lowered birth weight, related possibly to both shorter gestation and decreased intrauterine growth. Diminished prenatal growth is dose-related and may be more severe when alcohol is consumed in the third trimester.

Abnormal Neurobehavioral and Neural Development

In surviving fetuses, the most severe alcohol-related birth defects are those involving the central nervous system. Congenital anomalies, even those that are very severe, e.g., heart defects, are often repairable. Brain damage, however, is permanent and irreversible. Alcohol-related anatomical, electrophysiological, and biochemical abnormalities in the brain have now been identified and may underlie the observed behavioral deficits.

Behavioral development.--The adverse effects of alcohol on behavioral development may be divided into three main categories: abnormal neonatal behavior, as reflected in sleep problems and state regulation disorders; childhood hyperactivity; and mental retardation, as indicated by lowered IQ scores (Abel 1981a).

Infants born to heavy drinkers or alcoholics have been found to be more restless during sleep and not to sleep as much as other children (Rosett et al. 1979). Abnormal electroencephalographic (EEG) activity during sleep has been noted for as long as 6 weeks after birth in some of these children.

Hyperactivity in children with FAS has been noted in several clinical case studies (Abel 1981a). It has also been noted in 15 children who had physical abnormalities consistent with FAS, but whose IQ scores were within normal limits. All 15 of these children eventually received recommendation for special education services because of their restlessness, short attention spans, and distractibility (Shaywitz et al. 1980).

Lowered IQ scores in conjunction with heavy prenatal alcohol exposure have been noted in other countries. In the United States, the average IQ scores for a group of patients with FAS was found to be 65, reflecting moderate

mental retardation (Streissguth et al. 1978a), and when the same individuals were retested, the IQ scores did not change (Streissguth et al. 1978b). Similar results were obtained in the study from Sweden in which children with physical signs of FAS were found to have an average IQ below 70 (Olegard et al. 1979).

Two studies of mental and motor development identified infants exposed in utero to alcohol and investigated those who did not exhibit full FAS. Streissguth and her coworkers (1980) administered an infant development test to a group of infants at the age of 8 months. They recorded significant trends toward lower scores on mental and motor tests related to increased maternal alcohol use during pregnancy.

Golden and her colleagues (1982) evaluated neurobehavioral development in 12 infants with physical alcohol-related birth defects and a history of maternal alcohol abuse and compared them with 12 normal infants. These two groups of infants were examined at a mean age of 1 year without knowledge of the prenatal drinking history. Both the mental and motor scores were 20 points lower in the alcohol group, a notable difference. Furthermore, the alcohol group had significantly higher frequencies of postnatal growth retardation and microcephaly, i.e., abnormally small heads. Although it was not possible in this study to adjust for the confounding effects of maternal smoking, there was strong evidence that the abnormal neurobehavioral development of the infants was attributable to alcohol alone or in combination with cigarette smoking.

Landesman-Dwyer and her coworkers (1981) studied 272 offspring of women without alcohol problems and found that at age 4, the children with greater in utero alcohol exposure generally were less attentive and less compliant with parental commands. More recently, Streissguth and her colleagues (1983b), after observing 4-year-olds, found that maternal alcohol use during pregnancy is significantly related to decreased attention and longer reaction time. They suggested that alcohol use by pregnant women can have long-term behavioral effects in children.

As with physical anomalies, some studies in animals duplicate many of the behavioral abnormalities noted in humans with fetal alcohol exposure. For example, hyperactivity has been noted consistently in rats prenatally exposed to alcohol (Bond and DiGiusto 1976; Abel 1982c). Learning difficulties have also been identified in animals prenatally exposed to

alcohol (Bond and DiGiusto 1978; Riley et al. 1979a, b; Abel 1979). Riley and his coworkers (1979a, b) have proposed that many of the abnormal behavioral effects related to prenatal alcohol exposure suggest an underlying problem in response inhibition. The magnitude of this effect appears to be dose-related (Lochry and Riley 1980). On balance, the behavioral abnormalities found in animals are similar to those in children with fetal alcohol syndrome.

Neural development.—Neuroanatomic and biochemical abnormalities probably underlie alcohol-related abnormal neurobehavioral development; these areas are currently under study. Microcephaly, an important component in the diagnosis of FAS, indicates an overall decrease in brain growth. Moreover, anatomic abnormalities have been observed in brains of children born to alcoholic women as studied at autopsy, as well as in animals prenatally exposed to alcohol. These include hydrocephalus (increased fluid distending cavities in the brain), absence of the corpus collosum (the bundle of fibers connecting the two sides of the brain), and abnormal growth patterns of nerve and other brain cells (Clarren et al. 1978; Peiffer et al. 1979; Rasmussen and Christensen 1980).

Subtle changes in brain structure have been noted in animals following prenatal alcohol exposure. West et al. (1981) found that rats exposed to alcohol throughout gestation had abnormally distributed nerve fibers in the hippocampus (deep inside the brain), and Barnes and Walker (1981) identified a decrease in the cell content of rats' hippocampus. In addition, Davies and Smith (1981) identified a considerable decrease in the extent of the neural dendrites (parts of the nerve cell that receive signals) of the hippocampus in mice. Taken together, these anatomic abnormalities in the hippocampus may be the structural basis for some of the behavioral abnormalities observed in humans and animals, as this part of the brain is involved in learning/memory performance and in inhibitory control of behavior.

Alcohol Use, Abuse, and Dependence and Pregnancy

Drinking in Women

Approximately 60 percent of adult American women drink alcoholic beverages; 40 percent are abstinent. An estimated 6 percent of adult women in this country have been clas-

sified as problem drinkers (Clark and Midanik 1982). During the peak reproductive age range of 18-34, an estimated 5 percent of American women consume an average of 2 or more drinks per day or 14 or more drinks per week (Clark and Midanik 1982). Estimates of the proportion of women who drink at least this amount during pregnancy vary widely, ranging from a low of 0.5 percent in parts of California (Harlap and Shiono 1980) to a high of 16 percent in Buffalo (Russell and Bigler 1979), and a median of 2 percent (Abel in press). Because alcohol readily crosses the placenta, yielding levels of alcohol in the fetus almost identical to those in the mother, it is reasonable to conclude that a significant number of unborn children in the United States are exposed to the equivalent of two drinks a day.

Drinking Patterns in Pregnancy

The amount and timing of alcohol consumption that put the fetus at risk are still unknown. Scientists have considerable difficulty in addressing this issue because of problems in obtaining reliable drinking histories and in defining abusive drinking and dependence. Furthermore, drinking patterns often change during pregnancy.

Essentially all reported studies of drinking during pregnancy have relied on self-reports of patients. The inherent limitations of self-reports suggest that the alcohol intake information obtained must be understood in broad terms, rather than in precise measurements. The amount of alcohol actually imbibed in a drink might vary from 1 to 8 ounces, depending on the individual giving the history and the method by which the history is obtained (Rosett et al. 1983). A detailed recall of drinking behavior is likely to be inexact, particularly for the heavy or dependent drinker who may be unable or unwilling to recall specifically how much she drank. Self-reports of drinking during pregnancy are therefore of limited value. This in turn seriously limits the credibility of estimates relating a given number of drinks per day to a particular risk to the fetus. Moreover, it is likely that actual alcohol intake is underreported by the abusive drinker, the individual most likely to be at risk for giving birth to a child with alcohol-related birth defects (Sokol et al. 1981). This could lead investigators to overestimate the risk to the fetus of what might appear to be "two drinks a day."

All investigators studying drinking during pregnancy have been faced with the problems

of obtaining reliable drinking histories and have used a multitude of definitions of problem drinking, abusive drinking, heavy drinking, and alcohol dependence. For example, studies by Rosett et al. (1981), Streissguth et al. (1981), and Kuzma and Kissinger (1981) all used different criteria. Such variances in definition increase the difficulty of making comparisons across studies.

The stage of pregnancy is an important consideration in assessing women's drinking habits because women tend to decrease the amount they drink as pregnancy progresses, an observation first reported by Little et al. (1976) and subsequently corroborated by others (e.g., Rosett et al. 1978; Sokol et al. 1981; Weiner et al. 1983) and by the National Center for Health Statistics neonatal survey as discussed in chapter 1 of this report. Fried and his coworkers (1980) in a study of 217 Canadian women found that prior to pregnancy 18 percent of women were heavy (0.85 ounces per day) social drinkers, but that during the first trimester this proportion was reduced by two-thirds. The proportion of women who were drinking heavily continued to decrease during the last two trimesters of pregnancy. In the Cleveland study (Sokol et al. 1981), the heavier drinking group also was found to decrease its alcohol intake significantly as gestation progressed. These are encouraging findings, inasmuch as decreases in drinking would be expected to minimize birth defects. They point up the problem, however, of attempting to summarize a woman's drinking throughout pregnancy with a single number, e.g., "two drinks a day."

Paternal Drinking

Women who drink heavily tend to be married to men who drink heavily (Gomberg 1975). This suggests that some of the abnormalities described as alcohol-related birth defects might be related to paternal drinking. Long-term consumption by men of large amounts of alcohol is known to be associated with impotence, reduced sperm motility and ultimately loss of sperm, and abnormalities in sperm structure (Lester and Van Thiel 1977). Thus, fertilization with defective sperm is possible, which might result in fetal anomalies. Some investigators have argued that the male parent could contribute to alcohol-related birth defects (Anderson et al. 1981); however, no convincing evidence exists of such paternally mediated anomalies related to alcohol. Recently, Kuzma and Sokol (1982) examined 44

potential determinants of infant birth weight adjusted for gestational age and found no significant effect of paternal alcohol consumption. Similarly, Randall and her coworkers (1982) were unable to detect any effects of chronic paternal alcohol consumption on fetal growth and development in mice, in terms of the number of implantation sites, prenatal mortality, fetal weight, sex ratio, or the frequency of soft tissue malformation. These findings were confirmed by Bennett et al. (1982). Some studies reporting paternally mediated alcohol effects in conjunction with alcohol have had such methodologic flaws as lack of nutritional controls.

Knowledge and Attitudes

Public education efforts concerning risks associated with alcohol use during pregnancy have been actively pursued since the mid-1970s. Broad coverage in the media has been obtained for public health advisories from NIAAA, the American Medical Association, and the March of Dimes on a national level, as well as for an advisory from the Surgeon General (FDA 1981). Public education has also been attempted by many State governments. Most Americans have been exposed to such public health messages, and 90 percent of the respondents in a recent survey knew that drinking during pregnancy might be harmful. However, three-quarters of the respondents who thought abstinence is unnecessary believed that an average of more than three drinks per day was safe (Little et al. 1981). These findings suggest that current public education/prevention programs may not be fully adequate in permeating the public consciousness.

Although knowledge of potential adverse effects of alcohol on pregnancy outcome has changed, and public attitudes have shifted over the past 10 years, the question remains: Has the drinking behavior of women during pregnancy changed? The purpose of a recent study was to compare drinking patterns during pregnancy in the same population in Seattle over a 6-year interval (Streissguth et al. 1983a). Samples of pregnant women were interviewed in 1974-75 and in 1980-81. Although the proportion of women drinking during pregnancy was found to have decreased during the 6-year interval, the proportion of women drinking at least an ounce of absolute alcohol (two drinks) each day was relatively constant. Because precisely this limited proportion of the population incurs the highest risk for al-

cohol-related birth defects, these findings suggest cause for concern.

Conclusions

Although studies of drinking during pregnancy depend upon self-report and the reliability and precision of such data are limited, it appears that drinking during pregnancy is not uncommon. Many women who drink either decrease their drinking naturally or become abstinent during pregnancy. Public education efforts may well reinforce this change in behavior. However, persistently heavy drinking during pregnancy remains a problem for some women despite public education efforts.

Implications of Current Knowledge—Frontiers

The preceding sections have summarized the current status of information concerning risks for alcohol-related dangers to pregnancy and birth defects. Progress in scientific evaluation of these risks has clearly been very rapid over the past decade. However, it is equally clear that many questions remain.

Need for Further Research

Timing, pattern, and beverage source.—Because a reliable biochemical indicator for alcohol intake is not available, clinical investigators must depend on patient reports. However, as previously noted, human drinking is difficult to represent numerically in a single (and simple) measure of alcohol use; volume, frequency, and beverage source of alcohol intake must be considered. Also, other potential determinants of pregnancy outcome, e.g., other substance use, medical/obstetric factors, and socioeconomic status should be taken into account.

Improved measures of alcohol intake would heighten understanding of the stages of pregnancy when drinking is most dangerous to the fetus. Different alcohol-related birth defects might result from drinking during different critical periods, e.g., either early or late in pregnancy. Sulik and her colleagues (1981) suggest that heavy alcohol consumption early in pregnancy might contribute to such birth defects as abnormalities in the face and central nervous system. Studies of the relationship between early pregnancy drinking, particularly binge drinking, and physical congenital anomalies in the human could be valuable.

Heavy drinking in late pregnancy leads to an increased frequency of congenital anomalies (Rosett et al. 1978; Rosett and Weiner 1981). Although organogenesis, the development of the organs of the embryo, may be completed during very early pregnancy, nerve cells, for example, continue to make new connections through pregnancy. Heavy drinking could impair the growth of these connections. The results of animal studies suggest that after toxic substances are withdrawn, the fetus may compensate for earlier impairments with accelerated growth and repair before development is completed (Snow and Tam 1979; Anders and Persaud 1980). Further basic science studies in animals and clinical research in humans regarding critical periods of exposure and the capacity of the unborn offspring for compensatory growth and repair are needed, because the results of such studies might be useful in counseling patients whose fetuses have been exposed to significant levels of alcohol during early pregnancy.

Beverage source (wine, beer, whisky, etc.) may affect infants differentially. Animal studies have been few and inconclusive (Abel 1981b; Abel et al. 1981b). Nonetheless, possible relationships between beverage source and pregnancy outcome have been reported (Kaminski et al. 1978; Kuzma and Sokol 1982; Berkowitz et al. 1982).

Moderate drinking.—Moderate drinking may increase the risk for lowered birth weight (Little 1977), abnormal neurobehavioral development (Streissguth et al. 1980), and spontaneous abortion (Harlap and Shiono 1980; Kline et al. 1980). However, others (Rosett 1980; Sokol 1980b, 1982) have questioned whether the women manifesting these increased pregnancy risks are in fact moderate drinkers. They note that the women with alcohol-related pregnancy risk constitute only the heaviest drinking (5 to 10 percent) of pregnant women. As research continues to expand knowledge, the answers to this important clinical question will emerge.

Susceptibility.—Different species and different strains within a species of animal may differ in their susceptibility to alcohol-related birth defects. Also, differences among animals in the same exposed litter are commonly observed. As noted earlier, human fraternal twins may not be equally affected by alcohol exposure (Christoffel and Salafsky 1975). Such findings suggest the possibility of differential susceptibility on a genetic basis. Interactions

of alcohol consumption with other pregnancy risks, such as poor nutrition, other substance use, or medical illnesses, may also alter susceptibility. Information about interacting risks would help in designing more focused and potentially more effective prevention strategies. A profile of patients particularly at risk for the adverse effects of in utero alcohol exposure also would aid the practitioner in giving direct patient care.

Prevention and Treatment

Problems.—Although major progress has been made over the past decade in understanding the risks of alcohol use during pregnancy, much remains unknown. Methods are being developed to identify and treat pregnant women with alcohol problems (Rosett et al. 1981; Sokol et al. 1981). Education programs aimed at professionals may modify attitudes and clinical behavior (Weiner et al. 1982; Little et al. 1983). Professional education, based on firm scientific evidence concerning risks for alcohol-related birth defects, effective methods of detection of abusive drinking, and treatment methods for abusive drinking and alcohol dependence, appears to be a promising avenue for the prevention of alcohol-related birth defects and other anomalies.

Current statements and recommendations.—Based on currently available information, some broad statements with prevention and treatment implications appear to be warranted. Extensive public education efforts have alerted women to the dangers and fewer American women appear to be drinking during pregnancy. However, the frequency of heavy or abusive drinking may not have decreased. It can be stated with some certainty that those pregnant women who are the heaviest (most frequent) drinkers (about 5-10 percent) are exposing their unborn children to a significant risk. If these women abstain or significantly decrease their drinking during pregnancy, the evidence from both animal and human studies suggests that they can have healthier babies.

Effective prevention strategies for alcohol-related birth defects, including FAS, remain elusive. More information is needed about alcohol abuse and dependence in young women, so that more focused approaches to prevention can be developed. Professional education of

physicians, nurses, and other health care providers may afford a rational and cost-effective approach to prevention of alcohol-related birth defects. If these individuals can be educated to take an active role in influencing the drinking habits of their patients, it may be possible to decrease the occurrence of alcohol-related birth defects and improve the probability of successful pregnancies.

Summary

Heavy drinking during pregnancy adversely affects organic and behavioral fetal development and increases the risks of miscarriage. When consumed in large amounts, alcohol can result in fetal alcohol syndrome. It appears that the overall prevalence of FAS is in the range of 1 to 3 cases per 1,000 births. Alcohol-related birth defects occur more frequently than FAS.

Alcohol can induce abnormal fetal development that varies in intensity, depending on dose, genetic susceptibility, nutritional status, and other as yet unknown factors. Heavy maternal drinking may affect the fetal nervous system, which may result in abnormal neonatal behavior, childhood hyperactivity, and mental retardation.

Alcohol probably affects all organ systems of the fetus, but many questions remain as to the mechanisms and their potential reversibility. Many variables, such as the amount of alcohol and the time during pregnancy of consumption, may influence the nature and degree of adverse effects. Studies in animals suggest that, at high alcohol levels, birth anomalies are directly related to the amount of alcohol exposure.

Public education efforts concerning risks associated with alcohol use have been actively pursued over recent years. These education efforts have encompassed media messages, public health advisories, and programs at the State level. Most Americans have been exposed to public health messages; however, the impact on heavier drinking in pregnancy has been less than desired. One study found that while the proportion of women who drank during pregnancy decreased over the past 6 years, the proportion of women drinking at least two drinks a day had not changed.

References

- Abel, E.L. Prenatal effects of alcohol on adult learning in rats. Pharmacology, Biochemistry and Behavior 10:239-243, 1979.
- Abel, E.L. Behavioral teratology of alcohol. Psychological Bulletin 90:564-581, 1981a.
- Abel, E.L. Prenatal effects of beverage alcohol on fetal growth. Progress in Biochemical Pharmacology 18:111-114, 1981b.
- Abel, E.L. Alcohol and Reproduction: A Bibliography. Westport, Conn.: Greenwood Press, 1982a.
- Abel, E.L. Consumption of alcohol during pregnancy: A review of effects on growth and development of offspring. Human Biology 54:421-453, 1982b.
- Abel, E.L. In utero alcohol exposure and developmental delay of response inhibition. Alcoholism: Clinical and Experimental Research 6:369-376, 1982c.
- Abel, E.L. Marijuana, Tobacco and Alcohol Effects in Reproduction. Boca Raton, Fla.: CRC Press, in press.
- Abel, E.L.; Dintcheff, B.A.; and Bush, R. Behavioral teratology of alcoholic beverages compared to ethanol. Neurobehavioral Toxicology and Teratology 3:339-342, 1981a.
- Abel, E.L.; Dintcheff, B.A.; and Bush, R. Effects of beer, wine, whiskey and ethanol on pregnant rats and their offspring. Teratology 23:217-222, 1981b.
- Altshuler, H.L., and Shippenberg, T.S. A subhuman primate model for fetal alcohol syndrome research. Neurobehavioral Toxicology and Teratology 3:121-126, 1981.
- Anders, K., and Persaud, T.V.N. Compensatory embryonic development in the rat following maternal treatment with ethanol. Anatomischer Anzeiger 148:375-383, 1980.
- Anderson, R.A.; Furby, J.E.; Oswald, C.; and Zaneveld, L.J.D. Teratological evaluation of mouse fetuses after paternal alcohol ingestion. Neurobehavioral Toxicology and Teratology 3:117-120, 1981.
- Barnes, D.E., and Walker, D.W. Prenatal ethanol exposure permanently reduces the number of pyramidal neurons in rat hippocampus. Developmental Brain Research 1:333-340, 1981.
- Bennett, A.L.; Sorette, M.P.; and Greenwood, M.R.C. Effect of chronic paternal ethanol consumption on 19-day rat fetuses. Federation Proceedings 41:710, 1982.
- Berkowitz, G.S. An epidemiologic study of preterm delivery. American Journal of Epidemiology 113:81-92, 1981.
- Berkowitz, G.S.; Holford, T.R.; and Berkowitz, R.L. Effects of cigarette smoking, alcohol, coffee and tea consumption on preterm delivery. Early Human Development 7:239-250, 1982.
- Bond, N.W. Prenatal exposure to ethanol: Association between increased gestational length and offspring mortality. Neurobehavioral Toxicology and Teratology 4:501-503, 1982.
- Bond, N.W., and DiGiusto, E.L. Effects of prenatal alcohol consumption on open-field behaviour and alcohol preference in rats. Psychopharmacology (Berlin) 46:163-165, 1976.
- Bond, N.W., and DiGiusto, E.L. Avoidance conditioning and Hebb-Williams maze performance in rats treated prenatally with alcohol. Psychopharmacology (Berlin) 58:69-71, 1978.
- Brown, N.A.; Goulding, E.H.; and Fabro, S. Ethanol embryotoxicity: Direct effects on mammalian embryos in vitro. Science 206: 573-575, 1979.
- Cahalan, D.; Cisin, L.H.; and Crossley, H.M. American Drinking Practices: A National Study of Drinking Behavior and Attitude. Monograph No. 6. New Brunswick, N.J.: Rutgers Center of Alcohol Studies, 1969.
- Chernoff, G.F. The fetal alcohol syndrome in mice: An animal model. Teratology 15:223-229, 1977.
- Christoffel, K.K., and Salafsky, L. Fetal alcohol syndrome in dizygotic twins. Journal of Pediatrics 87:963-967, 1975.
- Clark, W., and Midanik, L. Alcohol use and alcohol problems among U.S. adults. Results of the 1979 national survey. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM)82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 3-52.
- Clarren, S.K., and Smith, D.W. The fetal alcohol syndrome: A review of the world literature. New England Journal of Medicine 298:1063-1067, 1978.
- Clarren, S.K.; Alvord, E.C.; Sumi, S.M.; Streissguth, A.P.; and Smith, D.W. Brain malformations related to prenatal exposure to ethanol. Journal of Pediatrics 92:64-67, 1978.
- Davies, D.L., and Smith, D.E. Effects of perinatally administered ethanol on hippocampal development (abstract). Alcoholism: Clinical and Experimental Research 5:147, 1981.
- Dehaene, P.; Samaille-Villette, C.; Samaille, P.; Crepin, G.; Walbaum, R.; Deroubaix, P.; and Blanc-Garin, A.P. Le syndrome d'alcoolisme foetal dans le nord de la France. [The fetal alcohol syndrome in the north of France.] Revue de l'Alcoolisme (Paris) 23: 145-158, 1977.
- Dexter, J.D.; Tumbleson, M.E.; Decker, J.D.; and Middleton, C.C. Fetal alcohol syndrome in Sinclair (S-1) miniature swine. Alcoholism: Clinical and Experimental Research 4:146-151, 1980.
- Ellis, F.W., and Pick, J.R. An animal model of the fetal alcohol syndrome in beagles. Alcoholism: Clinical and Experimental Research 4:123-134, 1980.
- Flynn, A.; Miller, S.L.; Martier, S.S.; Golden, N.L.; Sokol, R.J.; and Del Villano, B.C. Zinc status of pregnant alcoholic women: A determinant of fetal outcome. Lancet 1:572-575, 1981.
- Food and Drug Administration. Surgeon General's Advisory on Alcohol and Pregnancy. FDA Drug Bulletin 11:9-10, 1981.

- Fried, P.A.; Watkinson, B.; Grant, A.; and Knights, R.M. Changing patterns of soft drug use prior to and during pregnancy: A prospective study. Drug and Alcohol Dependence 6:323-343, 1980.
- Ghishan, F.K.; Patwardhan, R.; and Green, H.L. Fetal alcohol syndrome: Inhibition of placental zinc transport as a potential mechanism for fetal growth retardation in the rat. Journal of Laboratory and Clinical Medicine 100:45-52, 1982.
- Golden, N.L.; Sokol, R.J.; Kuhnert, B.R.; and Botto, S.F. Maternal alcohol use and infant development. Pediatrics 70: 931-934, 1982.
- Gomberg, E.S. Alcoholism and Women: State of Knowledge Today. New York: National Council on Alcoholism, 1975. 12 pp.
- Greizerstein, H.B., and Abel, E.L. Acute effects of ethanol on fetal body composition and electrolyte content in the rat. Bulletin of the Psychonomic Society 14:355-356, 1979.
- Hanson, J.W.; Streissguth, A.P.; and Smith, D.W. The effects of moderate alcohol consumption during pregnancy on fetal growth and morphogenesis. Journal of Pediatrics 92:457-460, 1978.
- Harlap, S., and Shiono, P.H. Alcohol, smoking and incidence of spontaneous abortions in the first and second trimester. Lancet 2:173-176, 1980.
- Henderson, G.L.; Hoyumpa, A.M., Jr.; McClain, C.; and Schenker, S. The effects of chronic and acute alcohol administration on fetal development in the rat. Alcoholism: Clinical and Experimental Research 3:99-106, 1979.
- Henderson, G.L.; Hoyumpa, A.M., Jr.; Rothschild, M.A.; and Schenker, S. Effect of ethanol and ethanol-induced hypothermia on protein synthesis in pregnant and fetal rats. Alcoholism: Clinical and Experimental Research 4:165-177, 1980.
- Henderson, G.L.; Turner, D.; Patwardhan, R.V.; Lumeng, L.; Hoyumpa, A.M.; and Schenker, S. Inhibition of placental valine uptake after acute and chronic maternal ethanol consumption. Journal of Pharmacology and Experimental Therapeutics 216:465-472, 1981.
- Hingson, R.; Alpert, J.J.; Day, N.; Dooling, E.; Kayne, H.; Morelock, S.; Oppenheimer, E.; and Zuckerman, B. Effects of maternal drinking and marijuana use on fetal growth and development. Pediatrics 70:539-546, 1982.
- Jones, K.L., and Smith, D.W. Recognition of the fetal alcohol syndrome in early infancy. Lancet 2:999-1001, 1973.
- Jones, K.L.; Smith, D.W.; Ulleland, C.N.; and Streissguth, A.P. Pattern of malformation in offspring of chronic alcoholic mothers. Lancet 1:1267-1271, 1973.
- Kaminski, M.; Rumeau-Rouquette, C.; and Schwartz, D. Alcohol consumption in pregnant women and the outcome of pregnancy. Alcoholism: Clinical and Experimental Research 2:155-163, 1978.
- Kaminski, M.; Franc, M.; Lebouvier, M.; du Mazau-brun, C.; and Rumeau-Rouquette, C. Moderate alcohol use and pregnancy outcome. Neurobehavioral Toxicology and Teratology 3:173-181, 1981.
- Kline, J.; Shrout, P.; Stein, Z.; Susser, M.; and Warburton, D. Drinking during pregnancy and spontaneous abortion. Lancet 2:176-180, 1980.
- Kuzma, J.W., and Kissinger, D.G. Patterns of alcohol and cigarette use in pregnancy. Neurobehavioral Toxicology and Teratology 3:211-221, 1981.
- Kuzma, J.W., and Sokol, R.J. Maternal drinking behavior and decreased intrauterine growth. Alcoholism: Clinical and Experimental Research 6:396-402, 1982.
- Landesman-Dwyer, S.; Ragozin, A.S.; and Little, R.E. Behavioral correlates of prenatal alcohol exposure: A four-year followup study. Neurobehavioral Toxicology and Teratology 3:187-193, 1981.
- Lee, M., and Leichter, J. Effect of litter size on the physical growth and maturation of the offspring of rats given alcohol during gestation. Growth 44:327-335, 1980.
- Lester, R., and Van Thiel, D.H. Gonadal function in chronic alcoholic men. In: Gross, M. M., ed. Alcohol Intoxication and Withdrawal. IIIa. Biological Aspects of Ethanol. Advances in Experimental Medicine and Biology 85a:399-414, 1977.
- Little, R.E. Moderate alcohol use during pregnancy and decreased infant birth weight. American Journal of Public Health 67:1154-1156, 1977.
- Little, R.E.; Schultz, F.A.; and Mandell, W. Drinking during pregnancy. Journal of Studies on Alcohol 37:375-379, 1976.
- Little, R.E.; Grathwohl, H.L.; Streissguth, A.P.; and McIntyre, C. Public awareness and knowledge about the risks of drinking during pregnancy in Multnomah County, Oregon. American Journal of Public Health 71:312-314, 1981.
- Little, R.E.; Streissguth, A.P.; Guzinski, G.M.; Frathwohl, H.L.; Blumhagen, J.M.; and McIntyre, C.E. Change in obstetrician advice following a two-year community educational program on alcohol use and pregnancy. American Journal of Obstetrics and Gynecology 146:23-28, 1983.
- Lochry, E.A., and Riley, E.P. Retention of passive avoidance and T-maze escape in rats exposed to alcohol prenatally. Neurobehavioral Toxicology and Teratology 2: 107-115, 1980.
- Lochry, E.A.; Randall, C.L.; Goldsmith, A.A.; and Sutker, P.B. Effects of acute alcohol exposure during selected days of gestation in C3H mice. Neurobehavioral Toxicology and Teratology 4: 15-19, 1982.
- National Center for Health Statistics. Monthly Vital Statistics Report: Annual Summary for the United States, 1979. Hyattsville, Md.: the Center, 1980.
- Neugut, R.H. Epidemiological appraisal of the literature on the fetal alcohol syndrome in humans. Early Human Development 5:411-429, 1981.
- Olegard, R.; Sabel, K.-G.; Aronsson, M.; Sandin, B.; Johansson, P.R.; Carlsson, C.; Kyllerman, M.; Iversen, K.; and Hrbek, A. Effects on the child of alcohol abuse during pregnancy: Retrospective and prospective studies. Acta Paediatrica Scandinavica (Supplement No. 275):112-121, 1979.

- Ouellette, E.M.; Rosett, H.L.; Rosman, N.P.; and Weiner, L. Adverse effects on offspring of maternal alcohol abuse during pregnancy. New England Journal of Medicine 297:528-530, 1977.
- Peiffer, J.; Majewski, F.; Fischbach, H.; Bierich, J.R.; and Volk, B. Alcohol embryo- and fetopathy: Neuropathology of three children and three fetuses. Journal of Neurological Science 41:125-137, 1979.
- Popov, V.B.; Vaisman, B.L.; Puchkov, V.E.; and Ignat'eva, T.V. Embryotoxic effect of ethanol and products of its biotransformation in post-implanted rat embryo culture. Byull Exsp Biol Med 92(12): 725-728, 1981.
- Potter, B.J.; Belling, G.B.; Mano, M.T.; and Hetzel, B.S. Experimental production of growth retardation in the sheep fetus after exposure to alcohol. Medical Journal of Australia 2:191-193, 1980.
- Randall, C.L. Alcohol as a teratogen in animals. In: National Institute on Alcohol Abuse and Alcoholism. Biomedical Processes and Consequences of Alcohol Use. Alcohol and Health Monograph No. 2. DHHS Pub. No.(ADM)82-1191. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982, pp. 291-307.
- Randall, C.L., and Taylor, W.J. Prenatal ethanol exposure in mice: Teratogenic effects. Teratology 19:305-312, 1979.
- Randall, C.L.; Taylor, W.J.; and Walker, D.W. Ethanol-induced malformations in mice. Alcoholism: Clinical and Experimental Research 1:219-224, 1977.
- Randall, C.L.; Burling, T.A.; Lochry, E.A.; and Sutker, P.B. The effect of paternal alcohol consumption on fetal development in mice. Drug and Alcohol Dependence 9:89-95, 1982.
- Randall, C.L.; Lochry, E.A.; Hughes, S.S.; and Sutker, P.B. Dose-response effect of prenatal alcohol exposure on fetal growth and development in mice. Substance and Alcohol Actions/Misuse 2:349-357, 1981.
- Rasmussen, B.B., and Christensen, N. Teratogenic effect of maternal alcohol consumption on the mouse fetus: A histopathological study. Acta Pathologica Microbiologica Scandinavica (Section A, Pathology) 88:285-289, 1980.
- Riley, E.P.; Lochry, E.A.; and Shapiro, N.R. Lack of response inhibition in rats prenatally exposed to alcohol. Psychopharmacology 62:47-52, 1979a.
- Riley, E.P.; Lochry, E.A.; Shapiro, N.R.; and Baldwin, J. Response perseveration in rats exposed to alcohol prenatally. Pharmacology, Biochemistry and Behavior 10:255-259, 1979b.
- Rosett, H.L. A clinical perspective on the fetal alcohol syndrome (editorial). Alcoholism: Clinical and Experimental Research 4:119-122, 1980.
- Rosett, H.L., and Weiner, L. Identifying and treating pregnant patients at risk from alcohol. Canadian Medical Association Journal 125:149-154, 1981.
- Rosett, H.L.; Weiner, L.; and Edelin, K.C. Strategies for prevention of fetal alcohol effects. Obstetrics and Gynecology 57:1-7, 1981.
- Rosett, H.L.; Ouellette, E.M.; Weiner, L.; and Owens, E. Therapy of heavy drinking during pregnancy. American Journal of Obstetrics and Gynecology 51:41-46, 1978.
- Rosett, H.L.; Weiner, L.; Lee, A.; Zuckerman, B.; Dooling, E. and Oppenheimer, E. Patterns of alcohol consumption and fetal development. Obstetrics and Gynecology 61: 539-546, 1983.
- Rosett, H.L.; Snyder, P.; Sander, L.W.; Lee, A.; Cook, P.; Weiner, L.; and Gould, J. Effects of maternal drinking on neonate state regulations. Developmental Medicine and Child Neurology 21:464-473, 1979.
- Russell, M. The impact of alcohol-related birth defects (ARBD) on New York State. Neurobehavioral Toxicology 2:277-283, 1980.
- Russell, M., and Bigler, L.R. Screening for alcohol-related problems in an outpatient obstetric-gynecologic clinic. American Journal of Obstetrics and Gynecology 134: 4-12, 1979.
- Samson, H.H., and Diaz, J. Altered development of brain by neonatal ethanol exposure: Zinc levels during and after exposure. Alcoholism: Clinical and Experimental Research 5:563-569, 1981.
- Shaywitz, S.E.; Cohen, D.J.; and Shaywitz, B.A. Behavior and learning difficulties in children of normal intelligence born to alcoholic mothers. Journal of Pediatrics 96: 978-982, 1980.
- Snow, M.H.L., and Tam, P.P.L. Is compensatory growth a complicating factor in mouse teratology? Nature 279:555-557, 1979.
- Sokol, R.J. Alcohol-in-pregnancy: Clinical research problems. Neurobehavioral Toxicology and Teratology 2:157-165, 1980a.
- Sokol, R.J. Alcohol and spontaneous abortion (letter to the editor). Lancet 2:1079, 1980b.
- Sokol, R.J. Alcohol and abnormal outcomes of pregnancy. Canadian Medical Association Journal 125:143-148, 1981.
- Sokol, R.J. Alcohol and pregnancy: A clinical perspective for laboratory research. Substance and Alcohol Actions/Misuse 3:183-186, 1982.
- Sokol, R.J.; Miller, S.I.; and Reed, G. Alcohol abuse during pregnancy: An epidemiologic study. Alcoholism: Clinical and Experimental Research 4:135-145, 1980.
- Sokol, R.J.; Miller, S.I.; and Martier, S. Identifying the alcohol abusing obstetric/gynecologic patient: A practical approach. In: National Institute on Alcohol Abuse and Alcoholism. Preventing Fetal Alcohol Effects: A Practical Guide for Ob/Gyn Physicians and Nurses. DHHS Pub. No (ADM)81- 1163. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981, pp. 5-20.
- Streissguth, A.P.; Herman, C.S.; and Smith, D.W. Intelligence, behavior and dysmorphogenesis in the fetal alcohol syndrome: A report on 20 patients. Journal of Pediatrics 92:363-367, 1978a.
- Streissguth, A.P.; Herman, C.S.; and Smith, D.W. Stability of intelligence in the fetal alcohol syndrome: A preliminary report. Alcoholism: Clinical and Experimental Research 2:165-170, 1978b.

- Streissguth, A.P.; Barr, H.M.; Martin, D.C.; and Herman, C.S. Effects of maternal alcohol, nicotine and caffeine use during pregnancy on infant mental and motor development at 8 months. Alcoholism: Clinical and Experimental Research 4:152-164, 1980.
- Streissguth, A.P.; Martin, D.C.; Martin, J.C.; and Barr, H.M. The Seattle longitudinal prospective study on alcohol and pregnancy. Neurobehavioral Toxicology and Teratology 3:223-233, 1981.
- Streissguth, A.P.; Darby, B.L.; Barr, H.M.; Smith, J.R.; and Martin, D.C. Comparison of drinking and smoking patterns during pregnancy over a six-year interval. American Journal of Obstetrics and Gynecology 145: 716-724, 1983.
- Streissguth, A.P.; Martin, D.C.; Barr, H.M.; Sandman, B.A.; Kirchner, G.L.; and Darby, B.L. Intra-uterine alcohol exposure and attentional decrements in 4-year-old children (abstract). Alcoholism: Clinical and Experimental Research 7:122, 1983.
- Sulik, K.K.; Johnston, M.C.; and Webb, M.A. Fetal alcohol syndrome: Embryogenesis on a mouse model. Science 214:936-938, 1981.
- Sullivan, W.C. A note on the influence of maternal inebriety on the offspring. Journal of Mental Science 45:489- 503, 1899.
- Tanaka, H.; Suzuki, N.; and Arima, M. Hypoglycemia in the fetal alcohol syndrome in the rat. Brain and Development 4:97-103, 1982.
- U.S. Department of Health and Human Services. Fourth Special Report to the U.S. Congress on Alcohol and Health. DeLuca, J.R., ed. DHHS Pub. No. (ADM)81-1080. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- U.S. Department of Health, Education, and Welfare. Third Special Report to the U.S. Congress on Alcohol and Health. Noble, E.P., ed. DHEW Pub. No. (ADM)79-832. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1978.
- Warner, R.H., and Rosett, H.L. The effects of drinking on offspring: An historical survey of the American and British literature. Journal of Studies on Alcohol 36:1395-1420, 1975.
- Weiner, L.; Rosett, H.L.; and Edelin, K.C. Behavioral evaluation of fetal alcohol education for physicians. Alcoholism: Clinical and Experimental Research 6:230-233, 1982.
- Weiner, L.; Rosett, H.L.; Edelin, K.C.; Alpert, J.J.; and Zuckerman, B. Alcohol consumption by pregnant women. Obstetrics and Gynecology 61:6-12, 1983.
- West, J.R.; Hodges, C.A.; and Black, A.C., Jr. Prenatal exposure to ethanol alters the organization of hippocampal mossy fibers in rats. Science 211:957-959, 1981.
- Wiener, S.G.; Shoemaker, W.J.; Koda, L.Y.; and Bloom, F.E. Interaction of ethanol and nutrition during gestation: Influence on maternal and offspring development in the rat. Journal of Pharmacology and Experimental Therapeutics 216: 572-579, 1981.
- Wilson, J.G. Environment and Birth Defects. New York: Academic Press, 1973. 305 pp.

Chapter VI

Adverse Social Consequences of Alcohol Use and Alcoholism

Heavy drinking and chronic alcoholism contribute to a variety of social problems. In addition to accidents involving various modes of transportation, accidents occur in the home, at work, and during recreational activities. Alcohol use also contributes to loss of work productivity, homicide, suicide, and assaultive behaviors. In a 1982 national Gallup survey, one-third of persons interviewed said alcohol had caused problems in their families (Gallup 1982).

Research on the contribution of heavy alcohol consumption and alcoholism to social problems varies in quality. Although the causal link is sometimes weak or nonexistent, the best evidence available strongly suggests that many of these problems are much more likely to arise among alcoholics or heavy drinkers than among other persons. Moreover, the potential for some of these problems to occur may be heightened by even moderate consumption (Moore and Gerstein 1981, pp. 44-45). Since moderate drinkers outnumber heavy drinkers, moderate drinking may actually contribute substantially to social problems. Because most individuals do not believe they personally will experience drinking-related problems, it has been difficult to sensitize the public to social problems caused by moderate drinking.

Alcohol and Accidents

Traffic Accidents

Traffic accidents across the country resulted in 49,268 deaths in 1981 (USDOT 1983). Nearly 150,000 persons are permanently disabled each year in automobile accidents, and 1.8 million persons sustain injuries that restrict their activities for at least 1 full day after a crash (National Safety Council 1982). During the past decade, more than 450,000 traffic accident fatalities constituted the fifth leading overall cause of death in the United

States and the leading cause of death in the 1- to 35-year-old age group (Ross 1982).

After analyzing case reports, simulated driving conditions, and epidemiologic data, researchers have consistently and unequivocally concluded that alcohol contributes significantly to traffic accidents. The higher the amount of alcohol consumed, the greater the likelihood that an accident will occur and that the accident will be serious or fatal.

In a report to the National Institute on Alcohol Abuse and Alcoholism (NIAAA), Cameron (1977) cited 23 studies showing that at least one-third of the drivers in fatal accidents had blood alcohol concentrations (BACs) exceeding .10 percent. This effect roughly equals that in a 155-pound man who consumed at least five or six drinks or more within an hour. Estimates place the annual number of deaths in traffic accidents in which at least one driver had been drinking at 16,000 to 30,000 (Roizen 1982). Between 20 and 40 percent of people killed in these drinking-driving accidents were persons other than the drunk driver (Reed 1981).

Borkenstein and colleagues (1974) interviewed Indiana drivers about their drinking and tested their blood alcohol contents. Regardless of the frequency with which drivers reported drinking, elevated BACs increased crash probabilities. Studies comparing drivers in fatal accidents with drivers not involved in accidents in similar locations at similar times suggest that drivers with BACs above .10 percent are 3 to 15 times more likely than nondrinking drivers to be involved in a fatal crash (Cameron 1977). For young drivers, in particular, the risk of a fatal crash increases sharply with increasing BAC levels (Perrine et al. 1971; Wagenaar et al. 1981). For further discussion of this topic, see tables 4 and 5 in chapter 1 of this volume.

Because of the high risk of fatal accident involvement after heavy drinking, the risks posed by driving after moderate drinking are sometimes overlooked. According to studies in New York City (McCarroll and Haddon 1962)

and Vermont (Perrine et al. 1971), even drivers with BACs of .05 percent to .099 percent are 1.25 to 3.25 times more likely to be involved in fatal accidents than nondrinking drivers. According to the U.S. Department of Transportation (Fell 1982), while 50 percent of fatally injured drivers had BACs above .10 percent, 12 percent had elevated BACs between .01 percent and .10 percent. Reed (1981) has estimated that if all drivers in this country in 1977 were only exposed to the risk of drivers who do not drive after drinking, there would have been 24 percent fewer accidental deaths (11,700 deaths prevented) and 156,000 to 300,000 fewer injuries.

Alcohol contributes to traffic accidents by affecting cognitive and behavioral function, often in multiple ways in the same accident. Alcohol in the blood at levels above .10 percent is known to (1) lower divided attention performance, (2) reduce dynamic visual activity, (3) reduce adaptation to brightness and glare resistance recovery, and (4) reduce ability to see flickering lights, e.g., turn signals (Perrine 1974). Some researchers believe alcohol also interferes with the brain's ability to process large quantities of information at one time (Moskowitz 1974). Still others cite the sedating, fear reducing, and disinhibitory effects of alcohol on aggressiveness or self-destructiveness. Cognitive effects such as short-term memory loss or learning deficits may also contribute to accidents (Barry 1974). Those willing to drive after heavy drinking, of course, may generally engage in high risk-taking behavior and may be impulsive and careless (Jones and Joscelyn 1978). This may place them at increased risk for highway accidents regardless of their drinking behavior.

Most researchers concur that chronic heavy drinkers or alcoholics are more likely than light drinkers or nondrinkers to be involved in traffic accidents (Fell 1982). Estimates vary widely, however, on the proportion of accidents involving each category, largely because of variable criteria for diagnosing alcoholics. Klatsky et al. (1981) compared 2,015 Kaiser Permanente plan members who averaged six or more drinks daily with 6,045 light drinkers and nondrinkers matched by age, sex, race, cigarette use, and city. Those who reported drinking more than six drinks daily were three times more likely than the rest of the sample to die in a traffic accident during the subsequent 10 years.

Although persons with a previous driving-while-intoxicated (DWI) arrest run a higher accident risk than other drivers, they are in-

involved in only a small fraction of fatal accidents. Sterling-Smith (1976) reported that of drivers known to be responsible for fatal accidents in the early 1970s in Massachusetts, only 4 percent had a previous DWI arrest, about one-tenth of those in the study with a BAC above .05 percent. The implication is that even highly successful rehabilitation programs aimed at the arrested drunk driver may influence only a small minority of drunk drivers. If the percentage of drunk drivers who are arrested increases, however, these successful rehabilitation percentages may increase.

Despite widespread awareness of its risk, drunk driving occurs relatively frequently. Researchers have estimated that at any given time 2 to 3 percent of the driving population are legally intoxicated, and that the percentage doubles at night or on weekends, the hours during which accidents are most frequent (Damkot et al. 1977; Haddon and Blumenthal 1981). As many as 20 percent of nighttime drivers randomly stopped have been found to have measurable BACs (Damkot et al. 1977). Statewide random sample surveys of nearly 1,000 adults in Massachusetts in 1981 indicated that 29 percent of drivers report driving after drinking at least four times in the prior month, and 4 percent report driving after six or more drinks at least four times a month. Males, persons who are unmarried, and those under age 25 most often reported driving after drinking heavily (Hingson et al. 1982a, 1983).

At least three reasons explain why people drive after drinking. First, a considerable number of people who drink do not know the level of drinking that makes individuals legally too intoxicated to drive. The 1981 Massachusetts survey indicated that 10 percent of the respondents who drive did not know the level of legal intoxication, and 17 percent felt that a person can drink six or more drinks without being legally too drunk to drive. Second, even though the relative risk of accident involvement for persons who drive under the influence is much higher than for nondrinkers, the probability of an accident during a trip when the driver is under the influence is still quite low, only about 2 per 1,000 trips (Ross 1982). The risk of arrest is roughly comparable, after which the drunk driver would still stand only a 50-50 chance of being punished (Jones and Joscelyn 1978; Ross 1982). Finally, driving after drinking has long been considered socially acceptable behavior. While political and social pressures have prompted 34 States to change their drunk driving laws during the past

2 years (Volpe 1982), it is not clear whether this will substantially alter public complacency about drunk driving and the frequency of its occurrence.

Pedestrian Accidents

At least 8,000 pedestrians are killed and 100,000 injured each year by motor vehicles. Research indicates measurable BACs among fatally injured pedestrians ranging from 35 to 74 percent (Blomberg et al. 1979).

Only two studies compared fatally injured pedestrians with noninjured pedestrians who passed the same spot at a similar time of day and day of week (Haddon et al. 1961; Blomberg et al. 1979). In the more recent and more extensive study (Blomberg et al. 1979), between July 1972 and March 1976, postmortem BAC results were obtained on all 86 fatally injured pedestrians taken to a New Orleans hospital. All 180 injured pedestrians taken to the same hospital between March 1975 and March 1976 were also tested. Pedestrians who passed the same site within 30 minutes of the accidents on the same day of the week and year were also stopped; 1,208 of 1,469 eligible control respondents were given blood alcohol tests and interviewed.

Thirty-six percent of the injured pedestrians had BACs over .10 percent, as did 45 percent of the pedestrians who died. Only 13 percent of controls had BACs at that level. Compared with age- and sex-matched nondrinkers, the risk of accident involvement for pedestrians was two times higher with BACs above .10 percent and five times higher for those with BACs above .20 percent. The relative risk rose rapidly as the blood alcohol concentration increased beyond .20 percent.

Driver error was less frequently the cause when the pedestrian had been drinking. Alcohol-involved accidents were more frequent among male pedestrians aged 20 to 49 and tended to cluster on weekends and at night.

Airplane Accidents

Alcohol is also a factor in aviation accidents (U.S. National Transportation Safety Board 1979). In 678 fatal plane crashes nationwide in 1979, alcohol impairment was identified in 30 pilots, most of whom had BACs above .10 percent. Because the proportion of pilots who fly after drinking and the proportion of flight time under the influence are not known, however, the relative risk of aviation accidents at various drinking levels is yet unclear.

Occupational Accidents

Work-related accidents were responsible for 13,000 fatalities in 1980, and 80,000 persons were permanently disabled, partially or totally (National Safety Council 1982). National survey data suggest that yearly 25 percent of workers sustain some sort of injury on the job (Quinn and Shepard 1974). About 35 percent of the work-related fatal accidents are represented in the traffic accident statistics already cited.

Accidents on the job are not as extensively researched as traffic accidents; less is known about their occurrence and contributory factors (including alcohol). Baker and associates (1982) indicated that in 1978, 148 Maryland workers died in job-related accidents. Blood alcohol concentration levels were obtained on 79 percent of those from whom BACs could technically have been obtained. About 11 percent of both motor vehicle fatalities and fatalities from other causes had BACs above .08 percent. In contrast, according to a national survey of workers (Quinn and Shepard 1974), only 2 percent of the working population can be expected to drink on the job on any given day. Although the types of jobs and worker characteristics in a national sample may systematically differ from those in Maryland, these data suggest that people who have fatal accidents on the job were much more likely than other employees to drink at work.

Given the well-established relationships between blood alcohol levels and poor coordination, faulty judgment, and lengthened reaction time, it is plausible that alcohol often contributes to accidents at work. Of course, the risk may well vary with the type of work.

Some data also suggest that alcoholics are more likely than other workers to experience work-related accidents, but the results have not been consistent. Shain (1982) studied one firm located in the eastern United States and found that alcoholics are two to three times more likely than other employees to be involved in work accidents, but alcoholism was not adequately defined in this study. Trice (1965) compared 72 alcoholic employees with "normal" employees and found no relation between alcoholism and accident involvement during the 5 years preceding and 1 year following diagnosis.

Additional research is needed on alcohol use and alcoholism as they affect accident involvement at work. Research priority should be given to (1) national or regional surveys to

determine the prevalence of on-the-job drinking and the incidence of accidents after drinking, (2) systematic BAC testing on all occupational fatalities, and (3) case control studies comparing alcohol use by employees involved in occupational accidents with that of employees who work at similar jobs but were not involved in accidents.

Valid data may be difficult to obtain because of legal questions concerning financial responsibility for defective products as well as worker compensation when the worker had been drinking prior to the accident. Furthermore, other characteristics that contribute to drinking on the job (job boredom, stress) could independently relate to accident involvement.

Home and Recreational Accidents

Nationwide, in 1980, 22,800 persons died and another 90,000 were permanently injured in home accidents. Falls accounted for about one-third of home accident deaths, and burns or injuries resulting directly from fires comprised another 20 percent. Outside the home, 21,000 persons died and 60,000 were permanently injured in non-motor-vehicle accidents such as falls, drownings, and other recreational accidents. From 1970 to 1980, the sharpest declines in fatal accident rates--about 25 percent--were recorded in these groupings (National Safety Council 1982).

The role of alcohol is less well understood in home and nontransportation accidents than in traffic accidents because on-the-scene investigations are less frequently conducted and most studies are undertaken in hospital emergency clinics. Moreover, because these accidents encompass a wider variety of circumstances, identifying specific causes for each accident type is more difficult. Since different researchers use different taxonomies, it is difficult to combine the results into effective generalizations.

The few studies available suggest that alcohol may contribute to some of these accidents. Wechsler et al. (1969) compared home accident admissions with emergency room admissions for other reasons, e.g., circulatory or digestive disorders. Twenty-two percent of 620 patients in home accidents had positive BACs compared with 9 percent of nonaccident patients. Eleven percent had blood alcohol concentration levels above .05 percent compared with 3 percent of nonaccident patients. The relationship was statistically significant even after adjusting for age, sex, and socioeconomic status of persons in each group.

In a review of the available literature, Wingard and Room (1977) identified only a half dozen U.S. reports since 1950 on alcohol and fatalities from falls, drowning, and fire-related injuries. Since studies showed a considerable variation in the proportion of fatal accident victims in whom alcohol was present, it is not clear whether alcohol is more likely to be involved in certain types of accidents.

Haberman and Baden (1978), in a report on 121 falling, fire, and drowning deaths in New York City between 1974 and 1975, indicated that 43 percent of such victims had blood alcohol concentrations above .10 percent. That proportion actually exceeded the proportion of motor vehicle and pedestrian accidents involving blood alcohol at that level during the same time period. Only about half the accidents in each of those categories during that time period were available for study. Yet, even if all the omitted accidents did not involve alcohol, 20 to 25 percent of the falls, burns, and drownings would still have occurred to persons with BACs above .10 percent. In contrast, on any given day one can expect only 3 percent of the population in a large urban area to consume the equivalent of five or more drinks, the amount needed to achieve a BAC of .10 percent (Hingson et al. 1981). However, the proportion of persons in similar social and demographic groups as those in accidents who consume that much has not been determined.

Alcohol's contribution to accidents may vary by type of accident. In some, such as falls, alcohol may reduce sensory motor coordination. In drownings, alcohol may contribute by increasing the risk-taking behaviors of swimmers or boaters or by reducing coordination so that capsizing is more likely. Once the person is in the water, alcohol may hasten a loss of body heat (Stanley and Siegal 1981). Alcohol may prompt smokers to fall asleep and risk cigarette fires (Levine and Radford 1977).

Some studies compare death rates from accidents of alcoholics or heavy drinkers with those of the rest of the population. When Brenner (1967), Nicholls et al. (1974), and Schmidt and de Lint (1972) compared alcoholic samples with matched age- and sex-specific populations, they found a 5 to 13 times greater risk of dying from falls among alcoholics. Schmidt and de Lint also reported a tenfold increase in death rates in fires among alcoholics. Social class and possible living environment differences between the groups were not, however, controlled in these studies. In the study of 2,015 Kaiser Permanente Health

Plan members (Klatsky et al. 1981), heavy drinkers were 2.7 times as likely as the overall sample to die in an accident (other than traffic accident) during the subsequent 10 years. Specific comparisons on the types of accident were not made and none of these studies examined alcohol consumption immediately prior to the accident.

Of course, using alcohol or being alcoholic is only one of many factors that contribute to such accidents, and its presence does not necessarily imply a causal role. Research is needed to assess when alcohol may have an independent effect beyond other factors such as age, architecture, poverty, poor housing, and smoking. The national fire survey does not query fire witnesses and victims about alcohol, even though drinkers are much more likely than nondrinkers to smoke and smoking is well known to relate to the incidence of fires (FEMA 1982). Case control studies that compare persons in falls with persons passing similar locations at similar times of day who do not fall would help establish the relative risk for intoxicated persons.

Alcohol and Crime

Homicide

In 1980, there were 22,516 murders in the United States or 9.8 per 100,000 inhabitants. The rate per 100,000 inhabitants rose 11 percent since 1976 (USDOJ 1981).

It has long been recognized that alcohol is frequently present in both perpetrators and victims. Wolfgang's landmark study (1958) in Philadelphia indicated that either the victim or the offender or both had been drinking in nearly two-thirds of the slayings he studied, and in 44 percent, both parties had been consuming alcohol.

Roizen and Schneberk (1977) identified 11 studies that reported a range of alcohol involvement in from 28 to 85 percent of arrested offenders. Greenberg's review (1981) of 28 studies indicated that a majority of victims or offenders or both drank prior to the crime. Reports of involvement varied somewhat by type of study. Studies of police reports and arrested populations identified alcohol in 66 percent of cases, whereas 9 prison studies identified its involvement in 44 percent of cases. In 4 of 5 studies reviewed by Greenberg (1981), at least 20 percent of the individuals who committed murder also had an alcoholism problem.

These findings have prompted some researchers to hypothesize a direct causal relationship between heavy alcohol consumption or alcoholism and murder. Numerous theories attempt to explain how alcohol may be directly related to violent crime (Pernanen 1981). To cite a few:

- Alcohol may reduce inhibition and release violent impulses.
- Alcohol may alter neurotransmitter activity.
- Chronic excessive alcohol use may lead to temporal lobe dysfunction, which in turn may contribute to violent behavior.

Still others argue that alcohol use contributes to violence only in the presence of such conditions as hypoglycemia, produced by poor nutrition among chronic heavy drinkers, or persistent lack of REM (rapid eye movement) sleep activity as a result of prolonged drinking. Either of these factors may produce heightened irritability among drinkers.

However plausible these theories may be, no studies to date provide clear evidence that alcohol is an independent causal factor in murder. According to Collins (1981), "the number of scientists arguing for direct and simple relations between the presence of alcohol and crime has decreased as the complexity of logic and sophistication of designs has increased." MacAndrew and Edgerton (1973), using cross-cultural analyses, suggest that behavior while intoxicated may depend more on what is considered socially acceptable while intoxicated than on disinhibitory effects.

Several researchers have also noted that some variables simultaneously contribute to both alcohol use or alcoholism and crime, possibly making the alcohol-crime relation coincidental rather than causal. For example, heavy drinking is more apt to occur in marital situations in which neither partner is satisfied or happy (Hingson et al. 1981). While such situations may also be more likely to result in murder, it is difficult to say whether alcohol and/or the underlying unhappiness contributed to such murders.

Similarly, Wolfgang (1967) has cautioned that drinking is a part of the mores of low-income young males, the group involved in most murders. But tension and frustration may also be more common among this subgroup, which in turn prompts some to commit murder as well as to drink. Also, alcohol use and vio-

lence are both more likely to be found in settings (e.g., bars) where low-income young males congregate. It is difficult to establish whether alcohol contributes to violence, or whether it at times even helps avert violence and murder by relieving tension and frustration.

Few studies in this area attempt to control for these possibly confounding factors. Of 35 studies on crime reviewed by Greenberg (1981), only 5 controlled for age, sex, and race, factors related not only to the incidence of murder but also to drinking.

Other methodologic limitations of the existing research on this topic have been discussed by Roizen and Schneberk (1977). Distinctions are seldom drawn between presence of alcohol and actual intoxication, and length of time since alcohol ingestion is rarely indicated. Data are collected at different points in the judicial process: arrest, court, and prison. The limited data available suggest that murderers who had been drinking and are caught are more likely to be convicted and hence overrepresented in prison populations (Greenberg 1981). In all studies of murder, the definition of the crime itself may be blurry; for example, manslaughter and homicide may be combined.

The presence of alcohol in the victim is not clearly and adequately understood. The few studies of victims suggest that they were frequently drinking. It is possible that drinking victims who had been drinking either lacked adequate judgment to avoid antagonizing the offender or were unable to modify the offender's anger in some way (Braucht et al. 1980). It is also possible that intoxication rendered victims less able than sober persons to defend themselves or to avoid an attack.

In studies of homicide and alcoholism, the presence of alcohol at the scene and time of the crime is seldom identified. Moreover, alcoholics are more likely to have court records such as DWI offenses, loitering, etc., and hence to be known by police. This has prompted some researchers to ask: Are alcoholic criminals more likely to get caught? (Roizen and Schneberk 1977).

It should be kept in mind that the majority of criminal offenders are not alcoholics and the majority of alcoholics never commit serious crimes (Collins 1981). Even explanations that identify alcohol as a potential contributor to violence cite other contributors as well, e.g., hereditary factors, brain dysfunction, availability of guns, media violence, and numerous other social and psychological factors

(Collins 1981; Menuck and Voineskos 1981). According to Blum (1981), "Under no circumstances will alcohol be a sole cause of violence."

In sum, alcohol use is part of the lifestyle of low-income young males, those most apt to commit or be involved in murder. Even if drinking may have disinhibitory effects or long-term effects on mental functioning that in turn contribute to violence, it is unrealistic to cite alcohol as the major cause of the frustration and anger in this population that also contribute to high murder rates. Nor, however, is it realistic to dismiss alcohol as irrelevant. Alcohol may alter perceptions, cognitive performance, moods, emotions, and response capabilities and preferences. The frequent association of alcohol among murderers and their victims suggests it is more than coincidental.

It is reasonable to expect that alcohol is more likely to contribute in some types of murders than in others, e.g., quarrels among family members, friends, and acquaintances that escalate into murder. Alcohol is less likely to be involved in premeditated murders in which the perpetrator makes careful plans to avoid detection. At this point, because of inherent difficulties in studying murder and limitations in available research about alcohol and murder, one can conclude only that alcohol may be one of numerous factors that at times may independently contribute to some types of murder.

Rape

In 1980, there were 36.4 forcible rapes reported per 100,000 persons in the population, or 82,088 rapes (USDOJ 1981). This rate has steadily increased 38 percent from 1976 to 1980 in part because of changing public and law enforcement attitudes, the push from women's groups to encourage victims to report rapes, and the general increase in violent crime in the United States.

Rape is an extremely difficult crime to investigate (Rabkin 1979). Local legal codes, police enforcement, and public attitudes toward rapists and victims vary greatly by region, resulting in different reporting and recording rates. Low reporting and conviction rates limit inferences about the characteristics of rapists. In general, studies of rape draw from interviews and other data collected from apprehended rapists, who may differ from undetected rapists.

In a study of violent crime in Western Canada (Gerson and Preston 1979), 60 percent of the rapes in a 12-month period were considered to be alcohol related; either the victim or the defendant had been drinking, and the crime involved violation of an alcohol statute or occurred in a licensed alcohol establishment. Groth (1979) found that 50 percent of a group of men who raped older victims reported being high on alcohol or on alcohol and drugs.

Rada and his colleagues (1978), in a study of 382 rapists in a State mental institution, found that 81 percent of the alcoholics and 25 percent of the nonalcoholics drank before the assault. In another population, 53 percent of mentally disturbed sex offenders reported drinking heavily (Rada et al. 1978). In contrast, in a general urban population 36 percent of males can be expected to drink on any given day (Hingson et al. 1981). A more appropriate comparison would match nonrapists with rapists on other social and economic characteristics to see whether their drinking levels differ.

Few studies have examined the related question of frequency of alcoholism among rapists. Rada et al. (1978) found that 33 percent of 77 rapists had a history of alcoholism. Twenty-seven percent of 82 rapists in another group of subjects (Barnard et al. 1979) were identified as alcoholics. The proportion of alcoholics in the general population is much lower.

These studies suggest that people who commit rape are disproportionately likely to have been drinking or to be alcoholics. However, the reasons for this alcohol-rape connection are not well understood. Wilson (1981) reviewed two major theories. Disinhibition theory suggests that alcohol releases otherwise well-controlled sexual or aggressive behaviors. An alternative explanation based on social learning principles suggests that behavior after consuming alcohol is determined by past learning about the effects of alcohol on actions and by personal expectations about the consequences of a certain course of action. A social learning model of alcohol-associated rape might suggest that alcohol consumption is seen as a way to disavow responsibility for actions and thereby avoid external punishment, social disapproval, or internal guilt for attempting rape.

In conclusion, it appears that alcohol consumption and rape may be associated, but this link may have been overstated. Some rapists may actually plead drunkenness or alcoholism as a way to avoid legal responsibility for the

crime. Thus, the role of alcohol in rapes is still poorly understood.

Other Crime

The relationship of alcohol to other crime is even less clear. Data on the characteristics of perpetrators should be considered with much greater caution than those of murder offenders since a lower proportion reach the attention of the police.

Fewer studies have been undertaken on alcohol involvement in other crimes than in murder. Table 1 indicates the relative alcohol involvement for these crimes in contrast to murder (Greenberg 1981). Compared with murder, the proportion of these crimes with alcohol involvement appears to be lower. Because, with the exception of aggravated assault, most of these crimes are premeditated and carried out with the intent of avoiding arrest, it is unlikely that alcohol will be as strong a potential contributor as in crimes of passion and violence.

Alcohol and the Family

Separation and Divorce

The annual divorce rate in 1977 in the United States among women over the age of 15 was 21 per 1,000. These rates have been climbing steadily since 1967 (USDHHS 1980). Evidence suggests that the rate of separation and divorce among alcoholics and their spouses is seven times that of the general population (Paolino and McCrady 1977). Forty percent of family court problems involve alcoholism in some way (Jacob and Seilhamer 1982), and it is estimated that 33 percent (Jacob and Seilhamer 1982) to 40 percent (USDHEW 1975) of intact alcoholic couples have poor marital relationships.

Alcohol is clearly associated with disrupted family functioning, but the marital and family problems may precede the alcoholism, may occur solely as a result of the alcoholic's drinking and behavior changes, or may be part of a complex interaction between preexisting weaknesses of the family or of individuals in the family and the drinking of one family member.

Family Violence

The abuse of children by their parents, and the abuse of spouses by their marital partners,

Table 1. Alcohol involvement in criminal events, by type of crime

Percentage of crime victims or offenders in whom alcohol was present during criminal event ^a	Frequency of studies							
	Homicide	Sexual offenses	Aggravated assault	Robbery	Burglary	Larceny	Auto theft	Forgery/ bad checks
0 - 10	2	-	-	-	-	1	-	-
11 - 20	4	-	1	1	1	-	-	-
21 - 30	-	2	2	-	-	-	-	-
31 - 40	2	1	-	-	2	-	-	-
41 - 50	2	3	1	-	-	1	1	1
51 - 60	4	1	-	1	1	1	1	2
61 - 70	4	1	3	2	1	1	1	-
71 - 80	2	-	-	1	-	1	-	-
81 - 90	7	-	-	-	1	-	-	-
91 - 100	1	1	1	-	-	-	-	-
Total studies ^b	28	9	8	5	6	5	3	3

^a These percentages reflect the presence of alcohol, as defined in a variety of ways, immediately prior to or during the crime. The number upon which the percentages are based are therefore either total cases (victims) or total offenders. In studies in which drinking on the part of both victims and offenders is reported, the percentage of victims only is reported in this table.

^b This table is based on the findings of 35 studies. The grand total does not add to 35 because some studies include data on only one type of crime, while others include two or more types of crime and therefore appear in more than one column.

Source: Greenberg 1981.

have received increasing attention in the media and in the professional literature. Self-help groups for parents who abuse their children (e.g., Parents Anonymous) and shelters for battered women have proliferated throughout the country.

The study of family violence, however, is an extremely sensitive, emotionally laden, and difficult pursuit. The definitions of abuse vary widely, and incidents of abuse are markedly unreported. Research in this area is hampered by the traditional sanctity of the family and the blurred lines between acceptable family behavior and abuse.

Most studies that have attempted to understand causes for family violence, or the links between violence and alcohol use, have

sampled individuals in treatment. Such individuals represent only that fraction of the abusing population who have admitted they have problems, or whose abuse has become severe enough to come to the attention of outside authorities, who required that they undergo treatment.

Finally, many attributions of violence are based on retrospective reports of the abuser, the victim, or a third party. Recall may be faulty, making attribution of violence to drinking or drunkenness suspect. One party may place primary emphasis on drinking that another would see as immaterial, or alcohol use may be presented as an excuse for violent behavior. Aarens et al. (1977) noted in one study of domestic violence that the com-

plainant stated that the abusive party was drinking or drunk much more often than was noted by the police. Finally, alcohol consumption may be irrelevant or coincidental or may have created or contributed to a conflict that culminated in violence.

Child abuse.--This discussion does not consider child neglect or failure to provide necessary food, shelter, or medical care but instead focuses on willful commitment of injury to the abused person. A national survey conducted in 1965 revealed that 2 to 4 percent of the adults surveyed personally knew families in which child abuse had occurred in the previous year (Gil 1970). Three literature reviews concerning drinking, alcoholism, and child abuse (Aarens et al. 1977; Hamilton and Collins 1981; Orme and Rimmer 1981) identified only three studies that determined the frequency of drinking during actual episodes of child abuse. Two of these note that 12 percent of abusers were drinking at the time (Gil 1970; Thomson et al. 1971), while only 3 to 6 percent were drinking in the third study (Scott 1973).

When considering the frequency of alcohol problems or alcoholism in child abuse cases, reports range from "no association" to 38 percent of abusers' having alcohol problems. The most frequently reported figures are that between 11 and 17 percent of abuse cases involve alcoholism.

Overall, the currently available evidence does not indicate a strong association between drinking or alcoholism and child abuse. As Orme and Rimmer (1981) concluded, "When estimates of alcoholism and alcohol problems in the general population were available, the prevalence appeared to be almost identical to that in child abusers" (p.284).

Spouse abuse.--Figures on spouse abuse tend to focus almost exclusively on the abuse of women by their husbands or lovers. During 1978, 6.6 percent of murder victims were women killed by either their husbands or their lovers (USDOJ 1981). Hamilton and Collins' (1981) review of 15 studies suggested that 17 to 35 percent of divorces involve violence.

Hamilton and Collins (1981) reported that most studies cite alcoholism or excessive drinking in 45 to 60 percent of spouse abuse. Most of these reports of alcohol problems come from the female victims of the abuse. These women may exaggerate or overestimate the seriousness of their partners' drinking or may prefer to believe their mates have a

drinking problem rather than accept other reasons for the abuse (Aarens et al. 1977; Hilberman 1980). Estimates of actual alcohol consumption at the time of abuse vary from 29 to 71 percent, although virtually all these figures are based on victim recall and were not verified by objective means such as BAC analyses.

Several theories have tried to explain the role of alcohol in spouse abuse. Gelles and Straus (1979) have suggested that alcohol is used as an excuse for violence because, when drunk, a person may deny responsibility for his or her actions. Denial of responsibility for actions when drinking is common in American society (Richardson and Campbell 1980). Social learning theorists (Wilson 1981) suggest that people learn to expect to behave more aggressively when drinking and therefore do so, even if the alcohol itself does not have a direct disinhibitory effect on their behavior. These two models are in direct contrast to disinhibition theory, which suggests that alcohol directly disinhibits people so that they express their real, underlying aggressive impulses. A fourth model suggests an interactive relationship between drinking and violence (Gelles 1972; Pernanen 1976). Arguments about a husband's drinking may escalate into arguments about many areas of marital problems. The alcohol may diminish the drinker's ability to reason and to control his behavior, resulting in a growing conflict that may culminate in violence.

Child Functioning in Alcoholic Families

The complexity of individual, family, and social factors that interact in a child's development makes it difficult to attribute a child's particular problems or personality solely to a parent's alcoholism. The study of children of alcoholics is further complicated by the methodologies usually employed. Children in treatment often form the basis of research samples, but they reflect only the fraction whose problems are serious enough to come to the attention of treatment agencies and who are able to find such agencies. Children whose parents are in alcoholism treatment are another common yet not fully representative sample, as many people with serious drinking problems never reach treatment (Hingson et al. 1981, 1982b). Attempting to gain access to children and their families who are not connected to the treatment or social services system, however, raises diagnostic and ethical issues. When asked about parents'

drinking, for example, a child may feel he or she is betraying the parent or may fear retaliation and therefore minimize parental drinking. Even if the situation is accurately identified, obtaining parental permission for the child to participate in a research study may be difficult. Ethical questions arise also if such children in the course of a study become labeled by a social institution (e.g., their school) as socially deviant by virtue of being the children of alcoholics.

Woodside (1982) has reviewed the available data on children of alcoholics. Hyperkinesis has been suggested as one consequence of parental alcoholism. Although early studies suggested that alcoholism was twice as frequent among parents of hyperkinetic children as among normal controls, more recent data fail to support this finding (Morrison 1980; Stewart et al. 1980). Children with at least one alcoholic parent are more likely to have a childhood history of school problems and anti-social behavior than other children (Sher and McCrady in press). Other studies reviewed by Woodside (1982) have found lower self-esteem, more emotional detachment, more anxiety symptoms, more social aggression, or more psychosomatic symptoms (such as headaches or stomach aches) among children of alcoholic parents than among control children. Most of these associations have not been replicated, however, and do not explore what mechanism may lead to the childhood problems or what protects some children from such problems.

Summaries of unstructured or semistructured interviews with children of alcoholics (Cork 1969) suggest that these children feel rejected by their parents, guilty or somehow responsible for the parent's alcoholism, resentful of the alcoholic parent, and in constant fear. However, most of these studies focus on the casualties of alcoholism--the children in trouble. One notable exception sought to understand what factors spare certain children (Wolin et al. 1980). They found that alcoholic families that maintained some predictable structures, particularly around the observance of family rituals (at meals, holidays, etc.), were less likely to produce alcoholic offspring than families with disrupted family rituals.

Alcohol and Suicide

About 27,000 Americans commit suicide each year (Hendin 1982), but 10 times as many persons make unsuccessful suicide attempts. The overall rate of suicide, at 12.5 per 100,000

persons, remained fairly stable from the 1940s to the 1960s and has increased only slightly since then. Suicide among young people 15 to 24 years of age has increased dramatically, however, by 250 percent among young women and more than 300 percent among young men. There are 100 to 200 suicide attempts for every completed suicide of a high school youth.

The other high-risk population group includes white men over 45 years of age, who commit 30 percent of all suicides each year, even though they represent only 10 percent of the population. Men who are alone or have suffered recent losses are at particular risk. Black men are at their highest risk for suicide between the ages of 20 and 34, rather than when they are older (Hendin 1982).

In some situations, alcoholism may contribute to a number of problems that may in turn precipitate suicide. Separation and divorce rates are high among alcoholics (Paolino et al. 1978). Depression is common among alcoholics during prolonged drinking bouts as well as during some phases of alcohol withdrawal, and alcoholism also causes numerous debilitating and life-threatening medical problems; suicide may become a response to any of these problems.

Because suicide has many causes, however, it is difficult to discern the contributory role of alcoholism when it coexists with other problems. As many as 25 percent of female alcoholics, for example, have diagnosable depression (Schuckit and Morrissey 1979). If such a patient commits suicide, it is impossible to determine to what the suicide should be ascribed--depression or alcoholism.

Individuals use alcohol in an attempt to cope with life problems. A person facing a divorce, the death of a parent, or a serious illness may drink and, in fact, may develop a serious drinking problem. If this person commits suicide, is the suicide a result of the alcohol abuse or of the original life problem?

A number of problems with research methods further muddy the waters. Social pressures often lead to underreporting of suicide, and family members may be reluctant to acknowledge the alcoholism of a deceased relative, thus obscuring the suicide-alcoholism association. Diagnosing alcoholism based only on survivors' reports is also difficult, especially when attempting to distinguish between alcoholism and other psychological disorders. Blood alcohol levels are not always determined when someone dies. Finally, the assessment of intent of a death is sometimes problematic.

For example, a person who dies from combined drug and alcohol ingestion may have planned his or her death, or may have been unaware of the way the substances could potentiate each other. Likewise, a person who dies in a single-car accident after drinking might have deliberately caused the accident or may have simply lost control of the vehicle.

Keeping in mind these research limitations, the Fourth Special Report (USDHHS 1981) cited studies indicating between 15 and 64 percent of suicide attempters and up to 80 percent of suicides had been drinking at the time of the attempt. The wide range of percentages makes it difficult to assess whether drinking is in fact causally linked with attempts. A review of violent deaths in the Cleveland area (Ford et al. 1979) found alcohol present in 25 percent of suicide victims. Of course, determining whether drinking is independently associated with suicide would require identifying populations of similar socioeconomic status, age, race, and sex as those who commit suicide. It is particularly important to compare populations with similar sociodemographic characteristics, as suicides occur disproportionately among the young and among older single males. The presence of alcohol in the blood stream of a suicide attempter at the time of the act provides insufficient data to conclude that alcohol is independently related to the attempt. Evidence is available that alcoholism or a history of drinking problems is more common among suicide attempters. Roizen (1982) indicates that most studies reported that more than 10 percent of attempters and completers had drinking problems.

Aarens et al. (1977) provide stronger data, comparing the proportion of problem drinkers or alcoholics in a random sample survey with Beck and associates' (1975) sample of suicide attempters in two large metropolitan hospitals. The survey data were standardized by age, sex, and marital status to match the suicide population. These data indicated that suicide attempters were four to six times more likely than the general population to report being problem drinkers or alcoholics.

Several studies have also shown that, as a group, alcoholics are more likely over the course of their lifetimes to commit suicide. Reviewing reports from 1935 to 1970, Miles (1977) estimated that 15 percent of alcoholics ultimately complete suicide. Another review suggests that 6 to 12 percent of alcoholics commit suicide, compared with 1 percent of the population. Klatsky et al. (1981) reported

that persons who consume more than six drinks a day as a group were twice as likely to die from suicide over a 10-year followup period than age- and sex-matched light drinkers and abstainers. It was not indicated whether these people were drinking at the time, but suicidal actions are more likely to be damaging when people have been drinking; combinations of alcohol with other drugs often have greater lethal potential (Eckardt et al. 1981).

Economic Costs of Alcohol Abuse

Alcohol and Work Productivity

Foregone work productivity is widely accepted to be an important component of the costs of alcohol abuse. The proliferation of occupationally based alcoholism programs may in part be attributed to increased employer awareness of excessive alcohol use and compromised job performance. No one has been able to precisely identify the worker productivity costs of alcohol abuse; the majority of studies have examined simple measures such as absenteeism.

A study of alcohol problems in seven railroad companies examined absenteeism as well as several empirical measures of lost productivity. Problem drinkers had almost twice the absenteeism of other workers and cost the company an estimated \$3.1 million, assuming 5.2 extra days of absence per year for the 8,670 employees for whom adequate attendance records were available and based on an average employee salary of \$18,000 (Mannello 1979).

Cost-benefit studies of occupational alcoholism programs generally support the claim that productivity losses attributable to alcoholic employees can be reduced. These studies generally focus on absenteeism rates among a treated group of alcoholic workers before and after an intervention. For example, Wood (1980) suggested that an occupational alcoholism program sponsored by New York Telephone Company saved the firm \$1.5 million, assuming that untreated alcoholics would have been absent 60 days a year (and also that they would have incurred \$2,000 in extra treatment costs). Jones (1977) attributed to Kennecott Copper Company's alcoholism program a 52-percent improvement in attendance, on average, among the 150 men who spent 12.7 months in treatment. Saxe et al. (1983) have recently reviewed similar studies of programs in the Philadelphia fire and police forces

(Jones and Vischi 1979), General Motors, and the U.S. Air Force (Orvis et al. 1981). All the studies indicate that reduced absences and cost-effectiveness are associated with those programs.

These studies focus primarily on absenteeism and illness costs (not productivity or working days), and they lack adequate controls that would suggest causal inferences. They explore only identified alcoholic employees and leave unresolved the crucial question of how these employees would have fared in the absence of treatment. Whether problem drinking causes substandard job performance or, conversely, substandard, unproductive, and hence unrewarded job performance contributes to problem drinking remains unanswered. Development of more precise estimates concerning the amount of work productivity lost because of drinking and the proportion that can be regained if employees with problems are identified and offered assistance should be a research priority in the 1980s.

Estimating the Costs of Alcohol Abuse

Given the numerous social problems associated with or caused by alcohol abuse, some attempts have been made to quantify its economic costs to society. Two major estimation procedures have been used. The population-specific approach (Berry and Boland 1977; Berry et al. 1977) compared per capita health care costs of alcohol abusers with those of nonabusers and multiplied the difference by an estimate of the prevalence of alcohol abuse, yielding a total cost of \$42.8 billion in 1975. This approach has been criticized because it fails to make corrections for other health risks more common among heavy drinkers. This would tend to inflate the estimates (Cruze et al. 1981).

The alternative approach identifies specific events or illnesses associated with alcohol abuse. For example, in calculating treatment costs, the illness-specific approach includes all expenditures for illnesses attributable solely to alcohol abuse and, for illnesses with multiple causes, only those expenditures that can be ascribed to alcohol abuse. This system would tend to produce a more conservative estimate.

Using this latter approach on 1977 data, Cruze et al. (1981) estimated the national economic costs of alcohol abuse at \$49.4 billion. "Direct core costs" (an estimated \$6.4 billion in 1977) are expenditures for hospitalization, outpatient care, professional ser-

vices, drugs and sundries, and other outlays involved in the diagnosis, treatment, continuing care, rehabilitation, and terminal care for alcohol-related illness and trauma. "Indirect core costs" (estimated for 1977 at \$36.8 billion) reflect the losses of productivity associated with alcohol-related morbidity (\$26.1 billion) and mortality (\$10.7 billion). Table 2 specifies the components of direct and indirect core costs.

"Other related costs," \$6.2 billion, include costs associated with motor vehicle crashes and fires and a proportionate share of society's expenditures for administering the systems of criminal justice, social welfare, fire protection, and highway safety. Also counted are productivity foregone by family and friends of alcoholics, productivity lost by 83,700 individuals in prisons in 1977 for crimes caused by alcohol abuse, and losses associated with automobile accidents experienced by individuals other than crash victims. Table 3 summarizes the breakdown of these other related costs.

Cruze et al. (1981) specified that when data are limited or unavailable, their estimates tend to err on the conservative side. When causal relations are imperfectly demonstrated between alcohol abuse and associated economic costs, on the other hand, their estimates tend to overstate the real costs. The issue of establishing causality was flagged by an advisory panel as "one of the most important factors in carrying out this study.... Failure to recognize the difference between causality and association will lead to unrealistically high estimates of the cost to society of alcohol abuse" (Cruze et al. 1981, p.D-3). The investigators nonetheless contend that, overall, their estimates "lean strongly in the conservative direction" (Cruze et al. 1981, p.6-1).

It is generally recognized that the total burden on society of alcohol abuse and alcoholism is substantially greater if social costs are considered. These include the pain and suffering of the alcohol abuser's family and a host of medical, psychological, and social consequences that a full accounting of costs would ultimately have to include in one way or another. Few studies do more than grant that these intangible costs would add billions of dollars to the overall economic costs to society, assuming they could be measured accurately in monetary terms (Cruze et al. 1981).

Nor has previous research ventured far into the analysis of the benefits of alcohol consumption, which from the standpoint of public

Table 2. Estimated core costs for alcohol abuse, 1977
(in \$ millions)

Total core costs	\$43,161
Direct	\$ 6,372
Treatment	5,637
Alcohol-abuse-specific illness	
—specialty setting	707
Alcohol-abuse-specific illness	
—general setting	2,001
Alcohol-abuse-related illness	
categories	1,711
Gastrointestinal tract	220
Liver disease	181
Nervous system	6
Heart	17
Endocrine system	608
Nutritional deficiency	108
Cancer	171
Mental disorder	293
Infectious disease	107
Alcohol-abuse-related trauma	1,217
Support	735
Research	28
Training and education	225
Construction	193
Health insurance administration	289
Indirect	\$36,789
Mortality	10,715
Direct primary causes—alcohol psychosis, alcoholism, alcoholic cirrhosis of liver, alcohol poisoning	2,617
Direct secondary causes—cirrhosis of liver—other, malignant primary liver neoplasms, other malignant neoplasms of gastrointestinal tract, pancreatitis, respiratory tuberculosis, other associated diseases	1,063
Indirect causes—motor vehicle crashes, falls, fires, other accidents, homicides, suicides	7,035
Morbidity	26,074
Lost productivity	23,593
Males	20,178
Females	3,415
Lost employment	2,481
Trauma	545
Residential treatment	328
Long-term disability	1,608

Source: Cruze et al. 1981.

policy could be considered relevant. Apart from the personal pleasures that prompt so many people to drink in the first place, the alcoholic beverage industry does make a substantial contribution to the economy. The Distilled Spirits Council of the United States, Inc., an association of producers, importers, and other industry representatives, reports that the industry paid \$15.7 billion in 1981 in taxes and other fees to Federal, State, and local governments and employed 826,000 people in 400,000 firms (DISCUS 1982). Additionally, as Gerstein (1981) points out, sales of alcoholic beverages provide not only contributions to government revenues and jobs in the economy, but also an important profit margin for many businesses.

The question for our society seems to be not whether the benefits outweigh the costs, but rather what can be done to reduce the considerable expense of alcohol abuse in terms of human lives, illness, and suffering.

Summary

In 1977, the last year for which there are reliable estimates, alcohol cost the Nation approximately \$50 billion—about \$26 billion in lost employment and productivity, \$17 billion in health care, and \$7 billion in property loss and crime. Although the costs are almost certainly much higher now after 5 years of inflation and sharply increased health care costs, alcohol's impact transcends monetary loss. In many fatalities, alcohol is an accessory before the fact.

Traffic accidents, especially those involving alcohol, continue to be a major problem in our society. According to one estimate, if no drivers drank in 1977, almost 12,000 deaths and perhaps 300,000 injuries would have been prevented. Drivers who drink in excess of the legal intoxication level are 3 to 15 times as likely as nondrinking drivers to be involved in a fatal crash. Even moderate drinking at legal levels can increase the likelihood of fatal accidents. Furthermore, pedestrians killed in traffic accidents are 3 times more likely to be intoxicated than noninjured pedestrians.

Although accidents in the home and workplace have not been as extensively researched as traffic accidents, evidence is ample and growing that alcohol contributes significantly to their incidence and severity. People who drink on the job are much more likely than their coworkers to have fatal accidents at work. Results indicate that alcoholics and

Table 3. Estimated other related costs
for alcohol abuse, 1977
(in \$ millions)

Total other related costs	\$ 6,213
Direct	\$ 4,441
Motor vehicle crashes	1,782
Crime	1,685
Public criminal justice system	1,479
Law enforcement	313
Legal and adjudication	139
Corrections	1,027
Private criminal justice system	191
Law enforcement	184
Legal and adjudication	7
Corrections	-
Property loss/damage	15
Social welfare programs	142
Fire losses	319
Fire protection	482
Highway safety	31
Indirect	\$ 1,772
Crime careers	-
Incarceration	1,418
Homicide	276
Felonious assault	120
Robbery	46
Burglary	49
Drug laws	0
Driving under the influence	287
Liquor laws	158
Public drunkenness	461
Other	21
Motor vehicle crashes	354

Source: Cruze et al. 1981.

heavy drinkers tend to be involved in fatal falls, drownings, and fires.

More alcoholics and heavy drinkers than abstainers are involved in homicides, as both victims and offenders. The alcohol association is somewhat weaker for other crimes such as rape, theft, robbery, and assault. While there are some statistical links between alcohol and crime, factors other than alcohol may explain the relationship.

Heavy drinking seems to affect family stability profoundly. In families with at least one alcoholic spouse, the rate of separation and divorce is seven times that of the general population. Although some researchers have speculated that the incidence of child abuse and spouse abuse may be alcohol-related, re-

liable data are difficult to collect. Suicides are higher among alcoholics, but drinking may be a result of depression or other problems that in turn lead to suicide.

More carefully designed studies are required to establish unequivocally the potential adverse consequences of alcohol. Meanwhile, there is clear reason for concern about the social effects of excessive alcohol consumption.

References

- Aarens, M.; Cameron, T.; Roizen, J.; Roizen, R.; Room, R.; Schneberg, D.; and Wingard, D. Alcohol, Casualties and Crime. Alcohol, Casualties and Crime Project Final Report. Report No. C-18. Berkeley: Social Research Group, University of California, 1977. 709 pp.
- Baker, S.P.; Samkoff, J.S.; Fisher, R.S.; and Van Buren, C.B. Fatal occupational injuries. Journal of the American Medical Association 248(6): 692-697, 1982.
- Barnard, G.W.; Holzer, C.; and Vera, H. Comparison of alcoholics and nonalcoholics charged with rape. Bulletin of the American Academy of Psychiatry and the Law 7:432-440, 1979.
- Barry, H. Motivational and cognitive effects of alcohol. In: National Highway Traffic Safety Administration. Alcohol, Drugs, and Driving, Perrine, M.W., ed. Report No. DOT HS-801 096. Washington, D.C.: NHTSA, 1974. pp. 71-105. NTIS No. PB-232-111.
- Beck, A.T.; Kovacs, M.; and Weissman, A. Hopelessness and suicidal behavior: An overview. Journal of the American Medical Association 234(11):1146-1149, 1975.
- Berry, R.E., Jr., and Boland, J.P. The Economic Cost of Alcohol Abuse. New York: The Free Press, 1977. 200 pp.
- Berry, R.E., Jr.; Boland, J.P.; Smart, C.N.; and Kanak, J.R. The Economic Cost of Alcohol Abuse - 1975. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1977. 222 pp. NTIS No. PB-278-606.
- Blomberg, R.D.; Preusser, D.F.; Hale, A.; and Ulmer, R.G. A Comparison of Alcohol Involvement in Pedestrians and Pedestrian Casualties. Report No. DOT HS-805 249. Washington, D.C.: National Highway Traffic Safety Administration, 1979. NTIS No. PB80-166275.
- Blum, R.H. Violence, alcohol and setting: An unexplored nexus. In: Collins, J.J., Jr., ed. Drinking and Crime: Perspectives on the Relationships Between Alcohol Consumption and Criminal Behavior. New York: Guilford Press, 1981. pp. 110-142.
- Borkenstein, R.F.; Crowther, R.F.; Shumate, R.P.; Ziel, W.B.; and Zylman, R. The role of the drinking driver in traffic accidents (The Grand Rapids study). Blutalkohol 11(Supplement 1):1-132, 1974.

- Braucht, G.N.; Loya, F.; and Jamieson, K.J. Victims of violent death: A critical review. Psychological Bulletin 87(2):309-333, 1980.
- Brenner, B. Alcoholism and fatal accidents. Quarterly Journal of Studies on Alcohol 28 (3):517-528, 1967.
- Cameron, T. Alcohol and traffic. In: Aarens, M.; Cameron, T.; Roizen, J.; Roizen, R.; Room, R.; Schneberk, D.; and Wingard, D. Alcohol, Casualties and Crime. Alcohol, Casualties and Crime Project Final Report. Report No. C-18. Berkeley: Social Research Group, University of California, 1977. pp. 120-288.
- Collins, J.J., Jr. Alcohol use and criminal behavior: An empirical, theoretical, and methodological overview. In: Collins, J.J., Jr., ed. Drinking and Crime. Perspectives on the Relationships Between Alcohol Consumption and Criminal Behavior. New York: Guilford Press, 1981. pp. 288-316.
- Cork, M. The Forgotten Children. Toronto: Addiction Research Foundation, 1969.
- Cruze, A.M., Harwood, H.J.; Kristiansen, P.L.; Collins, J.J.; and Jones, D.C. Economic Costs to Society of Alcohol and Drug Abuse and Mental Illness -- 1977. Rockville, Md.: Alcohol, Drug Abuse, and Mental Health Administration, 1981. 98 pp.
- Damkot, D.K.; Toussie, S.R.; Akley, N.R.; Geller, H.; and Whitmore, D. On the Road Driving Behavior and Breath Alcohol Concentration. Report No. DOT HS-802 264. Washington, D.C.: National Highway Traffic Safety Administration, 1977. 230 pp.
- Distilled Spirits Council of the United States, Inc. DISCUS Facts Book 1981. Washington, D.C.: DISCUS, 1982.
- Eckardt, M.J.; Harford, T.C.; Kaelber, C.T.; Parker, E.S.; Rosenthal, L.S.; Ryback, R.S.; Salmoiraghi, G.C.; Vanderveen, E.; and Warren, K.R. Health hazards associated with alcohol consumption. Journal of the American Medical Association 246(6):648-666, 1981.
- Federal Emergency Management Agency. Fire in the United States: Deaths, Injuries, Dollar Loss, and Incidents at the National, State, and Local Levels in 1978. 2d ed. Report No. FEMA-22. Washington, D.C.: FEMA, July 1982.
- Fell, J.C. Alcohol Involvement in Traffic Accidents: Recent Estimates From the National Center for Statistics and Analyses. NHTSA Technical Report No. DOT HS-806 269. Washington, D.C.: National Highway Traffic Safety Administration, 1982. 27 pp.
- Ford, A.B.; Rushforth, N.B.; Rushforth, N.; Hirsch, C.S.; and Adelson, L. Violent death in a metropolitan county. II. Changing patterns in suicides (1959-1974). American Journal of Public Health 69(5):459-464, 1979.
- Gallup, G. Alcohol Abuse: A Problem in One of Three American Families. Princeton, N.J.: The Gallup Poll, November 15, 1982.
- Gelles, R.J. The Violent Home: A Study of Physical Aggression Between Husbands and Wives. Beverly Hills: Sage Publications, 1972. 230 pp.
- Gelles, R.J., and Straus, M.A. Determinants of violence in the family: Toward a theoretical integration. In: Burr, W.R.; Hill, R.; Nye, F.D.; and Reiss, I.L., eds. Contemporary Theories About the Family. New York: The Free Press, 1979. pp. 549-581.
- Gerson, L.W., and Preston, D.A. Alcohol consumption and the incidence of violent crime. Journal of Studies on Alcohol 40(3):307-312, 1979.
- Gerstein, D.R. Alcohol use and consequences. In: Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. pp. 182-224.
- Gil, D.G. Violence Against Children: Physical Abuse in the United States. Cambridge, Mass.: Harvard University Press, 1970.
- Greenberg, S.W. Alcohol and crime: A methodologic critique of the literature. In: Collins, J.J., Jr., ed. Drinking and Crime: Perspectives on the Relationships Between Alcohol Consumption and Criminal Behavior. New York: Guilford Press, 1981. pp. 70-109.
- Groth, A.N. Older rape victim and her assailant. Journal of Geriatric Psychiatry 11:203-215, 1979.
- Haberman, P.W., and Baden, M.M. Alcohol, Other Drugs and Violent Death. New York: Oxford University Press, 1978. 134 pp.
- Haddon, W.; Valien, P.; McCarroll, J.R.; and Umberger, C.J. A controlled investigation of the characteristics of adult pedestrians fatally injured by motor vehicles in Manhattan. Journal of Chronic Diseases 14(6): 655-678, 1961.
- Haddon, W., and Blumenthal, M. Foreword. In: Ross, H.L. Detering the Drinking Driver. Legal Policy and Social Control. Lexington, Mass.: Lexington Books, 1981. pp. xiii-xxiii.
- Hamilton, C.J., and Collins, J.J., Jr. The role of alcohol in wife beating and child abuse: A review of the literature. In: Collins, J.J., Jr., ed. Drinking and Crime: Perspective on the Relationships Between Alcohol Consumption and Criminal Behavior. New York: Guilford Press, 1981. pp. 233-287.
- Hendin, H. Suicide in America. 1st ed. New York: W.W. Norton, 1982. 252 pp.
- Hilberman, E. Overview: The "wife-beater's wife" reconsidered. American Journal of Psychiatry 137:1336-1347, 1980.
- Hingson, R.; Heeren, T.; Kovenock, D.; Mangione, T.; Meyers, A.; Morelock, S.; Mucatel, M.; and Scotch, N. Impact of Maine's 1981 Operating Under the Influence Legislation. Submitted for publication, 1983.
- Hingson, R.; Heeren, T.; Mangione, T.; Morelock, S.; and Mucatel, M. Teenage driving after using marijuana or drinking and traffic accident involvement. Journal of Safety Research 13(1): 33-37, 1982a.
- Hingson, R.; Mangione, T.; Meyers, A.; and Scotch, N. Seeking help for drinking problems: A study in the Boston metropolitan area. Journal of Studies on Alcohol 43(3): 273-288, 1982b.
- Hingson, R.; Scotch, N.; Barrett, J.; Goldman, E.; and Mangione, T. Life satisfaction and drinking

- practices in the Boston metropolitan area. Journal of Studies on Alcohol 42(1):24-37, 1981.
- Jacob, T., and Seilhamer, R. The impact on spouses and how they cope. In: Orford, J., and Harwin, J., eds. Alcohol and the Family. New York: St. Martin's Press, 1982. pp. 114-126.
- Jones, K.R., and Vischi, T.R. Impact of alcohol, drug abuse and mental health treatment on medical care utilization: A review of the research literature. Medical Care 17(12, Supplement):1-82, 1979.
- Jones, O. Kennecott's INSIGHT Program. In: Schramm, C.J., ed. Alcoholism and Its Treatment in Industry. Baltimore: Johns Hopkins University Press, 1977. pp. 76-81.
- Jones, R.K., and Joscelyn, K.B. Alcohol and Highway Safety 1978: A Review of the State of Knowledge. Report No. DOT HS803 764. Washington, D.C.: National Highway Traffic Safety Administration, 1978. 123 pp.
- Klatsky, A.; Friedman, G.; and Sieglau, A. Alcohol and mortality: A ten-year Kaiser Permanente experience. Annals of Internal Medicine 95(2): 139-145, 1981.
- Levine, M.S., and Radford, E.P. Fire victims: Medical outcomes and demographic characteristics. American Journal of Public Health 67(11):1077-1080, 1977.
- MacAndrew, C., and Edgerton, R.B. Drunken Compartment: A Social Explanation. Chicago: Aldine Publishing Co., 1973. 197 pp.
- Mannello, T.A. Problem Drinking Among Railroad Workers: Extent, Impact and Solutions. Monograph Series No. 4. Washington, D.C.: University Research Corporation, 1979. 69 pp.
- McCarroll, J.R., and Haddon, W. A controlled study of fatal automobile accidents in New York City. Journal of Chronic Diseases 15: 811-826, 1962.
- Menuck, M., and Voineskos, G. The etiology of violent behavior: An overview. General Hospital Psychiatry 3(1):37-47, 1981.
- Miles, C.P. Conditions predisposing to suicide: A review. Journal of Nervous and Mental Diseases 164(4):231-246, 1977.
- Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. 463 pp.
- Morrison, J. Adult psychiatric disorders in parents of hyperactive children. American Journal of Psychiatry 137:825-827, 1980.
- Moskowitz, H. Alcohol influences upon sensory motor function, visual perception, and attention. In: National Highway Traffic Safety Administration. Alcohol, Drugs, and Driving, Perrine, M.W., ed. Report No. DOT HS-801 096. Washington, D.C.: NHTSA, 1974. pp. 49-69. NTIS No. PB-232-111.
- National Safety Council. Accident Facts 1981. Chicago: NSC, 1982.
- Nicholls, P.; Edwards, G.; and Kyle, E. Alcoholics admitted to four hospitals in England: General and cause specific mortality. Quarterly Journal of Studies on Alcohol 35(3):841-855, 1974.
- Orme, T.C., and Rimmer, J. Alcoholism and child abuse. A review. Journal of Studies on Alcohol 42(3):273-287, 1981.
- Orvis, B.R.; Armor, D.J.; Williams, C.E.; Barras, A.J.; and Schwarzbach, D.S. Effectiveness and Cost of Alcohol Rehabilitation in the U.S. Air Force. Report No. R-2813-AF. Washington, D.C.: U.S. Air Force, 1981. 216 pp. NTIS No. ADA-112-787.
- Paolino, T.J., Jr., and McCrady, B.S. The Alcoholic Marriage: Alternative Perspectives. New York: Grune & Stratton, 1977. 211 pp.
- Paolino, T.J., Jr.; McCrady, B.S.; and Diamond, S. Statistics on alcoholic marriages: An overview. International Journal of Addictions 13(8):1285-1293, 1978.
- Pernanen, K. Alcohol and crimes of violence. In: Kissin, B., and Begleiter, H., eds. Social Aspects of Alcoholism. The Biology of Alcoholism, Vol. 4. New York: Plenum Press, 1976. pp. 351-444.
- Pernanen, K. Theoretical aspects of the relationship between alcohol use and crime. In: Collins, J.J., Jr., ed. Drinking and Crime. Perspectives on the Relationships Between Alcohol Consumption and Criminal Behavior. New York: Guilford Press, 1981. pp. 1-69.
- Perrine, M.W. Alcohol influences upon driving related behavior: A critical review of laboratory studies of neurophysiological, neuromuscular, and sensory activity. In: National Highway Traffic Safety Administration. Alcohol, Drugs, and Driving, Perrine, M.W., ed. Report No. DOT HS-801 096. Washington, D.C.: NHTSA, 1974. pp. 13-48.
- Perrine, M.W.; Waller, J.A.; and Harris, L.S. Alcohol and Highway Safety: Behavioral and Medical Aspects. Report No. DOT HS-800 600. Washington, D.C.: National Highway Traffic Safety Administration, 1971. 308 pp.
- Quinn, R., and Shepard, L. The 1972-1973 Quality of Employment Survey: A Report to the Employment Standards Administration. Washington, D.C.: U.S. Department of Labor, 1974.
- Rabkin, J.G. The epidemiology of forcible rape. American Journal of Orthopsychiatry 49:634-647, 1979.
- Rada, R.T.; Kellner, R.; Laws, D.R.; and Winslow, W.W. Drinking, alcoholism and the mentally disordered sex offender. Bulletin of the American Academy of Psychiatry and the Law 6:296-300, 1978.
- Reed, D.S. Reducing the costs of drinking and driving. In: Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. pp. 336-387.
- Richardson, D.C., and Campbell, J.L. Alcohol and wife abuse: The effect of alcohol on attributions of blame for wife abuse. Personality and Social Psychology Bulletin 6(1):51-56, 1980.
- Roizen, J., and Schneberk, D. Alcohol and crime. In: Aarens, M.; Cameron, T.; Roizen, J.; Roizen, R.; Room, R.; Schneberk, D.; and Wingard, D. Alcohol, Casualties and Crime. Alcohol, Casualties and Crime Project Final Report. Report No. C-18. Berkeley: Social Research Group,

- University of California, 1977. pp. 289-465.
- Roizen, J. Estimating alcohol involvement in serious events. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM) 82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp.179-219.
- Ross, H.L. Detering the Drinking Driver: Legal Policy and Social Control. Lexington, Mass.: Lexington Books, 1982. 129 pp.
- Saxe, L.; Dougherty, D.; Esty, K.; and Fine, M. Effectiveness and Costs of Alcoholism Treatment. Health Technology Case Study 22. Report No. OTA-HCS-22. Washington, D.C.: Congress of the United States, Office of Technology Assessment, 1983. 108 pp.
- Schmidt, W., and de Lint, J. Cause of death of alcoholics. Quarterly Journal of Studies on Alcohol 33(1):171-185, 1972.
- Schuckit, M.A., and Morrissey, E.R. Psychiatric problems in women admitted to an alcoholic detoxification center. American Journal of Psychiatry 136:611-617, 1979.
- Scott, P.D. Fatal battered baby cases. Medicine, Science and the Law 13(3):197-206, 1973.
- Shain, M. Alcohol, drugs and safety: An updated perspective on problems and their management in the workplace. Accident Analysis and Prevention 14(3):239-246, 1982.
- Sher, K.J., and McCrady, B.S. The MacAndrew Alcoholism Scale: Severity of alcohol abuse and parental alcoholism. Addictive Behaviors, in press.
- Stanley, R.J., and Siegal, G.P. Death by drowning: An overview. Minnesota Medicine 64(5):295-297, 1981.
- Sterling-Smith, R.S. Psychosocial Identification of Drivers Responsible for Fatal Vehicular Accidents in Boston. Part I. Report No. DOT HS-801 915. Washington, D.C.: National Highway Traffic Safety Administration, 1976.
- Stewart, M.A.; DeBlois, C.S.; and Cummings, C. Psychiatric disorder in the parents of hyperactive boys and those with conduct disorder. Journal of Child Psychology and Psychiatry 21: 283-292, 1980.
- Thomson, E.M.; Paget, N.W.; Bates, D.W.; Mesch, M.; and Putnam, T.I. Child Abuse: A Community Challenge. Buffalo: Stewart Press, 1971. 169 pp.
- Trice, H.M. Alcoholic employees: A comparison of psychotic, neurotic, and "normal" personnel. Journal of Occupational Medicine 17(3):94-99, 1965.
- U.S. Department of Health and Human Services. National Estimates of Marriage Dissolution and Survivorship: United States Vital Statistics. DHHS Pub. No. (PHS) 81-1403. Series No. 3, Vol. 19. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1980.
- U.S. Department of Health and Human Services. Fourth Special Report to the U.S. Congress on Alcohol and Health. DeLuca, J.R., ed. DHHS Pub. No. (ADM)81-1080. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- U.S. Department of Health, Education, and Welfare. Second Special Report to the U.S. Congress on Alcohol and Health. Chafetz, M.E., ed. DHEW Pub. No. (ADM)75-212. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1975.
- U.S. Department of Justice, Federal Bureau of Investigation. Crime in the United States. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- U.S. Department of Transportation. United States Fatal Accident Reporting System. DOT HS 806 251, Washington, D.C.: the Department, Jan. 1983.
- U.S. National Transportation Safety Board. Briefs of Accidents as a Cause/Factor: U.S. General Aviation. Washington, D.C.: U.S. NTSB, 1979. NTIS No. PB-82-138-492.
- Volpe, J., Chairman. An Interim Report to the Nation of the President's Commission on Drunk Driving. Washington, D.C.: PCDD, 1982. 75 pp. and appendix.
- Wagenaar, A.C.; Douglass, R.L.; Compton, C.P.; and Pettis, L.C. Raising the Legal Drinking Age in Michigan and Maine. Report No. UM-HSRI-81-58. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1981. 367 pp.
- Wechsler, H.; Kasey, E.; Thum, D.; and Demone, H. Alcohol level and home accidents. Public Health Reports 84(12):1043-1050, 1969.
- Wilson, G.T. Effects of alcohol on human sexual behavior. In: Mello, N.K., ed. Advances in Substance Abuse: Behavioral and Biological Research. Vol. 2. Greenwich, Conn.: JAI Press, 1981. pp. 1-40.
- Wingard, D., and Room, R. Alcohol and home, industrial and recreational accidents. In: Aarens, M.; Cameron, T.; Roizen, J.; Roizen, R.; Room, R.; Schneberk, D.; and Wingard, D. Alcohol, Casualties and Crime. Alcohol, Casualties and Crime Project Final Report. Report No. C-18. Berkeley: Social Research Group, University of California, 1977. pp. 39-119.
- Wolfgang, M.E. Patterns in Criminal Homicide. Philadelphia: University of Pennsylvania, 1958.
- Wolfgang, M. A sociological analysis of criminal homicide. In: Wolfgang, M.E., ed. Studies in Homicide. New York: Harper and Row, 1967, pp. 15-28.
- Wolin, S.J.; Bennett, L.A.; Noonan, D.L.; and Teitelbaum, M.S. Disrupted family rituals: A factor in the intergenerational transmission of alcoholism. Journal of Studies on Alcohol 41(3):199-214, 1980.
- Wood, L.W. "Lifestyle Management Strategies at New England Telephone." Paper presented at Leadership Strategies-Health Conference, Project Hope, 1980.
- Woodside, M. Children of Alcoholics. Albany: New York State Division of Alcoholism and Alcohol Abuse, 1982. 50 pp.

Chapter VII

Treatment: Emerging Trends in Research and Practice

During the past decade, several issues surrounding the treatment of alcoholism have been subject to controversy. Does the unitary disease concept permit reliable diagnosis for the purposes of early detection and treatment planning? Is treatment effective and, if so, can the costs of treatment be contained? Are there distinct subgroups of alcoholic persons? Can treatment efficacy be improved by matching individual patients to specific types of treatment? Should abstinence be the singular goal of treatment or can some problem drinkers engage in nonabusive drinking? If some can, how can these individuals be identified? The issues raised by these questions imply much about the theoretical and practical challenges facing the treatment field.

This chapter will focus on the state of the art of alcoholism treatment, giving special attention to developments not covered in the Fourth Special Report (USDHHS 1981). These developments include innovations in diagnosis, improved procedures for screening and early intervention, new findings on the costs and benefits of treatment, a continuing debate on the use and viability of controlled drinking therapies, and emerging models of treatment efficacy. An encouraging development in recent years has been the enhanced investment in research and clinical training in the alcoholism field, and the concomitant emergence of a multidisciplinary research community actively involved in the problems of treating alcoholism. As the quality and sophistication of clinical research improve, it is likely that many of the unanswered questions will be solved.

Recent Advances in Diagnosis and Nomenclature

Diagnosis, the process of identifying and labeling specific disease conditions, uses precise attributes, or diagnostic criteria, to

classify a sick person as having a disease. The importance of diagnostic criteria derives from their usefulness in making clinical decisions, estimating disease prevalence, understanding etiology, and planning treatment. While the diagnosis of alcoholism may seem straightforward to the concerned layperson, it is in fact a complex process.

Limitations in current diagnostic procedures were recently reviewed by McIntosh (1982), who compared data collected in 31 studies of the prevalence of alcohol-related problems in general hospital populations. Variations in the use of diagnostic criteria, as well as lack of conceptual precision in differentiating alcohol dependence from related disabilities, have led to widely differing prevalence estimates of the number of alcoholic patients. In spite of the fact that these patients were in hospitals, most individuals with drinking problems had neither been diagnosed as such nor received appropriate treatment for their alcohol problems.

The traditional unitary disease concept of alcoholism has been challenged by the observation that there may be multiple patterns of dysfunctional alcohol use, which result in multiple kinds of disability. A corollary of the unitary disease concept has been the assumption that alcoholics could be clearly differentiated from nonalcoholics on the basis of their distinctive disease characteristics. Known as the binary classification rule, this assumption has led to the search for universal and singular criteria applicable to all alcoholics. This dichotomous approach has not been particularly helpful to programs interested in early intervention (namely, secondary prevention), nor has it been useful in differentiating prognoses within patient samples in ways that could clarify important questions about treatment efficacy. An alternative approach (Marlatt 1981) characterizes alcohol dependence along a continuum of severity, with no clear demarcation between the beginning of alcoholism and the end of social

drinking. An advantage of this approach is that early detection may be improved when levels of risk have been established.

An important development in the area of diagnosis has been the publication in 1980 of the third edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 1980), better known as DSM-III. As a major revision of the manner in which various disorders, including alcoholism, are diagnosed and classified, DSM-III introduced several innovations designed to address problems in earlier classificatory systems (Spitzer et al. 1980). In contrast to previous editions of DSM, alcoholism is now included within the separate category of substance use disorders rather than as a subcategory of personality disorder. Reflecting a trend toward increased semantic precision, the term "alcohol dependence" is used in preference to the more generic "alcoholism." In addition, a separate category of "alcohol abuse" is added to permit greater differentiation. As delineated in table 1, alcohol dependence is differentiated from alcohol abuse by the presence of tolerance or withdrawal symptoms. Both diagnoses include a pattern of pathological use or impairment in social or occupational functioning due to alcohol. Modeled after diagnostic procedures initially developed for research purposes (Feighner et al. 1972), these criteria provide a systematic, standardized approach to diagnostic decisionmaking.

DSM-III permits evaluation of the individual's condition in terms of five independent dimensions or axes. Axis I describes the major clinical syndromes, including substance use disorders, while Axis II is reserved for concomitant personality disorders. Axis III classifies physical disorders and conditions, a number of which can be coded as alcohol-induced, including alcohol intoxication, alcohol withdrawal, alcohol withdrawal delirium, alcohol hallucinosis, alcohol amnestic disorder, and dementia associated with alcoholism. Axis IV draws attention to the severity of psychosocial stress (e.g., occupational, interpersonal, physical) that may modify the course of the current disorder. Finally, Axis V permits the clinician to indicate the patient's highest level of adaptive functioning during the past year in terms of social relations, occupational functioning, and use of leisure time.

Preliminary evaluations of DSM-III suggest that it provides reliable and valid identification of alcoholics in clinical settings, but that it may be less useful in detecting alcoholics who are not institutionalized (Mulford

and Fitzgerald 1981; Helzer et al. 1981; Hesselbrock et al. 1982). Thus, while the DSM-III alcohol-related diagnoses contain many innovative features, the usefulness of this approach for early identification, epidemiological surveys, and treatment planning remains to be evaluated.

The recent development and successful field testing of two standardized interview schedules, the Schedule of Affective Disorders (Weissman et al. 1980) and the Diagnostic Interview Schedule (Helzer et al. 1981), represent a major advance in clinical diagnosis and psychiatric epidemiology. Both schedules provide objective, standardized procedures for diagnosing alcoholism and other clinical syndromes using DSM-III criteria. As these instruments are used with greater frequency to study clinical and population samples, they promise to advance basic knowledge about alcoholism and its relationships to psychiatric syndromes and personality disorders. Data from household surveys using the Diagnostic Interview Schedule in three National Institute of Mental Health Epidemiology Catchment Areas suggest that, at some time during their lives, one in seven adults 18 years of age or older met criteria for alcohol abuse or alcohol dependence.

A related development in the area of alcoholism diagnosis is the international program on diagnosis and classification sponsored jointly by the U.S. Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA) and the World Health Organization (WHO 1982). The purpose of this project is to recommend improvements in sections of the International Classification of Diseases (ICD) dealing with alcoholism, drug abuse, and mental health. The first phase was devoted to a systematic examination of concepts and nomenclature pertaining to alcohol and drug abuse (Edwards et al. 1981). Of particular interest to the alcoholism treatment field is the emerging set of concepts, definitions, and criteria that may well provide the basis for the next revision of ICD. Central to the WHO approach to alcoholism is the concept of a dependence syndrome that is distinguished from alcohol-related disabilities (Edwards et al. 1976, 1981). As outlined in table 2, the dependence syndrome is an interrelated cluster of cognitive, behavioral, and physiological symptoms. Alcohol-related disabilities, on the other hand, consist of those physical, psychological, and social dysfunctions that follow directly or indirectly from excessive drinking and dependence.

Table 1. DSM III criteria for alcohol abuse and alcohol dependence

Diagnostic criteria for alcohol abuse	Diagnostic criteria for alcohol dependence
A. Pattern of pathological alcohol use: need for daily use of alcohol for adequate functioning, inability to cut down or stop drinking, repeated efforts to control or reduce excess drinking by "going on the wagon" (periods of temporary abstinence) or restricting drinking to certain times of the day, binges (remaining intoxicated throughout the day for at least 2 days), occasional consumption of a fifth of spirits (or its equivalent in wine or beer), amnesic periods for events occurring while intoxicated (blackouts), continuation of drinking despite a serious physical disorder that the individual knows is exacerbated by alcohol use, drinking of nonbeverage alcohol. B. Impairment in social or occupational functioning due to alcohol use: violence while intoxicated, absence from work, loss of job, legal difficulties (e.g., arrest for intoxicated behavior, traffic accidents while intoxicated), arguments or difficulties with family or friends because of excessive alcohol use. C. Duration of disturbance of at least 1 month.	A. Pattern of pathological alcohol use: need for daily use of alcohol for adequate functioning, inability to cut down or stop drinking, repeated efforts to control or reduce excess drinking by "going on the wagon" (periods of temporary abstinence) or restricting drinking to certain times of the day, binges (remaining intoxicated throughout the day for at least 2 days), occasional consumption of a fifth of spirits (or its equivalent in wine or beer), amnesic periods for events occurring while intoxicated (blackouts), continuation of drinking despite a serious physical disorder that the individual knows is exacerbated by alcohol use, drinking of nonbeverage alcohol. B. Impairment in social or occupational functioning due to alcohol use: violence while intoxicated, absence from work, loss of job, legal difficulties (e.g., arrest for intoxicated behavior, traffic accidents while intoxicated), arguments or difficulties with family or friends because of excessive alcohol use. Either tolerance or withdrawal: C. Tolerance: need for markedly increased amounts of alcohol to achieve the desired effect, or markedly diminished effect with regular use of the same amount. D. Withdrawal: development of alcohol withdrawal (e.g., morning "shakes" and malaise relieved by drinking) after cessation of or reduction in drinking.

Source: American Psychiatric Association 1980.

According to the WHO dependence model, a complete description of an individual's alcohol-related pathology must include statements concerning the nature and severity of dependence, the kinds and degrees of disability, and the personal and environmental factors that influence the drinking problem. The WHO model departs significantly from DSM-III and the older binary classification schemes by em-

phasizing that both dependence and alcohol-related disabilities exist in degrees rather than in an all-or-none state.

In the brief period since it was first introduced, the dependence syndrome construct has received considerable attention from researchers and clinicians. A number of instruments have been developed to assess its usefulness (Stockwell et al. 1979; Skinner and

Table 2. Constituent elements of the alcohol dependence syndrome

Elements	Interpretation/examples
Narrowing of the drinking repertoire	The drinking pattern tends to become stereotyped around a regular schedule of almost continuous daily consumption.
Salience of drink-seeking behavior	Drinking is given higher priority than other activities in spite of its negative consequences.
Increased tolerance to alcohol	More and more alcohol is required to produce behavioral, subjective, and metabolic changes; large amounts of alcohol can be tolerated.
Repeated withdrawal symptoms	Tremulousness, sweatiness, nausea, etc., appear after short periods of abstinence.
Relief drinking	Withdrawal symptoms are relieved or avoided by further drinking, especially in the morning.
Compulsion to drink	Subjective awareness of craving for alcohol exists, as well as impaired control over quantity and frequency of intake.
Readdiction liability	The syndrome tends to be rapidly reinstated when drinking is recommenced after a period of abstinence.

Source: Adapted from Edwards et al. 1976, 1981.

Allen 1982; Hesselbrock et al. 1983). Research in both experimental and clinical settings suggests that the severity of dependence can correlate positively with attendance at a treatment clinic (Skinner and Allen 1982), cravings for alcohol after a "priming" drink (Kaplan et al. 1983), and failure to control drinking following relapse (Orford et al. 1976; Polich et al. 1981). While further research is needed, the dependence syndrome construct shows considerable promise for early detection, diagnosis, and treatment planning.

Differential Assessment

Both DSM-III and the WHO dependence model represent a general movement toward differential assessment as evidenced by the use of multiple criteria to provide a comprehensive evaluation for treatment planning. Ideally, specificity of diagnosis should lead to

specificity of treatment, an important goal in the delivery of treatment services for three essential reasons: (1) economy of cost, (2) avoidance of inappropriate or ineffective treatment, and (3) increased efficacy. Surprisingly, outside the area of personality assessment, little interest has existed until recently in differential assessment of the alcoholic person. This is due in part to assumptions about the unitary nature of alcoholism, and in part to the limited methods available to evaluate alcoholics.

Recent advances in evaluation methodology (Meyer et al. 1981) have stimulated research in this area, and a number of assessment procedures have been developed. In addition to those already discussed, several investigators (Skinner and Allen 1982) have demonstrated the usefulness of assessing drinking patterns and alcohol-related problems in an effort to identify individual differences related to

treatment outcome. Other instruments have been developed recently to provide differential assessment of stages of alcoholism (Mulford 1980), client liabilities associated with poor treatment response (Costello and Baillargeon 1981), severity of addiction-related problems (McLellan et al. 1980), and stability of the male alcoholic's marital situation (O'Farrell et al. 1981). With the burgeoning application of computer technology to individual testing and statistical data analyses, the benefits of differential assessment may well become routine in clinical settings.

Screening and Early Detection of Alcoholism

Consonant with recent conceptual developments in the diagnosis of alcoholism, there has been increasing interest in the development of a simple and accurate screening procedure that could facilitate early identification of alcohol dependence. Clearly, early casefinding can improve the effectiveness of treatment and reduce its costs. Research on early identification has progressed on two relatively independent planes: (1) the search for biochemical markers for alcoholism and (2) the elucidation of psychosocial indicators.

Biochemical Markers

The accumulation of research findings, some of which have already been noted in previous chapters of this report, suggests that several laboratory tests may be useful in the early detection of alcohol abuse. Serum gamma-glutamyl transpeptidase (GGTP) has been suggested as an indicator of heavy alcohol consumption among problem drinkers (Reyes and Miller 1980). Although its usefulness may be affected by concurrent liver pathology, drug use, and individual differences among heavy drinkers (Garvin et al. 1981), its discriminative ability is enhanced considerably when interpreted in conjunction with mean corpuscular volume (MCV) and other tests (Mayfield and Johnston 1980; Chick et al. 1981). That routine blood chemistries may be a useful adjunct for detecting alcoholism is suggested by the results of several studies (Ryback et al. 1980, 1982) that have found that the profiles of 25 biochemical tests differentiated between known alcoholics and nonalcoholics with a high degree of accuracy.

Psychosocial, Clinical, and Combined Assessments

Because some psychosocial symptoms (e.g., escape drinking, concern about a drinking problem, alcohol-related accidents) may appear relatively early in the alcoholic's drinking career, their assessment by means of interviews or self-administered questionnaires has received increasing attention by researchers interested in screening and early diagnosis (Skinner et al. 1981).

Clearly, the most investigated self-administered diagnostic instrument continues to be the Michigan Alcoholism Screening Test (MAST). Consisting of 25 true/false statements describing the typical medical, social, and behavioral problems associated with excessive drinking, the MAST has demonstrated a considerable degree of validity in distinguishing between known groups of male alcoholics and male nonalcoholics (Brady et al. 1982). Concurrent with validation of the MAST has been the development of a variety of similar screening instruments (Brown and Lyons 1981; Skinner et al. 1981). These tests are rapid, inexpensive, and relatively accurate when used under proper conditions. Their simplicity, or gender specificity, however, may limit their usefulness for general population screening, particularly as the obvious intent of the questions leaves them vulnerable to deliberate falsification or unconscious denial.

Recognizing that psychosocial tests have limitations when used in isolation, investigators have begun to use clinical, laboratory, and psychosocial tests in combination. The results of one study (Skinner et al. 1980) underscore the advantages of this approach. Using the Munich Alcoholism Test, a new instrument that combines clinical signs and symptoms (as identified by the physician) with self-report information provided by the patient, it was found that the two types of indicators occurred with relative independence of each other. Psychosocial problems predominated in the younger patients, while clinical signs and symptoms were more frequent in the older drinkers.

From Screening to Early Intervention

Developments in several countries indicate that concerted efforts are now underway to link new screening technologies to low-cost early intervention strategies. Building on previous research (Edwards et al. 1977), which

suggested that one session of advice can be as effective as conventional treatment for some alcoholics, a pilot program is currently being evaluated in Scotland to determine the effectiveness of a brief intervention with problem drinkers identified in a general hospital setting (Chick et al. 1982). Preliminary results of a 1-year followup indicate that a single 35-minute counseling session may reduce drinking and its consequences in a significant proportion of newly identified problem drinkers.

In Sweden, an early intervention program was conducted with middle-aged heavy drinkers identified by means of elevated GGTP values (Kristenson 1982). Individuals randomly assigned to a control group received advice to limit their alcohol consumption. Those assigned to an intervention group were repeatedly encouraged to drink less and, in addition, received feedback about their GGTP levels. Compared with the controls, the intervention group showed significant reductions in absenteeism, hospitalizations, and mortality up to 6 years after the initial screening.

In France, screening for problem drinking and alcohol dependence is conducted routinely in industry, health care settings, and the courts. Individuals identified by means of a simple clinical exam (the Le Go grid method) and biochemical tests (GGTP and MCV) are referred to a nationwide system of more than 130 early intervention clinics. While the effectiveness of these clinics has not been studied systematically, the program demonstrates that, from a public health perspective (Babor et al. 1983), early intervention in a large population is now feasible.

Job-Based Interventions

The process by which problem-drinking employees within a work organization are identified and engaged in treatment has been the subject of much discussion (Googins and Kurtz 1980; Roman 1982). Initially, it was advocated that supervisors and managers be trained to identify impaired job performance, and then to confront the employee to determine the necessary corrective action. Supervisors have been found to be inconsistent in confronting their problem-drinking employees (Kurtz et al. 1980), depending on their own attitudes about alcohol use and abuse and their perceptions of the union's support for the program (Beyer et al. 1980). With the impressive expansion of job-based employee assistance programs during the past decade, there has been a shift in

emphasis away from the sole identification of alcohol problems to include any employee problem that adversely affects performance (Roman 1981). In the more recently developed employee assistance programs, the role of supervisor as diagnostician has been deemphasized in favor of the more traditional roles of supervising performance and focusing on unacceptable changes in performance as the basis for intervention and referral.

One recent study investigated how the threat of disciplinary action affects treatment outcome (Freedberg and Johnston 1980). Men who chose to enter treatment as an alternative to disciplinary action were compared with others who sought treatment voluntarily. Both groups were similar with respect to job functioning and drinking behavior after 1 year, a finding that denotes the disciplinary process as a useful way to engage employees in treatment. Moberg et al. (1982) report followup data on several hundred inpatients from an abstinence-oriented industrial program. Forty-six percent were abstinent 3 months after treatment, while 37 percent were abstinent after 9 months. About 20 percent were drinking moderately at each followup, but those listed as moderate drinkers at 3 months had a high relapse rate 9 months after treatment. Although social support and employer involvement had a positive influence on outcome, type of referral (voluntary vs. coercive) made little difference.

Despite increasing interest in the evaluation of occupational programs, the relative lack of studies in this area, together with limited access to the worksite, have made it difficult to assess the overall contribution of these programs to improved job performance and reduced health care costs.

Alcoholism Treatment: Programs and Therapeutic Approaches

The treatment system that emerged following the Second World War is varied, complex, and still in the process of development. Many of its programmatic components (e.g., detoxification facilities, inpatient hospital and residential programs, halfway houses, and outpatient clinics), and the therapeutic approaches employed in these components, have only recently begun to receive systematic research attention. What is the most appropriate setting for treatment? What are the most efficacious therapeutic approaches? What is the

optimal reimbursement policy for medicare, medicaid, and private insurance carriers?

Detoxification

The management of acute alcohol intoxication and the concomitant alcohol withdrawal syndrome is commonly referred to as detoxification. In the late 1960s, specialized detoxification facilities were developed to address the needs of the public inebriate; these facilities expanded rapidly following widespread adoption by the States of the 1974 Uniform Alcoholism and Intoxication Treatment Act decriminalizing public intoxication. At first these facilities were closely associated with hospital emergency services, but in recent years several alternatives to hospital-based detoxification have emerged. A recent survey of State Alcoholism Authorities (Den Hartog 1982) indicated a threefold increase in the number of States operating or purchasing non-hospital-based detoxification since 1975. The most controversial of these alternatives has been "social setting" detoxification, in which the use of drugs such as diazepam and chlor-diazepoxide is avoided in the management of withdrawal in favor of a supportive social climate designed to engage the patient in further treatment.

Because the different detoxification methods have not been assessed directly in controlled comparisons, little is known about the relative merits of various approaches in relieving the distress associated with alcohol withdrawal, preventing the risks of medical complications, and facilitating the process of referral to long-term treatment. The need for hospital-based, medical detoxification, as well as the referral to social setting facilities, depends on the patient's physical condition, nutritional status, severity of alcohol dependence, and overall medical evaluation.

The growth of social setting detoxification has resulted from its lower cost, the changing attitudes about the use of sedatives and tranquilizers in withdrawal management, and evidence indicating that a significant number of patients do not experience serious medical complications during withdrawal (Diesenhaus 1982). A recent review of the published literature in the United States, Canada, and Europe (Den Hartog 1982) concluded that detoxification could occur humanely, safely, and efficiently in nonhospital settings. These reports need to be substantiated by systematic research, since it is not clear what proportion of alcoholics require detoxification in hospital

settings. Moreover, while the proponents of social setting detoxification contend that it will facilitate referrals to long-term treatment, insufficient evidence exists to conclude that this type of referral will occur more efficiently in either social or medical settings.

What is clear from the experience of non-hospital detoxification programs is that medically oriented inpatient detoxification may not be necessary for the majority of referrals who are not severely dependent and who are otherwise in good health (Diesenhaus 1982). With the increasing availability of ambulatory and inpatient social setting detoxification, careful study of the nature and role of detoxification in the rehabilitation process would seem warranted. The long-range implications of this trend need to be considered.

The high rate of relapse among detoxified patients is another major concern to service providers. Although it may be unrealistic to expect an initial care component to accomplish much more than detoxification and referral, at present a small proportion of the public inebriate population utilizes a disproportionate share of detoxification resources. Further, these patients often refuse recommended referrals to intermediate care facilities. One study of a predominantly skid row population (Fagan and Mauss 1978) found that fewer than one-third accepted their recommended treatment referral after detoxification. Another study (Richman and Smart 1981) found that the probability of further referral was lowest in those patients having a history of multiple detoxifications. Two possible solutions to the problem of "resource absorption" by detoxification repeaters have been suggested. The first is the greater availability of comprehensive services to the skid row population; the second is the use of legal coercion to motivate the public inebriate to enter and remain in treatment (Fagan and Mauss 1978).

Benefits of Treatment

The rapid growth of insurance coverage for alcoholism treatment has been predicated in part on the assumption that such treatment is cost-beneficial, i.e., the long-term costs to both the individual and society will be reduced. Alcoholics and their families have been found to use a disproportionate amount of inpatient and outpatient medical services for a wide variety of physical problems related to excessive drinking (Rohmann et al. 1981; Putnam 1982a). Reflecting an increased concern with the implications of treatment re-

imbursement policy, a number of new studies have used the improved methods of cost-effectiveness and cost-benefit analysis to show that cost savings can be accrued over a period of time (Swint and Nelson 1977). The first major review of research conducted during the 1970s (Jones and Vischi 1979) found surprising consistency across 12 studies. Despite some methodological problems, these studies indicated significant reductions in medical care use and expenditures related to various kinds of alcoholism treatment, amounting to a 40-percent median reduction in sick days and accident benefits. Additional studies since that time have confirmed this conclusion (Saxe et al. 1983).

Substantiating these findings is a recently conducted study of State employees of California in which 90 families with an alcoholic member, all enrolled in Blue Cross/Blue Shield, were followed for a period of 5 years. Results indicated that the total medical care costs per family member decreased substantially over time once the alcoholic member entered treatment (Holder and Hallan 1981).

These studies have been conducted predominantly within health maintenance organizations (HMOs), primarily because alcoholics are relatively easy to track within these comprehensive health programs. A major study of alcoholism treatment in HMOs recently completed by the Group Health Association of America (Plotnick et al. 1982) compared the utilization of four HMO outpatient alcoholic treatment programs by alcoholic persons from 2 years before entering treatment to 48 months after treatment. The study revealed significant reductions in general health care use for the alcoholic persons and their family members. In another HMO study (Forsythe et al. 1982), treated alcoholics were compared with nonalcoholics over a 4-year period. Although the cost differential between the two groups was substantial during the entire study periods, there was a significant decline in alcoholics' demand for services following referral to treatment.

Focusing on the illnesses that may precipitate alcoholics' referral to treatment, Putnam (1982a) found that alcoholics manifested a high number of physical and emotional illnesses. Problems most likely to be associated with the alcoholics' hospitalizations entailed psychosocial difficulties such as anxiety, depression, and marital discord, as well as accidents, drug overdoses, and violence. In many cases, the alcoholics were found to be seeking

inappropriate care and receiving inappropriate treatment, since the alcoholism underlying their illness was often not properly diagnosed.

In a related study of the same HMO (Putnam 1982b), alcoholics who received outpatient alcoholism treatment were compared with alcoholics who were identified but refused to accept treatment. An examination of the utilization rates for medical care services before and after referral to treatment revealed that both groups showed a higher level of service use in the 4-month period prior to referral. Those who accepted alcoholism treatment, however, showed a progressive decrease in use of services during the subsequent period, while those who refused treatment increased their demand for services approximately 15 months after the attempted referral. This increase is related to a high incidence of problems associated with accidents, drug overdoses, and violence, and highlights the importance of appropriate diagnosis and treatment to reduce both the human and the social costs of alcoholism.

Finally, a significant non-HMO-based study (Holder and Hallan 1981) followed the families of 90 State employees in California for a period of 5 years. Each family had an alcoholic member and all were enrolled in Blue Cross/Blue Shield. The study indicated that total medical care costs per family member decreased substantially over time once the alcoholic family member entered treatment. At the end of the study, inpatient costs per person per month of both the comparison families (N=83) and the alcoholic families (N=90) were similar, and outpatient costs of the comparison families were actually higher.

Although many of the studies of alcoholics' use of health services are limited in scope, methodology, and populations investigated, the evidence suggests that: (1) alcoholics and their families initially use more health care services than nonalcoholics; (2) this elevated demand can be reduced substantially by treatment for alcoholism; and (3) the benefits of alcoholism treatment clearly outweigh its costs.

Treatment Settings

Studies comparing the effectiveness of inpatient programs with outpatient and day hospital treatment are important because of the potential for improved cost-effectiveness and the appeal these approaches may have for alcoholics who reject inpatient or residential care. In one study (Longabaugh et al. in press), patients participating in a day hospital pro-

gram (while returning home at night) were given the same behavioral treatment program that hospitalized inpatients were receiving. At 6-month followup, both groups were comparable on measures of drinking behavior, need for rehospitalizations, and social and occupational functioning. The partial hospitalization group was superior on measures of psychological well-being. Given the lesser cost of partial hospitalization, the authors conclude that this form of treatment is more cost-effective. In another study, comparable findings are reported after 1-year followup of 100 patients who were randomly assigned either to a day clinic or to an inpatient facility offering a similar treatment program (McLachlan and Stein 1982). These results support the conclusion that the two types of treatment setting may be equally effective.

Studies comparing the results of inpatient and outpatient programs have generally not shown significant differences in the effects of treatment setting (see review by Cole et al. 1981). However, it seems likely that day hospital and outpatient treatment programs will prove to be substantially less effective for certain groups of patients, and Spicer et al. (1981) caution that conclusions regarding equal effectiveness should be limited to clients who are appropriate for each type of program. Many studies comparing outpatient with inpatient treatment have not controlled for the possibility that patients choosing outpatient settings are less severely dependent. Inpatient treatment seems indicated when motivation is weak to continue treatment, when patients are psychotic, depressed, or suicidal, and when complicating physical disabilities are present. Other factors which may also influence the choice of treatment setting include patients' social stability and the number and severity of their symptoms, as well as the ability of programs to respond to individual needs (Cole et al. 1981; Spicer et al. 1981). Research is needed to identify the characteristics of patients most likely to benefit from an outpatient program, followed by treatment-matching studies to test the validity of the identified patient types. An additional obstacle to the substitution of residential treatment by outpatient treatment is the high rate of client attrition usually encountered in the outpatient setting.

Matching Clients with Programs

Considerable selection takes place on the part of both the client and the referral agent

in the natural process of identifying alcoholics and referring them to treatment (Pattison and Kaufman 1981; Beckman and Kocel 1982). Despite the increasing variety of programs, settings, and treatment modalities, many alcoholics do not have the opportunity to find an informal match between their own specific needs and the type of treatment available. This is particularly true of special needs populations, such as women, ethnic minorities, the multidisabled, the elderly, and skid row alcoholics. For these groups, access to treatment and successful rehabilitation are often impeded by cultural barriers, financial constraints, and program design characteristics. Since considerable attention has already been given to the needs of these groups in the Fourth Special Report (USDHHS 1981) and other sources (Diesenhaus 1982; NIAAA 1982), this section will provide a brief update on the issues common to all special needs populations.

Because middle-aged white men represent the typical clientele of most treatment facilities, programs often are not designed with the needs of other groups in mind. These groups may thus be reluctant to seek help because of a number of structural and group-specific barriers that restrict access to appropriate facilities. For example, a survey of 53 California treatment facilities (Beckman and Kocel 1982) suggested that women alcoholics were less likely to enter programs lacking child care services, professional staff, and aftercare programs. However, Institute experience in providing services for women reveals that many women do not utilize child care services even when provided. Other barriers to treatment for some groups are language differences and composition of treatment staff. Minority staff members represent only a small proportion of the Nation's alcoholism treatment personnel (NIAAA 1980). Yet another barrier may be financial constraints. Women and minorities tend to be overrepresented in publicly funded facilities and underrepresented in private ones (NIAAA 1980). Without programs' sensitivity to the needs of these populations, many individuals may fail to seek treatment until their alcoholism has reached a severe stage of development.

With the growing recognition that utilization rates may be improved by removing barriers to access, greater attention is now being given to special population groups in the design of treatment programs. Some facilities seeking to attract Hispanic and Native American clients are using folk medicine and native healing approaches as alternatives or adjuncts

to traditional medical and psychiatric treatments. Counselors and other treatment staff members are being matched to the sex and ethnic background of their clients. Also, conveniently located, community-based programs, at times staffed and planned in collaboration with special population groups, are appearing more frequently (NIAAA 1982).

What is uncertain, at this time, is the impact the ADM block grant program will have on treatment programs. Of particular interest will be the differential rates of service use across States, by both the general population and special population groups. As these programs become available to special population groups, research will need to move from program descriptions to actual evaluation studies in which programs designed for special population groups are compared with more traditional approaches.

One exception to the trend to develop services for special population groups and subtypes of alcoholic persons has been the small number of combined programs serving both alcoholics and drug dependent persons. Although combined treatment for alcoholics and drug abusers has not been widely adopted, the feasibility of such programs may have important implications for the planning of future treatment services, given the increased tendency for patients to present multiple abuse or addiction patterns at the time of treatment (Sokolow et al. 1981).

Therapeutic Approaches

Several diverse treatments are often delivered within the context of alcoholism services, depending on the resources and needs of clients, as well as the specific training or orientation of the staff. In many cases, a combination of therapeutic interventions is provided to all clients, under the assumption that multiple treatments stand a good chance of meeting at least some of each client's needs. As the alcoholism treatment system has grown in size and complexity, evaluating individual therapeutic approaches in isolation from one another has become more difficult. Many studies reported in the literature involve adding a treatment of interest to a facility's standard therapeutic program, or are descriptive evaluations rather than clinical trials comparing one or several treatments with the absence of treatment. Studies discussed here generally employed an experimental method-

ology, as firm conclusions about efficacy can be made only through systematic comparisons.

Behavior Therapy

Behavioral elements most frequently applied in treatment programs include social skills and assertiveness training, contingency management, deep muscle relaxation, self-control training, and cognitive restructuring (Miller and Mastria 1977). Despite their widespread adoption, these methods have not been subject to systematic evaluation with random assignment of large numbers of patients to various treatment conditions.

Aversion therapy.--This approach to treatment grew from Pavlovian conditioning theory, which predicts that the sight, smell, and taste of alcohol will acquire aversive properties if repeatedly paired with noxious stimuli. A thorough review of the research in this area (Miller and Hester 1980) concluded that emetic (nausea producing) aversive conditioning can be effective for employed, married, well-motivated alcoholics, but that the effectiveness of electrical aversion is doubtful. These conclusions are supported by recent work (Cannon et al. 1981) comparing these two aversion procedures when used in addition to a standard treatment program. Patients exposed to emetic conditioning exhibited significantly greater improvement after 6 months than patients exposed to conditioning with electric shock, but the advantage after 1 year was not significant.

Work has also proceeded on covert sensitization, a procedure in which imagined scenes of alcohol ingestion are paired with nausea induced by verbal suggestion. This method may overcome some of the practical disadvantages of emetically based aversion therapy. Some recent studies have offered support for this procedure. One study found that patients receiving covert sensitization remained abstinent longer and demonstrated better psychosocial adjustment than patients in control groups (Elkins 1980). In a related study, patients receiving covert sensitization in a standard inpatient treatment program showed greater improvement over a 4-year period than patients who received insight-oriented therapy (Olson et al. 1981).

Extinction.--Recent experiments have shown that alcohol-related stimuli (such as the sight or smell of an alcoholic beverage) can induce both physiological changes and altered behavior in alcoholic patients (Kaplan et al. 1983; Pomerleau et al. 1983). Because condi-

tioning processes related to alcohol craving and tolerance development may affect the rapidity of relapse after a period of abstinence, some have proposed that conditioned extinction be made an essential part of treatment (Hinson and Siegel 1980). Extinction of the effectiveness of alcohol-related cues is achieved by repeatedly presenting them unaccompanied by either alcohol ingestion or intoxication. This may theoretically weaken the link between these stimuli and drinking behavior, with a consequent improvement in treatment outcome. To date, a few successful case studies have been reported (e.g., Blakey and Baker 1980). Additional systematic investigation should clarify these data from clinical research studies.

Coping skills training.—Behavior therapists have recognized the importance of teaching new, adaptive coping skills to patients who engage in dysfunctional behaviors. These skills include altering conditions before or after drinking, as well as developing alternative ways of coping with persons, events, and feelings that may have served to maintain abusive drinking (Miller and Mastria 1977). A number of studies have demonstrated the benefits of teaching social and other coping skills (e.g., Ferrell and Galassi 1981; Oei and Jackson 1980, 1982), although others have not confirmed these findings (c.f., Sanchez-Craig and Walker 1982). Chaney et al. (1978) have tested a cognitive-behavioral model of treatment, characterized as "relapse prevention" because of its focus on identifying highrisk situations. Patients given skills training had less severe (though no less frequent) relapse episodes, were more likely to be employed, and attended aftercare more regularly than patients in control groups. A replication with alcoholic persons of higher socioeconomic status than the population originally studied found both skill training and discussion groups to be superior to a minimal-treatment group, but not different from each other (Jones et al. 1982). Further study should capitalize on the increasingly widespread use of these methods.

Selecting Treatment Goals

Interest in therapies with alternative drinking goals was sparked in part by findings that a small proportion of treated alcoholics reportedly were able to return to moderate drinking without serious problems (Pattison 1976). The Rand study (Armor et al. 1978), for example, reported that some "definitely alcoholic" persons were capable of drinking

moderately 6 months after treatment and of maintaining moderation up to 12 months later. More recently, Moos, Finney, and Chan (1981) found that 12 months after abstinence-oriented treatment, 25 percent of the recovered patients were drinking in moderation. It should be noted that the term moderation in these studies refers to an average consumption of less than 5 ounces of absolute alcohol per day, in the absence of alcohol-related symptoms, work problems, or rehospitalizations.

Several explanations for these findings seem to be emerging from studies employing longer followup intervals and more refined assessments of patient characteristics. The second part of the Rand study (Polich et al. 1981) revealed that the relapse rate 4 years after treatment was higher (41 percent) among those who had been drinking moderately at 18 months than among those who had been abstinent at that time (30-percent relapse rate). Other research (Orford et al. 1976; Finney and Moos 1981), as well as the Rand studies, suggests that those individuals characterized as moderate drinkers at followup tend to have had less severe alcohol dependence and fewer drinking problems prior to treatment. A 2-year followup of patients receiving abstinence-oriented treatment (Finney and Moos 1981) compared persons drinking moderately at 6 months following treatment with persons who abstained. Those who returned to moderate drinking soon after treatment were more likely to relapse to heavy drinking than those who remained abstinent for 6 months and who then attempted moderate drinking. Thus, while moderate drinking seems possible for some patients following treatment, this phenomenon may be explained by the highly variable course of alcoholism over time (some alcoholics take longer to relapse than others), and by the existence of individual differences among patients in severity of alcohol dependence, social supports for moderate drinking, and a variety of other mitigating factors.

Another possible explanation lies in the nonstandard interpretation of the term alcoholic. In many studies that report moderate drinking by alcoholics, little information has been provided about the patients' severity of dependence, history of alcohol abuse, or extent of alcohol-related disability. In studies reporting such data, moderate drinking patients tend to be less "alcoholic" at the time they enter treatment. Had stringent criteria for alcohol dependence been applied, it is conceivable that the drinking problems of many of these patients would be classified as

alcohol abuse rather than alcoholism. Similar semantic problems cloud the meaning of moderation. Several studies (Armor et al. 1978; Moos et al. 1981) have included amounts of up to 5 ounces of absolute alcohol per day within their definitions of moderate drinking. While many patients were drinking far below this limit, the use of such a high cutoff point would tend to inflate estimates of the proportion of moderate drinkers. Furthermore, to the extent that cognitive impairment and other alcohol-related disabilities may result from consuming these amounts (Wilkinson and Sanchez-Craig 1981), this liberal definition of moderation may be questioned in terms of its health implications.

The controversies generated by such studies as the Rand reports (Armor et al. 1978; Polich et al. 1981) illustrate the need to apply sophisticated and systematic research methodology to explorations of the following questions: In the small number of alcoholic subjects who have reportedly established a pattern of moderate drinking, how stable is that pattern over time? How much is this drinking pattern altered by stress, anxiety reactions, and environmental factors, such as media advertising and the cost of alcoholic beverages? What are the risks of resuming drinking compared with abstinence in this group? What are the characteristics of patients who have developed moderate drinking patterns in terms of age, sex, socioeconomic level, marital status, and drinking history? To what extent does the existence of a belief in moderate drinking increase the risk of relapse in alcoholic patients?

Despite questions raised about treatment goals for alcoholic persons, research has nevertheless proceeded on the effects of teaching moderation to socially stable problem drinkers (Heather and Robertson 1981). Pomerleau et al. (1978) have demonstrated that behavioral treatment can be used effectively for teaching moderation to middle-income nonalcoholic problem drinkers, while others have explored some dimensions of teaching nonproblem alcohol consumption skills. Effective elements of treatment that have been identified include modeling and repeated practice of a clearly delineated pattern of moderation (Strickler et al. 1981), and empathy on the part of therapists (Miller et al. 1980). These must be viewed as suggestive findings, since the results of these treatment studies have yet to be confirmed with various subgroups of problem drinkers, in natural environment settings, or with studies of long-term outcomes.

In summary, the issues surrounding moderate, nonproblem drinking as a viable treatment option for nonalcoholic problem drinkers are not likely to be completely resolved until progress has been made in those areas most relevant to this controversy: improved nomenclature, more precise diagnostic criteria, new techniques for differential assessment, earlier treatment interventions, better matching of client needs to specific treatments, and improved research methodologies. It should be clear that while continued exploration and assessment of a variety of treatment options and goals for nonalcoholic and/or prealcoholic persons are appropriate endeavors, the consensus of clinical opinion is that the most appropriate goal for alcoholic persons is abstinence.

Alcoholics Anonymous

With nearly 50 years of service to alcoholic persons and their families, the fellowship of Alcoholics Anonymous (AA) has not been the subject of systematic research to study its long-term and short-term efficacy. To this end, Glaser and Ogborne (1982) reviewed the clinical and research literature, and have proposed a number of research questions and designs to highlight the most effective elements of the AA program and describe those persons for whom AA participation is the preferred approach.

Efforts to identify patients who are best suited to the AA approach have thus far sought to identify characteristics of those who have become actively involved in the AA program (e.g., Boscarino 1980; O'Leary et al. 1980). Some common characteristics have been identified, including need for structure and for affiliation with a group. As yet, no consistent profile has emerged to characterize successful AA members in terms of degree of impairment, social stability, or emotional disruption. This state of affairs has been attributed to inconsistencies of findings among studies, differing focuses of attention across studies, and methodological inadequacies (Ogborne and Glaser 1981).

Alcoholics Anonymous is a major voluntary resource in the treatment of alcoholism, with a reported membership of 476,000 in the United States and Canada in 1980. The most comprehensive information on membership comes from the organization itself (Alcoholics Anonymous 1981). The triennial sample sur-

veys conducted since 1968 profile characteristics and trends of the membership. For example, membership has increased from 170,000 in 1968 to 476,000 in 1980; the proportion of women increased from 22 percent in 1968 to 31 percent in 1980; the proportion of people 30 and under has increased to 14.7 percent in 1980 from 11.3 percent in 1977 when this trend toward younger membership was first noted; and the proportion who state counseling agencies and treatment facilities as important in their attending their first AA meeting increased from 19 percent in 1977 to 26 percent in 1980. (Of those members beginning AA since the 1977 survey, 33 percent indicate counseling and treatment referrals were most responsible for first attendance.)

Dynamic Psychotherapy and Group Therapy

Numerous descriptions of the application of dynamic psychotherapy to the treatment of alcoholism exist (Bean and Zinberg 1981; Zinberg 1982), but almost no outcome studies evaluating this approach to treatment have been conducted. In recent years, psychotherapeutic approaches have given way to, or have been combined with, behaviorally oriented treatment in many settings. Some clinicians have argued against the use of psychotherapy on the grounds that it may strip patients of defense mechanisms that could be used in the service of sobriety (Wallace 1978), and that it makes demands that alcoholics are unready to meet in the early phases of recovery (Vaillant 1981). Nevertheless, there are indications that psychodynamically oriented group psychotherapy may in fact be preferred for certain subgroups of alcoholic persons (Kissin 1977).

A considerable body of literature describes group therapy techniques in the treatment of alcoholism (e.g., Vannicelli 1982), but with only a few evaluative studies to support its efficacy. A recent study by Oei and Jackson (1980) compared group with individual therapy, each of these approaches being used to provide social skills training to some patients and traditional supportive therapy to others. Significant improvement was found only in patients given social skills training, with greater improvement in those trained in a group setting than among those treated individually. Given the potential cost-effectiveness of group therapy, as well as its widespread use, these findings should be followed up with comprehensive evaluative studies.

Family Treatment

The accumulating evidence documenting the deleterious effects of alcoholism on families has prompted heightened interest in family treatment of alcoholism, as described in some detail in the Fourth Special Report (USDHHS 1981). Although the therapeutic value of family treatment is well documented and heralded for many related problem behaviors (Stanton and Todd 1982), its unique contribution and efficacy with alcoholism treatment remain to be fully demonstrated and evaluated (Steinglass 1979; Pattison and Kaufman 1981). Preliminary results from a study comparing degrees of spouse involvement in outpatient alcoholism treatment indicate certain advantages for marital therapy, but 18-month outcome data have not yet been reported (McCrary and Noel 1982). Considerable work in evaluating family therapy with alcoholic families is needed, especially efforts to match specific treatments to the problems presented by different families.

Pharmacotherapy

The value of pharmacological agents such as disulfiram (Antabuse) as a deterrent to drinking and various patient factors associated with favorable outcome is of continuing research interest. In recent work, Fuller and Roth (1979) randomly assigned 128 men to receive either a standard dose of disulfiram, a pharmacologically inactive dose, or a daily dose of the vitamin riboflavin. The standard dose of disulfiram was more effective than riboflavin in producing abstinence after 1 year, although differences were small (Fuller and Williford 1980).

One aspect of treatment with disulfiram has been patient noncompliance, that is, the patient's unwillingness to continue regular ingestion. Azrin et al. (1982) studied compliance-enhancement procedures with patients in a rural outpatient clinic. A behavioral "disulfiram assurance" program, involving five sessions of stimulus control training, role playing, and communication skills training, was highly effective in promoting abstinence in married clients, but had little effect on single clients. However, the addition of behavior therapy (training in drink refusal, social skills, and muscle relaxation, and counseling in recrea-

tion activities and job-finding) was sufficient to produce nearly complete abstinence for single clients. These are intriguing results, especially since they were obtained in relatively few sessions and with only monthly followup contacts thereafter.

Another study tested the effects of a compliance-enhancement procedure in an industrial setting (Robichaud et al. 1979) and demonstrated the effectiveness of closely supervised ingestion of disulfiram for reducing absenteeism, but only during the treatment period. The findings regarding disulfiram should be regarded as preliminary, but they do suggest that disulfiram may have a useful role in treatment if compliance can be ensured. Future studies of compliance with broader ranges of clients are needed and may be facilitated by newly developed methods for monitoring use of disulfiram (e.g., Paulson et al. 1977; Neiderhiser and Fuller 1982).

A possible role for lithium in the treatment of alcoholism has been considered because of its effectiveness in treating affective disorders and the relatively high incidence of these disorders in alcoholics. The findings thus far have been equivocal. In one study, depressed patients taking lithium had fewer drinking episodes compared with placebo control patients, but without any greater alleviation of the depression than occurred in the control patients (Kline et al. 1974). In other studies, however, depressed patients did not consume less alcohol during lithium therapy (Pond et al. 1981). The possible value of lithium in treating alcoholics thus remains uncertain, with more definitive studies required, employing double-blind procedures and comparisons with other forms of treatment (McMillan 1981).

Ciraulo and Jaffe (1981) have reviewed the use of tricyclic antidepressants in alcoholics and report success in treating initial symptoms of withdrawal, such as anxiety, depression, and somatic discomfort, only within the first 2 to 3 weeks after cessation of drinking. In a subsequent study, however, they demonstrated that alcoholics show greater clearance and lower plasma levels of imipramine than non-alcoholics, suggesting that previous studies which found persistent restlessness and malaise may have utilized dosages that were inadequate for alcoholics (Ciraulo et al. 1982). Future studies should seek to define subtypes of depression in alcoholics, monitor plasma levels of antidepressants, and evaluate drug effects both on depression and on drinking behavior.

Factors Affecting Treatment Outcome

Quality of Treatment

As in other areas of psychotherapy, treatment outcome in alcoholism is affected by the perspectives and attitudes of the therapists, which in turn are functions of their training, experience, and self-esteem (Cartwright 1980). Treatment outcome is also affected by the interpersonal skills of the alcoholism counselor. Valle (1981) evaluated treatment outcomes in patients randomly assigned to eight recovered alcoholic counselors and found that counselors having a higher level of interpersonal functioning tended to have patients who drank less, relapsed less often, and recovered more quickly after a relapse.

Evaluations of training programs for alcoholism counselors indicate that they are able to achieve gains in such areas as participants' knowledge, attitudes, ego strength, capacity for self-disclosure, and effectiveness of counseling (Gideon et al. 1980). The success of alcoholism counselor training programs has prompted their use as a treatment method with chronic alcohol- and drug-dependent individuals, with positive results sustained for at least 1 year (Kahn and Stephen 1981).

Response to Treatment

Treatment research has increasingly focused on identifying personal and environmental factors that predict positive or negative treatment outcomes. Positive outcomes are no longer conceived solely in terms of total abstinence from alcohol, since posttreatment functioning in such domains as physical health, psychological adjustment, social functioning, and occupational performance may not deteriorate automatically with relapse to drinking (Finney et al. 1980); moreover, successful abstinence is not necessarily associated with good functioning in other areas of adjustment.

One form of negative outcome is attrition, that is, failure to complete the treatment program. Client factors found to be related to attrition include inadequate financial resources, low social stability, and youth (Welte et al. 1981; Keil and Esters 1982). Treatment variables such as program duration and size of treatment groups also can influence the

dropout rate (Schroeder et al. 1982). Undoubtedly, attrition results from an interaction between individual and program factors and may be reduced by improvements in program design and the method of assigning patients to treatments.

It is generally believed that treatment outcome is affected less by the treatment process itself than by the personal resources and characteristics the client brings to the treatment situation. Numerous studies have shown good prognosis to be associated with social stability and marital adjustment (Baekeland 1977). Personal characteristics recently reported to be associated with poor prognosis include cognitive impairment (Abbott and Gregson 1981) and depression (Hatsukami et al. 1981). While client characteristics seem to be important determinants of outcome regardless of the quantity or quality of treatment, other research has shown that this effect may be mediated indirectly by the environment to which the client returns after residential treatment (Cronkite and Moos 1980). Another study found that three situational factors (negative mood states, interpersonal conflicts, and social pressure to drink) were most likely to precipitate relapse after treatment (Cummings et al. 1980). These studies indicate that therapeutic efforts must deal not only with the individual characteristics of the patient, but also with the environmental contexts in which the patient is expected to function after treatment.

With this in mind, some have advocated giving more attention to restructuring of the client's environment through marital or family therapy, while others have called for a more general approach focusing on frequent aftercare contact in the period after treatment (Costello 1980). As a neglected dimension in the treatment system, aftercare consists of (a) ongoing supportive activities, such as professional and self-help programs designed to maintain treatment gains, (b) prevention of costly rehospitalizations, and (c) improvement in social and occupational functioning. At present, the resources most frequently available to fulfill these functions are halfway houses, AA groups, and program-sponsored support groups. In general, affiliation with aftercare groups is associated with better treatment outcome (Costello 1980). Further research is needed to determine the relative efficacy of different kinds of aftercare, the optimal frequency and duration of aftercare, and how clients can be induced to comply with aftercare without dropping out.

Matching Patients to Therapies

The emerging concept that alcoholism is not a unitary disorder has stimulated renewed interest in delineating different subgroups or types of alcoholic persons. As discussed previously, the goal of this research is to facilitate treatment planning and improve treatment outcome by matching types of alcoholic persons with the most appropriate treatment interventions. Although this typological approach is not new to the field of alcoholism, recent studies have benefited from improvements in assessment technology (Meyer et al. 1981) and from the application of more sophisticated statistical techniques (Skinner 1982).

Attempts to differentiate alcoholics on the basis of personality characteristics account for most typological research efforts (Morey and Blashfield 1981). Several independent studies have denoted two common subgroups: (1) passive-dependent alcoholic persons characterized by antisocial personality disorder and (2) neurotic alcoholic persons who may use alcohol as a coping mechanism. In a study designed to explore the often-noted association between alcoholism and antisocial (psychopathic) personality, Hesselbrock et al. (in press) compared alcoholics having an early history of social deviance with those who were relatively free from problems before the onset of alcoholism. Alcoholics with antisocial personality were found to have an earlier onset of alcoholism, as well as a more rapid and severe progression of drinking problems. Several other studies (McLellan et al. 1981; Zivich 1981) suggest the importance of psychiatric disturbance, particularly in the alcoholic's response to treatment. Alcoholic subgroups characterized by poor psychological adjustment were found to show little or no improvement following treatment, whereas those having high adjustment levels showed significant improvement. McLellan et al. (1981) conclude that because patients having less severe psychological disturbance respond to most kinds of treatment, cost considerations would recommend this subgroup to outpatient settings.

The probability of a genetic predisposition to alcoholism has prompted the search for an alcoholic subtype related to family pedigree. One study of more than 7,000 alcoholic men found that those with a family history of alcoholism had more severe symptomatology, more antisocial behavior and other psychopathology, less stable employment histories,

and more severe physical symptoms (Frances et al. 1980). Another approach to the classification of alcoholics is based on certain indicators known to predict treatment outcome. One study (Gibbs 1981) classified alcoholics on the dimensions of social stability and intellectual functioning and showed that mutually exclusive types were often assigned to the same treatment regimen despite their widely different rehabilitation needs.

In an attempt to directly test the clinical implications of differentiating subgroups of alcoholics, several investigators have studied how certain types of patients respond to different treatments. Using a classification system that differentiates chronic alcoholics from a less severe type of behaviorally impaired drinker, Brown and Lyons (1981) found that alcoholics do slightly better in programs having a high medical orientation, while the behaviorally impaired drinkers respond better to treatment having a high psychological rehabilitation orientation. Using a different classification scheme, Finney and Moos (1979) studied the treatment response of alcoholics classified in terms of high or low social competence. Contrary to the findings of Brown and Lyons (1981), no evidence was found to indicate that various treatment programs were differentially effective for different types of patients.

Although evidence supporting treatment matching remains equivocal, there may be important methodological reasons why the matching hypothesis has not been adequately tested. First, despite improvements in the methods and theory of classification, the optimal classification system has yet to be developed. Even though family history, psychiatric disorder, alcoholic symptomatology, and organic brain dysfunction seem to be promising differentiating characteristics, there has been little attempt to integrate information from these disparate levels of analysis into more comprehensive typologies. Second, many matching studies have not been designed to detect predicted interactions. In a recent review of the matching literature (Skinner 1981), it was found that evidence supporting the matching hypothesis came primarily from those experimental studies that randomly assigned patients to treatment conditions, while the results of nonexperimental correlational studies tended to be less supportive. Finally, in a number of studies it is possible that the assigned treatments were not sufficiently distinct to produce a differential effect. Thus, while the matching hypothesis still holds

promise as an avenue to improved treatment efficacy, the systematic study of patient-treatment interactions will have to await the development of better typologies and improved scientific research methodology.

Emerging Trends and Future Directions

A consensus appears to be developing among clinicians, researchers, and policymakers that treatment research is passing through a transitional period during which basic assumptions are being reevaluated and a new approach to treatment efficacy is emerging. The elements of this trend include refinements in the definition of terms, improvements in the technology of diagnosis, a more sophisticated approach to the planning of treatments, and a new awareness of the complexity of evaluation methodology.

The advances noted in this chapter in the areas of conceptualization, assessment, and treatment intervention can only serve to enhance the quality of services available to alcoholic persons. The notion that different degrees of alcohol dependence can be measured, that different types of alcoholic persons can be classified, and that different types of disability can be diagnosed has important implications for treatment and research. Recent reviews of treatment-related priorities conducted by the Institute of Medicine (1980), the *Journal of Studies on Alcohol* (Keller 1979), and the World Health Organization (Edwards et al. 1981) suggest that these themes will constitute a promising but ambitious agenda for the 1980s. As articulated in the writings of many specialists in the field, a new approach is emerging regarding the ways in which treatment is conceptualized, conducted, and evaluated.

The traditional model, which still dominates much thinking about alcoholism treatment, describes how heterogeneous groups of patients are assigned to multimodal treatment programs. After a period of time, the relative success or failure of treatment is evaluated primarily on the basis of the proportion of patients remaining abstinent, and secondarily by global assessments of functioning in other areas of living. Because both treatment variables and client variables are aggregated in this approach, treatment effects may be obscured when the improvements of some patients are averaged with the lack of improvement or even deterioration of other patients. Even when treatment effects are observed, it

is not clear which parts of the treatment process are responsible. In addition to the conceptual limitations of this model, much of the evaluation research conducted within this tradition is difficult to interpret because patient characteristics differ from one study to another, outcome criteria have not been sufficiently specified, comparison groups have not been included in the research design, and the treatment process has not been adequately described. Furthermore, the posttreatment environment has not been taken into account, and there have been unrealistic expectations about what the treatment will accomplish.

In contrast to the traditional model of treatment, the emerging model stresses the heterogeneous nature of the client population, the need for more specific and efficient interventions, the importance of maintaining treatment gains in the posttreatment environment, and the diversities of different outcomes (Skinner 1981; Cronkite and Moos 1980). This model differentiates among types of alcoholic persons (e.g., less dependent or more dependent, depressed or not depressed, cognitively impaired or not impaired) and jointly attempts to match each type with the most appropriate combination of treatment interventions (e.g., pharmacotherapy, behavior therapy, family treatment, etc.). The efficacy of various treatment combinations is evaluated by comparing patients who are matched to appropriate treatments with those who are mismatched or assigned randomly to a standard package of interventions. Within the new model, greater attention is given to evaluating changes in behavior, attitudes, physical health, and psychosocial functioning taking place during the process of treatment. Another area of focus is the posttreatment environment, where the patient's recovery may be impeded or supported by what takes place in the family setting or in the job situation. Ideally, the treatment process would continue during the posttreatment period in the form of various kinds of specialized aftercare. Finally, the new model recognizes that outcome may vary along a variety of dimensions, and that abstinence is just one goal of a more ambitious treatment strategy that includes rehabilitation in other important areas of functioning.

Summary

The traditional concept of alcoholism as a unitary disease has been challenged. Over the past decade, researchers and clinicians have

come to realize that multiple patterns of alcohol use may result in multiple forms of disability. Accordingly, a new emerging model of treatment stresses the heterogeneous nature of the client population, the need for more specific and efficient treatments, and the importance of maintaining gains after treatment. This model differentiates among alcoholics (e.g., depressed vs. nondepressed) and attempts to match each type with the most appropriate combination and configuration of treatments.

Recognizing the importance of accurate patient descriptions, the American Psychiatric Association has developed systematic criteria for classifying alcoholic patients along a number of dimensions. The technique, a part of DSM III (Diagnostic and Statistical Manual of Mental Disorders) promises to provide more standardized and comprehensive patient diagnoses.

Several methods are currently used to identify alcoholics before they come for treatment. Laboratory tests for biological markers or indicators can be a powerful aid to detecting alcoholism. Current research on the use of biochemical indicators in the early detection of alcoholism suggests that a single specific biochemical marker for alcoholism may be elusive. The combination of GGTP, MCV, and several other tests, however, appears to offer, at relatively low cost, a strong indication of recent excessive alcohol consumption. In addition, a widely used and validated self-administered test, the Michigan Alcoholism Screening Test (MAST), elicits responses to medical, social, and behavioral statements. The MAST is rapid, inexpensive, and relatively accurate.

Studies show that untreated alcoholics and their families are disproportionately high users of medical services. Insurance programs, especially HMOs, in recognition of the cost implications have begun to target alcoholism for increased attention. Preliminary studies suggest that partial hospitalization or outpatient programs may be as effective as inpatient programs for some patients; moreover, the lower costs of the former may lead to increased use.

Treatments continue to rely largely on psychotherapy and behavior therapy (including social skills and assertiveness training, self-control training, cognitive restructuring, and aversion therapy, which pairs alcohol with unpleasant stimuli). In addition, group approaches like Alcoholics Anonymous are widespread. Behavior therapy in conjunction

with disulfiram (Antabuse) seems to be highly effective in producing abstinence; however, abstinence alone does not imply successful psychosocial adjustment.

References

- Abbott, M.W., and Gregson, R.A.M. Cognitive dysfunction in the prediction of relapse in alcoholics. *Journal of Studies on Alcohol* 42(3): 230-243, 1981.
- Alcoholics Anonymous, General Service Office. *Analysis of the 1980 Survey of the Membership of A.A.* New York: AA, 1981. 18 pp.
- American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders. (DSM III).* 3rd ed. Washington, D.C.: APA, 1980.
- Armor, D.J.; Polich, J.M.; and Stambul, H.B. *Alcoholism and Treatment.* New York: John Wiley & Sons, 1978.
- Azrin, N.H.; Sisson, R.W.; Meyers, S.B.; and Godley, M. Alcoholism treatment by disulfiram and community reinforcement therapy. *Journal of Behaviour Therapy and Experimental Psychiatry* 13(2):105-112, 1982.
- Babor, T.F.; Treffardier, M.; Weill, J.; Fegueur, L.; and Ferrant, J-P. Early detection and the secondary prevention of alcoholism in France: A *quoi bon?* *Journal of Studies on Alcohol* 44(4): 600-616, 1983.
- Baekeland, F. Evaluation of treatment methods in chronic alcoholism. In: Kissin, B., and Begleiter, H., eds. *Treatment and Rehabilitation of the Chronic Alcoholic.* New York: Plenum Press, 1977. pp. 383-440.
- Bean, M.H., and Zinberg, N.E., eds. *Dynamic Approaches to the Understanding and Treatment of Alcoholism.* New York: Free Press, 1981. 214 pp.
- Beckman, L.J., and Kocel, K.M. The treatment-delivery system and alcohol abuse in women: Social policy implications. *Journal of Social Issues* 38(2):139-151, 1982.
- Beyer, J.M.; Trice, H.M.; and Hunt, R.E. The impact of Federal sector unions on supervisors' use of personnel policies. *Industrial Labor Relations Review* 33:212-231, 1980.
- Blakey, R., and Baker, R. An exposure approach to alcohol abuse. *Behaviour Research and Therapy* 18:319-325, 1980.
- Boscarino, J. Factors related to "stable" and "unstable" affiliation with Alcoholics Anonymous. *International Journal of the Addictions* 15(6): 839-848, 1980.
- Brady, J.P.; Foulks, E.T.; Childress, A.R.; and Pertschuk, M. The Michigan Alcoholism Screening Test as a survey instrument. *Journal of Operational Psychiatry* 13(1):27-31, 1982.
- Brown, J., and Lyons, J.P. A progressive diagnostic schema for alcoholism with evidence of clinical efficacy. *Alcoholism: Clinical and Experimental Research* 5(1):17-25, 1981.
- Cannon, D.S.; Baker, T.B.; and Wehl, C.K. Emetic and electric shock alcohol aversion therapy: Six- and twelve-month follow-up. *Journal of Consulting and Clinical Psychology* 49(3):360-368, 1981.
- Cartwright, A.K.J. The attitudes of helping agents towards the alcoholic client: The influence of experience, support, training, and self-esteem. *British Journal of Addiction* 75(4):413-431, 1980.
- Chaney, E.; O'Leary, M.; and Marlatt, G.A. Skill training with alcoholics. *Journal of Consulting and Clinical Psychology* 46(3):1092-1104, 1978.
- Chick, J.; Kreitman, N.; and Plant, M. Mean cell volume and gamma-glutamyl-transpeptidase as markers of drinking in working men. *Lancet* 1:1249-1251, 1981.
- Chick, J.; Lloyd, G.; and Crombie, E. "Natural History and Effect of Minimal Intervention in Newly Identified Problem Drinkers in a General Hospital." Paper presented at 33rd International Congress on Alcoholism and Drug Dependence, Tangier, Morocco, October 1982.
- Ciraulo, D.A., and Jaffe, J.H. Tricyclic antidepressants in the treatment of depression associated with alcoholism. *Journal of Clinical Psychopharmacology* 1(3):146-150, 1981.
- Ciraulo, D.A.; Lloyd, M.A.; Chapron, D.J.; Jaffe, J.H.; Subbarao, B.; and Kramer, P.A. Imipramine disposition in alcoholics. *Journal of Clinical Psychopharmacology* 2(1):2-7, 1982.
- Cole, S.G.; Lehman, W.E.; Cole, E.A.; and Jones, A. Inpatient vs. outpatient treatment of alcohol and drug abusers. *American Journal of Drug and Alcohol Abuse* 8(3):329-345, 1981.
- Costello, R.M. Alcoholism aftercare and outcome: Cross-lagged panel and path analyses. *British Journal of Addiction* 75:49-53, 1980.
- Costello, R.M., and Baillargeon, J.G. Reliability analysis of the treatment difficulty scale. *American Journal of Drug and Alcohol Abuse* 8(1):117-121, 1981.
- Cronkite, R.C., and Moos, R.H. Determinants of the posttreatment functioning of alcoholic patients: A conceptual framework. *Journal of Consulting and Clinical Psychology* 48(3):305-316, 1980.
- Cummings, C.; Gordon, J.R.; and Marlatt, G.A. Relapse: Prevention and prediction. In: Miller, W.R., ed. *The Addictive Behaviors: Treatment of Alcoholism, Drug Abuse, Smoking and Obesity.* New York: Pergamon Press, 1980. pp. 291-321.
- Den Hartog, G.L. "A Decade of Detox": Development of Non-Hospital Approaches to Alcohol Detoxification - A Review of the Literature. Substance Abuse Monograph Series. Jefferson City, Mo.: Division of Alcohol and Drug Abuse, 1982. 67 pp.
- Diesenhaus, H. Current trends in treatment programming for problem drinkers and alcoholics. In: National Institute on Alcohol Abuse and Alcoholism. *Prevention, Intervention and Treatment: Concerns and Models.* Alcohol and Health Monograph No. 3. DHHS Pub. No. (ADM) 82-1192. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 219-290.
- Edwards, G.; Arif, A.; and Hodgson, R. Nomenclature and classification of drug- and alcohol-

- related problems: A WHO memorandum. Bulletin of the World Health Organization 59(2):225-242, 1981.
- Edwards, G.; Gross, M.M.; Keller, M.; and Moser, J., eds. Alcohol-related problems in the disability perspective. Journal of Studies on Alcohol 37(9):1360-1382, 1976.
- Edwards, G.; Orford, J.; Egert, S.; Guthrie, S.; Hawker, A.; Hensman, C.; Mitcheson, M.; Oppenheimer, E.; and Taylor, C. Alcoholism: A controlled trial of "treatment" and "advice." Journal of Studies on Alcohol 38(5):1004-1031, 1977.
- Elkins, R.L. Covert sensitization treatment of alcoholism: Contributions of successful conditioning to subsequent abstinence maintenance. Addictive Behaviors 5(1):67-89, 1980.
- Fagan, R.W., and Mauss, A.L. Padding the revolving door: An initial assessment of the Uniform Alcoholism and Intoxication Treatment Act in practice. Social Problems 26(2):232-247, 1978.
- Feighner, J.P.; Robins, E.; Guze, S.B.; Woodruff, R.A.; Winokur, G.; and Munoz, R. Diagnostic criteria for use in psychiatric research. Archives of General Psychiatry 26:57-63, 1972.
- Ferrell, W.L., and Galassi, J.P. Assertion training and human relations training in the treatment of chronic alcoholics. International Journal of the Addictions 16(5):959-968, 1981.
- Finney, J.W., and Moos, R.H. Treatment and outcome for empirical subtypes of alcoholic patients. Journal of Consulting and Clinical Psychology 47(1):25-38, 1979.
- Finney, J.W., and Moos, R.H. Characteristics and prognoses of alcoholics who become moderate drinkers and abstainers after treatment. Journal of Studies on Alcohol 42(1):94-105, 1981.
- Finney, J.W.; Moos, R.H.; and Mewborn, C.R. Posttreatment experiences and treatment outcome of alcoholic patients six months and two years after hospitalization. Journal of Consulting and Clinical Psychology 48(1):17-29, 1980.
- Forsythe, A.B.; Griffiths, B.; and Reiff, S. Comparison of utilization of medical services by alcoholics and non-alcoholics. American Journal of Public Health 72(6):600-602, 1982.
- Frances, R.J.; Timm, S.; and Bucky, S. Studies of familial and nonfamilial alcoholism. Archives of General Psychiatry 37:564-566, 1980.
- Freedberg, E.J., and Johnston, W.E. Outcome with alcoholics seeking treatment voluntarily or after confrontation by their employer. Journal of Occupational Medicine 22:83-86, 1980.
- Fuller, R.K., and Roth, H.P. Disulfiram for the treatment of alcoholism: An evaluation in 128 men. Annals of Internal Medicine 90(6):901-904, 1979.
- Fuller, R.K., and Williford, W.O. Life-table analysis of abstinence in a study evaluating the efficacy of disulfiram. Alcoholism: Clinical and Experimental Research 4(3):298-301, 1980.
- Garvin, R.B.; Foy, D.W.; and Alford, G.S. A critical examination of gamma-glutamyl transpeptidase as a biochemical marker for alcohol abuse. Addictive Behaviors 6:377-383, 1981.
- Gibbs, L.E. The need for a new design for evaluating alcoholism treatment programs. Drug and Alcohol Dependence 8(4):287-299, 1981.
- Gideon, W.L.; Little, A.S.; and Martin, D.W. Evaluation of a training program for certified alcoholism counselors. Journal of Studies on Alcohol 41(1):3-20, 1980.
- Glaser, F.B., and Osborne, A.C. What we most like to know: Does A.A. really work? British Journal of Addiction 77(2):123-129, 1982.
- Googins, B., and Kurtz, N.R. Factors inhibiting supervisory referrals to occupational alcoholism intervention programs. Journal of Studies on Alcohol 41(11):1196-1208, 1980.
- Hatsukami, D.; Pickens, R.W.; and Svikis, D. Post-treatment depressive symptoms and relapse to drug use in different age groups of an alcohol and other drug abuse population. Drug and Alcohol Dependence 8(4):271-277, 1981.
- Heather, N., and Robertson, L. Controlled Drinking. London: Methuen, 1981. 294 pp.
- Helzer, J.E. "Methodologies and Data to Measure Numbers of Alcoholics." Paper presented at the 34th ADPA annual conference, Washington, D.C., August 30, 1983.
- Helzer, J.E.; Robins, L.N.; Croughan, J.L.; and Welner, A. Renard Diagnostic Interview: Its reliability and procedural validity with physicians and lay interviewers. Archives of General Psychiatry 38(4):393-398, 1981.
- Hesselbrock, M.; Babor, T.F.; Hesselbrock, V.; Meyer, R.E.; and Workman, K. "Never believe an alcoholic?" On the validity of self-report measures of alcohol dependence and related constructs. International Journal of the Addictions 18(5):593-609, 1983.
- Hesselbrock, M.N.; Hesselbrock, V.M.; Babor, T.F.; Stabenau, J.R.; Meyer, R.E.; and Weidenman, M. Antisocial behavior, psychopathology and problem drinking in the natural history of alcoholism. In: Goodwin, D.W.; Van Dusen, K.; and Mednick, S.A., eds. Longitudinal Studies of Alcoholism. Copenhagen, Denmark: Nijhoff Publishing Co., in press.
- Hesselbrock, V.; Stabenau, J.; Hesselbrock, M.; Mirkin, P.; and Meyer, R. A comparison of two interview schedules: The schedule for affective disorders and schizophrenia-lifetime and the National Institute of Mental Health Diagnostic Interview Schedule. Archives of General Psychiatry 39(6):674-677, 1982.
- Hinson, R.E., and Siegel, S. The contribution of Pavlovian conditioning to ethanol tolerance and dependence. In: Rigter, H., and Crabbe, J.C., eds. Alcohol Tolerance and Dependence. Amsterdam: Elsevier/North-Holland Biomedical, 1980. pp. 181-199.
- Holder, H.D., and Hallan, J.B. Medical Care and Alcoholism Treatment Costs and Utilization: A Five-Year Analysis of the California Pilot Project to Provide Health Insurance Coverage for Alcoholism. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1981. 57 pp.
- Institute of Medicine. Alcoholism, Alcohol Abuse, and Related Problems: Opportunities for Re-

- search. Publication IOM 80-04. Washington, D.C.: National Academy Press, 1980. 195 pp.
- Jones, K.R., and Vischi, T.R. Impact of alcohol, drug abuse and mental health treatment on medical care utilization: A review of the research literature. Medical Care 17 (12, Supplement): 1-82, 1979.
- Jones, S.L.; Kanfer, R.; and Lanyon, R.L. Skill training with alcoholics: A clinical extension. Addictive Behaviors 7(3):285-290, 1982.
- Kahn, M.W., and Stephen, L.S. Counselor training as a treatment method for alcohol and drug abuse. International Journal of the Addictions 16(8):1415-1424, 1981.
- Kaplan, R.F.; Meyer, R.E.; and Stroebe, C.F. The symptoms of alcohol withdrawal as predictors of behavioral and physiological responses to an ethanol stimulus. In: National Institute on Drug Abuse. Problems of Drug Dependence 1982. Louis S. Harris, ed. Research Monograph Series 43. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1983. pp. 273-279.
- Keil, T.J., and Esters, R.A. Treatment dropouts: The effect of client and ecological variables. International Journal of the Addictions 17(1): 95-108, 1982.
- Keller, M., ed. Research priorities on alcohol. Proceedings of a symposium sponsored by the Rutgers Center of Alcohol Studies and Rutgers University. Journal of Studies on Alcohol. Supplement No. 8, November 1979. 342 pp.
- Kissin, B. Patient characteristics and treatment specificity in alcoholism. In Idestrom, C.-M., ed. Recent Advances in the Study of Alcoholism. Amsterdam: Excerpta Medica, 1977. pp. 110-122.
- Kline, N.S.; Wren, J.C.; Cooper, T.B.; Varga, E.; and Canal, O. Evaluation of lithium therapy in chronic and periodic alcoholism. American Journal of the Medical Sciences 268(1):15-22, 1974.
- Kristenson, H. "Studies on Alcohol Related Disabilities in a Medical Intervention Programme in Middle-aged Males." Doctoral dissertation, Lund University, Sweden, 1982.
- Kurtz, N.R.; Googins, B.; and Williams, C. Clients' views of an alcoholism program. Labor-Management Alcoholism Journal 10(3):102-113, 1980.
- Longabaugh, R.; McCrady, B.; Fink, E.; and Stout, R. Cost effectiveness of alcoholism treatment in partial vs. inpatient settings: Six month outcomes. Journal of Studies on Alcohol, in press.
- Mariatt, G.A. The drinking history: Problems of validity and reliability. In: Meyer, R.E.; Babor, T.F.; Glueck, B.C.; Jaffe, J.H.; O'Brien, J.E.; and Stabenau, J.R., eds. Evaluation of the Alcoholic: Implications for Research, Theory and Treatment. NIAAA Research Monograph No. 5. DHHS Pub. No. (ADM)81-1033. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981. pp. 25-39.
- Mayfield, D.G., and Johnston, R.G.M. Screening techniques and prevalence estimation in alcoholism. In: Fann, W.E.; Karacan, L.; Pokorny, A.D.; and Williams, R.L., eds. Phenomenology and Treatment of Alcoholism. New York: Spectrum Publishers, 1980. pp. 33-44.
- McCrady, B.S., and Noel, N.E. "Assessing the Optimal Mode of Spouse Involvement in Outpatient Behavioral Alcoholism Treatment." Paper presented at the 16th Annual Meeting of the Association for the Advancement of Behavior Therapy, Los Angeles, November 1982.
- McIntosh, L.D. Alcohol-related disabilities in general hospital patients: A critical assessment of the evidence. International Journal of the Addictions 17(4):609-639, 1982.
- McLachlan, J.F.C., and Stein, R.L. Evaluation of a day clinic for alcoholics. Journal of Studies on Alcohol 43(3):261-278, 1982.
- McLellan, A.T.; Luborsky, L.; Woody, G.E.; and O'Brien, C.P. An improved diagnostic evaluation instrument for substance abuse patients. The Addiction Severity Index. Journal of Nervous and Mental Disease 168(1):26-33, 1980.
- McLellan, A.T.; Luborsky, L.; Woody, G.E.; O'Brien, C.P.; and Kron, R. Are the "addiction-related" problems of substance abusers really related? Journal of Nervous and Mental Disease 169(4):232-239, 1981.
- McMillan, T.M. Lithium and the treatment of alcoholism: A critical review. British Journal of Addiction 76(3):245-258, 1981.
- Meyer, R.E.; Babor, T.F.; Glueck, B.C.; Jaffe, J.H.; O'Brien, J.E.; and Stabenau, J.R., eds. Evaluation of the Alcoholic: Implications for Research, Theory and Treatment. NIAAA Research Monograph No. 5. DHHS Pub. No. (ADM)81-1033. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981. 409 pp.
- Miller, P. M., and Mastria, M.A. Alternatives to Alcohol Abuse: A Social Learning Model. Champaign, Ill.: Research Press, 1977. 190 pp.
- Miller, W.R., and Hester, R.K. Treating the problem drinker: Modern approaches. In: Miller, W.R., ed. The Addictive Behaviors: Treatment of Alcoholism, Drug Abuse, Smoking, and Obesity. New York: Pergamon Press, 1980. pp. 11-141.
- Miller, W.R.; Taylor, C.A.; and West, J.C. Focused versus broad-spectrum behavior therapy for problem drinkers. Journal of Consulting and Clinical Psychology 48(5):590-601, 1980.
- Moberg, D.P.; Krause, W.K.; and Klein, P.E. Post-treatment drinking behavior among inpatients from an industrial alcoholism program. International Journal of the Addictions 17(3):549-567, 1982.
- Moos, R.H.; Finney, J.W.; and Chan, D.A. The process of recovery from alcoholism. I. Comparing alcoholic patients and matched community controls. Journal of Studies on Alcohol 42(5):383-402, 1981.
- Morey, L.C., and Blashfield, R.K. Empirical classifications of alcoholism: A review. Journal of Studies on Alcohol 42(11):925-937, 1981.
- Mulford, H.A. On the validity of the Iowa Alcoholic Stages Index. Journal of Studies on Alcohol 41(1):86-88, 1980.
- Mulford, H.A., and Fitzgerald, J.L. On the validity of the Research Diagnostic Criteria, the Feighner Criteria, and the DSM-III for diagnosing al-

- coholics. Journal of Nervous and Mental Disease 169(10):654-658, 1981.
- National Institute on Alcohol Abuse and Alcoholism. National Drug and Alcoholism Treatment Utilization Survey. Executive Report. Rockville, Md.: NIAAA, September 1980. 9 pp. and appendixes.
- National Institute on Alcohol Abuse and Alcoholism. Special Population Issues. Alcohol and Health Monograph No. 4. DHHS Pub. No. (ADM) 82-1193. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. 446 pp.
- Neiderhiser, D.H., and Fuller, R.K. High-performance liquid chromatographic method for the determination of diethylamine, a metabolite of disulfiram, in urine. Journal of Chromatography 229(2):470-474, 1982.
- Oei, T.P.S., and Jackson, P. Long-term effects of group and individual social skills training with alcoholics. Addictive Behaviors 5:129-136, 1980.
- Oei, T.P.S., and Jackson, P. Social skills and cognitive behavioral approaches to the treatment of problem drinking. Journal of Studies on Alcohol 43(5):532-547, 1982.
- O'Farrell, T.J.; Harrison, R.H.; Schulmeister, C.A.; and Cutter, H.S. Closeness to Divorce Scale for wives of alcoholics. Drug and Alcohol Dependence 7(4):319-324, 1981.
- Ogborne, A.C., and Glaser, F.B. Characteristics of affiliates of Alcoholics Anonymous: A review of the literature. Journal of Studies on Alcohol 42(7):661-675, 1981.
- O'Leary, M.R.; Calsyn, D.A.; Haddock, D.L.; and Freeman, C.W. Differential alcohol use patterns and personality traits among three Alcoholics Anonymous attendance level groups: Further considerations of the affiliation profile. Drug and Alcohol Dependence 5:135-144, 1980.
- Olson, R.P.; Devine, V.T.; Ganley, R.; and Dorsey, G.C. Long-term effects of behavioral versus insight-oriented therapy with inpatient alcoholics. Journal of Consulting and Clinical Psychology 49(6):866-877, 1981.
- Orford, J.; Oppenheimer, E.; and Edwards, G. Abstinence or control: The outcome for excessive drinkers two years after consultation. Behaviour Research and Therapy 14(6):409-418, 1976.
- Pattison, E.M. Nonabstinent drinking goals in the treatment of alcoholism: A clinical typology. Archives of General Psychiatry 33(8):923-930, 1976.
- Pattison, E.M., and Kaufman, E. Family therapy in the treatment of alcoholism. In: Lansky, M.W., ed. Family Therapy and Major Psychopathology. New York: Grune & Stratton, 1981. pp. 203-229.
- Paulson, S.M.; Krause, S.; and Iber, F.L. Development and evaluation of a compliance test for patients taking disulfiram. Johns Hopkins Medical Journal 141(3):119-125, 1977.
- Plotnick, D.E.; Adams, K.M.; Hunter, H.R.; and Rowe, J.C. Alcoholism Treatment Programs Within Prepaid Group Practice HMOs: A Final Report. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1982. 130 pp. and appendixes.
- Polich, J.M.; Armor, D.J.; and Braiker, H.B. The Course of Alcoholism: Four Years After Treatment. New York: John Wiley & Sons, 1981. 334 pp.
- Pomerleau, O.F.; Fertig, J.; Baker, L.; and Cooney, N. Reactivity to alcohol cues in alcoholics and non-alcoholics: Implications for a stimulus control analysis of drinking. Addictive Behaviors 8(1):1-10, 1983.
- Pomerleau, O.F.; Pertschuk, M.; Adkins, D.; and Brady, J.P. A comparison of behavioral and traditional treatment methods for middle-income problem drinkers. Journal of Behavioral Medicine 1:187-200, 1978.
- Pond, S.M.; Becker, C.E.; Vandervoort, R.; Phillips, M.; Bowler, R.M.; and Peck, C.C. An evaluation of the effects of lithium in the treatment of chronic alcoholism. I. Clinical results. Alcoholism: Clinical and Experimental Research 5(2):247-251, 1981.
- Putnam, S.L. Alcoholism, morbidity and care-seeking: The inpatient and ambulatory service utilization and associated illness experience of alcoholics and matched controls in a health maintenance organization. Medical Care 20(1):97-121, 1982a.
- Putnam, S.L. Short-term effects of treating alcoholics for alcoholism: Utilization of medical care services by alcoholics in a health maintenance organization. The Group Health Journal 3(1):19-30, 1982b.
- Reyes, E., and Miller, W.R. Serum gamma-glutamyl transpeptidase as a diagnostic aid in problem drinkers. Addictive Behaviors 5:59-65, 1980.
- Richman, A., and Smart, R.G. After how many detoxifications is rehabilitation probable? Drug and Alcohol Dependence 7(3):233-238, 1981.
- Robichaud, C.; Strickler, D.; Bigelow, G.; and Liebson, I. Disulfiram maintenance employee alcoholism treatment: A three-phase evaluation. Behavior Research and Therapy 17(6):613-621, 1979.
- Rogghmann, K.J.; Roberts, J.S.; Smith, T.S.; Wells, S.M.; and Wersinger, R.P. Alcoholics' versus nonalcoholics' use of services of a health maintenance organization. Journal of Studies on Alcohol 42(3):312-322, 1981.
- Roman, P.M. Barriers to the use of constructive confrontation with employed alcoholics. Journal of Drug Issues 12(4):369-382, 1982.
- Roman, P. From employee alcoholism to employee assistance: De-emphases on prevention and alcohol problems in work-based programs. Journal of Studies on Alcohol 42(3):244-272, 1981.
- Ryback, R.S.; Eckardt, M.J.; Felsher, B.; and Rawlings, R.R. Biochemical and hematologic correlates of alcoholism and liver disease. Journal of the American Medical Association 248(18):2261-2265, 1982.
- Ryback, R.S.; Eckardt, M.J.; and Pautler, C.P. Biochemical and hematological correlates of alcoholism. Research Communications in Chemical Pathology and Pharmacology 27(3):533-550, 1980.

- Sanchez-Craig, M., and Walker, K. Teaching coping skills to chronic alcoholics in a coeducational halfway house: I. Assessment of programme effects. British Journal of Addiction 77(1):35-50, 1982.
- Saxe, L.; Dougherty, D.; Esty, K.; and Fine, M. The Effectiveness and Costs of Alcoholism Treatment. Health Technology Case Study 22. Report No. OTA-HCS-22. Washington, D.C.: Congress of the United States, Office of Technology Assessment, 1983. 108 pp.
- Schroeder, D.J.; Bowen, W.T.; and Twemlow, S.W. Factors related to patient attrition from alcoholism treatment programs. International Journal of the Addictions 17(3):463-472, 1982.
- Skinner, H.A. Different strokes for different folks: Differential treatment for alcohol abuse. In: Meyer, R.E.; Babor, T.F.; Glueck, B.C.; Jaffe, J.H.; O'Brien, J.E.; and Stabenau, J.R., eds. Evaluation of the Alcoholic: Implications for Research, Theory and Treatment. NIAAA Research Monograph No. 5. DHHS Pub. No. (ADM) 81-1033. Washington, D.C.; Supt. of Docs., U.S. Govt. Print. Off., 1981. pp.349-367.
- Skinner, H.A. Statistical approaches to the classification of alcohol and drug addiction. British Journal of Addiction 77(3):259-273, 1982.
- Skinner, H.A., and Allen, B.A. Alcohol dependence syndrome: Measurement and validation. Journal of Abnormal Psychology 91(3):199-209, 1982.
- Skinner, H.A.; Holt, S.; Allen, B.A.; and Haakonson, N.H. Correlation between medical and behavioral data in the assessment of alcoholism. Alcoholism: Clinical and Experimental Research 4(4):371-377, 1980.
- Skinner, H.A.; Holt, S.; and Israel, Y. Early identification of alcohol abuse: I. Critical issues and psychosocial indicators for a composite index. Canadian Medical Association Journal 124(9):1141-1152, 1981.
- Sokolow, L.; Welte, J.; Hynes, G.; and Lyons, J. Multiple substance use by alcoholics. British Journal of Addiction 76(2):147-158, 1981.
- Spicer, J.; Nyberg, L.R.; and McKenna, T.R. Apples and Oranges: A Comparison of Inpatient and Outpatient Programs. Center City, Minn.: Hazelden Foundation, 1981.
- Spitzer, R.L.; Williams, J.B.W.; and Skodol, A.E. DSM-III: The major achievements and an overview. American Journal of Psychiatry 137(2):151-164, 1980.
- Stanton, M.D., and Todd, T.C. The Family Therapy of Drug Abuse and Addiction. New York: Guilford Press, 1982.
- Steinglass, P. Family therapy with alcoholics: A review. In: Kaufman, E., and Kaufmann, P.N., eds. Family Therapy of Drug and Alcohol Abuse. New York: Gardner Press, 1979. pp. 147-186.
- Stockwell, T.; Hodgson, R.; Edwards, G.; Taylor, C.; and Rankin, H. The development of a questionnaire to measure severity of alcohol dependence. British Journal of Addiction 74:79-87, 1979.
- Strickler, D.P.; Bradlyn, A.S.; and Maxwell, W.A. Teaching moderate drinking behaviors to young adult heavy drinkers: The effects of three training procedures. Addictive Behaviors 6(4):355-364, 1981.
- Swint, J.M., and Nelson, W.B. Prospective evaluation of alcoholism rehabilitation efforts: The role of cost-benefit and cost-effectiveness analyses. Journal of Studies on Alcohol 38(7):1386-1404, 1977.
- U.S. Department of Health and Human Services. Fourth Special Report to the U.S. Congress on Alcohol and Health. DeLuca, J.R., ed. DHHS Pub No. (ADM)81-1080. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- Vaillant, G.E. Dangers of psychotherapy in the treatment of alcoholism. In: Bean, M.H., and Zinberg, N.E., eds. Dynamic Approaches to the Understanding and Treatment of Alcoholism. New York: Free Press, 1981. pp. 36-54.
- Valle, S.K. Interpersonal functioning of alcoholism counselors and treatment outcome. Journal of Studies on Alcohol 42(9):783-790, 1981.
- Vannicelli, M. Group psychotherapy with alcoholics: Special techniques. Journal of Studies on Alcohol 43(1):17-37, 1982.
- Wallace, J. Working with the preferred defense structure of the recovering alcoholic. In: Zinberg, S.; Wallace, J.; and Blume, S.B., eds. Practical Approaches to Alcoholism Psychotherapy. New York: Plenum, 1978. pp. 19-29.
- Weissman, M.M.; Meyers, J.K.; and Harding, P.S. Prevalence and psychiatric heterogeneity of alcoholism in a United States urban community. Journal of Studies on Alcohol 41(7):672-681, 1980.
- Welte, J.W.; Hynes, G.; Sokolow, L.; and Lyons, J.P. Comparison of clients completing inpatient alcoholism treatment with clients who left prematurely. Alcoholism: Clinical and Experimental Research 5(3):393-399, 1981.
- Wilkinson, D.A., and Sanchez-Craig, M. Relevance of brain dysfunction to treatment objectives: Should alcohol-related cognitive deficits influence the way we think about treatment? Addictive Behaviors 6(3):253-260, 1981.
- World Health Organization. Diagnosis and Classification of Mental Disorders and Alcohol and Drug-Related Problems. Summaries of working papers from an international conference, Copenhagen, Denmark, April 1982.
- Zinberg, S. Office psychotherapy of alcoholism. In: Solomon, J., ed. Alcoholism and Clinical Psychiatry. New York: Plenum Medical Books, 1982. pp. 213-229.
- Zivich, J.M. Alcoholic subtypes and treatment effectiveness. Journal of Consulting and Clinical Psychology 49(1):72-80, 1981.

Chapter VIII

Prevention: A Broad Perspective

This chapter will describe current perspectives on prevention of alcohol-related problems, summarize recent research findings, and highlight activities at national, State, and community levels to reduce alcohol-related problems.

Current Perspectives

Within recent years, prevention of alcohol-related problems has emerged as a key issue not only in the United States but internationally as well (Mosher 1981). There has been enormous growth of attention on the part of many countries as it becomes increasingly clear that few, if any, countries are free of problems associated with the consumption of alcohol. Legislators, educators, researchers, and others throughout the world are seeking ways to reduce alcohol-related problems and to decrease the personal, social, and economic costs associated with them.

The emerging focus on prevention is related to a number of factors. One is the realization that the treatment system alone cannot be expected to deal with all alcohol-related problems (USDHHS 1981). Another is that not only heavy chronic drinkers cause and suffer from alcohol problems. The National Academy of Sciences' report, Alcohol and Public Policy: Beyond the Shadow of Prohibition (Moore and Gerstein 1981), indicates that:

Chronic drinkers with high consumption both cause and suffer far more than their numerical share of the adverse consequences of drinking, (but) their share of alcohol problems is still only a fraction--typically less than half--of the total. Alcohol problems occur throughout the drinking population . . . at lower rates but among much greater numbers as one moves from the heaviest drinkers to more moderate drinkers. (p. 44)

In addition, significant grassroots groups concerned about alcohol problems have developed over the past few years. Some of these have focused national attention on and stimulated nationwide action to reduce alcohol-related highway accidents. Others have focused on education, mutual support groups, server responsibility, concern about alcohol beverage advertising, and support for additional enforcement and legislative actions that have prevention potential.

Empirical research on alcohol-related problems among special population groups (e.g., women, young black men, Hispanic Americans, American Indians and Native Alaskans, and the elderly) indicates a need for and has contributed to a growing interest in prevention efforts for specific at-risk populations.

Furthermore, research in a number of areas offers significant promise for future prevention efforts. These areas include:

- The effects of alcohol consumption on neurotransmitters in the nervous system
- Means for manipulating tolerance to alcohol in the brain
- Genetic factors or markers that may help to explain why individuals seem to differ in their vulnerabilities to the problems associated with alcohol consumption
- Role of known risk factors in the development of alcohol-related problems
- Assessment of the impact of legislative and regulatory measures
- Early identification and diagnosis of alcohol problems
- Increased awareness of the need to further evaluate various educational and community approaches

Since the Fourth Special Report to Congress (USDHHS 1981), there appears to have developed an emerging consensus that the public health approach can provide a useful conceptual framework for developing prevention strategies. This model derives from epidemiological studies of communicable disease indicating that proper planning requires knowledge not only of host, agent, and environment, but also of their interaction.

Concurrent with the growing acceptance of this approach, there has been a significant public and private effort directed toward health promotion and disease prevention—and growing evidence of effectiveness. Several recent publications have documented improvements in the Nation's health over recent years, as well as gains that may continue. Among these improvements are:

- The proportion of adults who smoke has declined by more than 20 percent over the past 15 years.
- The proportion of the population with high serum cholesterol levels has declined by 12-22 percent.
- The proportion of adults who exercise regularly has increased by as much as 100 percent. (USDHHS 1982)

Applied to alcohol-related problems, the public health approach directs simultaneous attention to (1) individuals at risk for developing problems, (2) the availability and distribution of alcohol, and (3) the drinking environment. Objectives of the Nation for 1990 include more than a dozen specifically related to alcohol (see box, page 124) and reflect attention to each of these three areas.

The Alcohol, Drug Abuse, and Mental Health Administration (1981) has defined primary prevention as actions or interventions designed to reduce the incidence of alcohol disorders and related problems. (Secondary prevention, or intervention, is concerned with the early identification and treatment of these disorders, to reduce their prevalence or the prevalence of dysfunctional behaviors associated with them.)

In 1981, the National Academy of Sciences completed a study (under contract to NIAAA) of alternative approaches to the prevention of alcohol-related problems. Based on this study, the Academy concluded that: The possibilities for reducing the (alcohol) problem are modest but real and should increase with experience

(Moore and Gerstein 1981). This section of the chapter will set forth findings of research on two major instruments for preventing alcohol-related problems: (1) education, information, and training and (2) law and regulation.

Education, Information, and Training

School-Based Alcohol Education Programs

Alcohol education programs for youths 18 years of age and younger have focused on both general unselected youth populations and those at risk for alcohol problems (Hewitt 1982). Alcohol education programs for the former have ranged from those delivering only alcohol-related information to those with little or no alcohol-related content, and those designed to foster the personal development of participants to reduce the likelihood of later alcohol problems. Several programs have combined alcohol-specific education with personal development (NIAAA 1979, 1981b).

Educational efforts also have been directed at groups of adults who interact with young people and who are in a position to influence their drinking (Hewitt 1982). Programs for parents and other family members, teachers, other community professionals, and lay people have been developed, on the assumption that providing these key "gatekeepers" with information on alcohol and alcoholism may well affect the youth for whom they are responsible.

Evaluations of alcohol education programs indicate that: (1) significant increases in knowledge about alcohol are often observed (Fullerton 1979; Staulcup et al. 1979; Wittman 1982); (2) less dramatic but nonetheless real changes in attitudes toward alcohol or toward self and others also occur, especially when attitude change is an explicit goal of the program (Evans et al. 1979; Weisheit et al. 1979; Wittman 1982); (3) difficult-to-interpret or modest changes in actual drinking behavior occur, although fewer programs set out to measure behavioral change than changes in attitudes toward or knowledge about alcohol (Fullerton 1979; Staulcup et al. 1979; Wittman 1982); and (4) although evaluation designs are becoming more sophisticated, future studies will need to develop measurable and precise program objectives and goals (Wittman 1982).

Elementary-age children.--This group of children has recently become the focus of in-

**1990 Objectives
Alcohol Abuse and Alcoholism**

1. By 1990, fatalities from motor vehicle accidents involving drivers with blood alcohol levels of .10 percent or more should be reduced to fewer than 9.5 per 100,000 population per year. (In 1977, there were 11.5 per 100,000 population.)
2. By 1990, fatalities from other (non-motor-vehicle accidents, indirectly attributable to alcohol use, e.g., falls, fires, drownings, ski mobile, aircraft) should be reduced to 5 per 100,000 population per year. (In 1975, there were 7 per 100,000 population.)
3. By 1990, the cirrhosis mortality rate should be reduced to 12 per 100,000 population. (In 1978, the rate was 13.8 per 100,000 population.)
4. By 1990, the incidence of infants born with fetal alcohol syndrome should be reduced by 25 percent. (In 1977, the rate was 1 per 2,000 deaths, or approximately 1,650 cases.)
5. By 1990, per capita consumption of alcohol should not exceed the current levels. (In 1978, about 2.82 gallons of absolute alcohol were consumed per year per person aged 14 and over.)
6. By 1990, the proportion of adolescents 12 to 17 years old who abstain from using alcohol (or other drugs) should not fall below 1977 levels. (In 1977, the proportion of abstainers was 46 percent for alcohol.)
7. By 1990, the proportion of adolescents 12 to 17 years old who report acute drinking-related problems during the previous year should be reduced to below 17 percent. (In 1978, it was estimated to be 19 percent based on 1974 data.)
8. By 1990, the proportion of problem drinkers among all adults aged 18 and over should be reduced to 8 percent. (In 1979, it was about 10 percent.)
9. By 1990, the proportion of women of childbearing age aware of risks associated with pregnancy and drinking--in particular, the fetal alcohol syndrome--should be greater than 90 percent. (In 1979, it was 73 percent.)
10. By 1990, the proportion of adults who are aware of the added risk of head and neck cancer for By 1990, the proportion of adults who are aware of the added risk of head and neck cancer for people with excessive alcohol consumption should exceed 75 percent. (No baseline data are available.)
11. By 1990, 80 percent of high school seniors should state they perceive great risk associated with alcohol intoxication. (In 1979, only 35 percent perceived "great risk" associated with having five or more drinks per occasion once or twice each weekend.)
12. By 1990, the proportion of workers in major firms whose employers provide a substance abuse prevention and referral program (employee assistance program) should be greater than 70 percent. (In 1976, 50 percent of a sample of the Fortune 500 firms offered some type of employee assistance program.)
13. By 1990, a comprehensive data capability should be established to monitor and evaluate the status and impact of misuse of alcohol (and drugs) on: health status, motor vehicle accidents, accidental injuries in addition to those from motor vehicles, interpersonal aggression and violence, sexual assault, vandalism and property damage, pregnancy outcomes, and emotional and physical development of infants and children.

NOTE: Acute drinking-related problems have been defined as problems such as episodes of drunkenness, driving while intoxicated, or drinking-related problems with alcohol authorities.

creased prevention activities. Bartlett's (1981) analysis of alcohol education programs and their impact on elementary and high school age youth cautions against overemphasizing cognitive learning and lecture-oriented teaching methods, failing to assist students' receptivity to the material and to appreciate the problems caused by the "captive population" syndrome, and overlooking the importance of coordinating alcohol education programs with related community resources. Nontraditional programs that involve students in curriculum design and interactive learning seem most successful.

One innovative prevention approach to grade-school children--the Alpha Center Prevention Model--is in place in two Florida counties (Pringle et al. 1981). This model is based on the assumption that alcohol abuse, like other behavioral problems, derives from early developmental problems. Students aged 8 to 12 who display maladaptive behaviors at home and school attend special classes to improve social and academic skills. Their teachers learn classroom behavior management techniques and interpersonal communication skills, and a counselor works with parents at home. The Alpha Center concept remains essentially unproven, however.

Adolescents.--Most youth begin to drink in adolescence. This is also the time when youth are most at risk for the most catastrophic consequences of drinking: alcohol-related traffic accidents.

A recent study on adolescent alcohol abuse relevant to prevention efforts found that (1) alcohol is the most widely used drug by youth between the ages of 12 and 17 (making this group an obvious prevention target), (2) problem drinking increases sharply with advancing age during adolescence, (3) adolescent problem drinkers use illicit drugs much more often than nondrinkers, (4) heavy drinking during adolescence is typically accompanied by other antisocial behaviors, (5) both parental and peer influences strongly affect problem drinking, and (6) adolescent problem drinkers differ from non-problem-drinking adolescents--they are more tolerant of deviance, less successful in school, and prize more highly independence than academic success (Braucht 1982). Research on self-reported drug usage and problem behaviors indicates significant associations between such behaviors and use of alcohol, tobacco, illicit drugs, painkillers (e.g., aspirin), and tea or coffee (Hundleby et al.

1982). These findings emphasize the complex phenomena that prevention programs for youth should address, as well as the special characteristics and changing needs of the adolescent problem drinker.

The CASPAR program in Somerville, Massachusetts, took this complexity into consideration when it included teacher training, peer teaching programs, and an alcohol-specific curriculum to teach decisionmaking, problem solving, and coping skills. It found substantial increases in knowledge and improved attitudes. The peer leadership training was found effective in raising levels of self-esteem, feelings toward home environment, and optimism. A significant inverse relationship was found between levels of self-esteem and likelihood of problem drinking (NIAAA 1979). Changes in drinking behavior did occur in the experimental group as compared with the control group, which was not exposed to the program over a period of 3 years (Wittman 1982). These effects decayed when junior high youth moved into the high school, however. The experimental group showed as high a level for alcohol-related problems as did the control group in high school (NIAAA 1979).

The Here's Looking At You curriculum program in Seattle, Washington, found similar results with a multifaceted program that paid particular attention to the alcohol discussion content of the curriculum in relation to the developmental age of the child. The most encouraging of the findings was that for the age cohort sixth-eighth grade (the longitudinal subsample first measured while they were in sixth grade and then during the succeeding 2 years), the impact from the curriculum was found not only in knowledge, but also in increasing self-esteem and, in some, improving decisionmaking skills (NIAAA 1981b). Further study of this curriculum is expected to yield more knowledge about the impact of the curriculum on intervening variables and actual behavioral outcomes.

One program for high-risk youth at the high school level has proven effective in terms of behavioral change. The Student Assistance Program, implemented in 23 schools in Westchester County, New York, has a primary prevention component aimed at newcomers, seniors, children of alcoholics, and young people with interpersonal problems. Using small groups facilitated by a trained counselor, the program has proven effective in reducing the incidence of both alcohol problems and absenteeism (often associated with alcohol and other drug problems) (NIAAA 1983).

Another innovative program focusing on a high-risk group of delinquent adolescents included an intensive training program for the staff of the Partners Program, a nonprofit volunteer organization designed to provide services to youths in trouble with the law in Denver, Colorado (Resource Alternatives Corp. 1982). Another essential component of the program was peer education. The program's evaluation study design provided for three control groups, all of whom were enrolled in the program before the alcohol education/prevention training component was provided to the staff. Results of the evaluation revealed that youths' perceptions of the negative consequences of drug and alcohol use increased after their counselors had been exposed to alcohol education. More important, the experimental groups experienced less severe alcohol and other drug problems than did the control group (NIAAA 1981a).

A project conducted jointly by the American Medical Association and the American Association of Motor Vehicle Administrators (Hames and Petrucelli 1980) was based on the assumption that teenagers in the process of getting their driver's licenses are maximally motivated to cooperate with an alcohol education program attached to that process. The program required teenagers applying for license in New York, Wisconsin, or Oklahoma to view an alcohol education film in high school driver education classes, after which they were tested for knowledge increase and retention. Results indicated that the information was retained at least for a short time; however, the impact of the program on subsequent rates of drunken driving was not assessed.

One of the most thorough studies of the impact of alcohol education on adolescents (Stuart 1980) included lectures given to almost 1,000 junior and senior high school students on the physiology and pharmacology of drugs including alcohol and the legal, social, and psychological ramifications of their use. While information levels increased, drug and alcohol use also increased.

A study by Goodstadt and his colleagues (1982) found that educating 7th to 10th grade students on myths about alcohol, beverage advertising, reasons for drinking, and effects of alcohol on the family, driving, sports, fitness, and sexuality improved their knowledge but had mixed effects on their attitudes toward alcohol. Moreover, during the program, the students decreased their alcohol consumption. Since this is one of the few adolescent

alcohol education projects that assessed behavior change and clearly found evidence of change, it deserves careful scrutiny and possible replication.

Goodstadt and Sheppard (1983) also recently compared three short-term programs--a cognitive program, a decisionmaking program, and a values-clarification program--for high school students. The cognitive program improved levels of alcohol knowledge significantly more than the other programs, even though there was no change in behavioral expectations or attitudes. The decisionmaking program was successful in leading students to a better understanding of major decisionmaking concepts, demonstrating the viability of this program in teaching about decisionmaking. The values-clarification program was most likely not appropriate for the target audience, so no generalizations could be made.

The authors did draw, however, some significant conclusions. They found, for instance, that while attitudes were not changed by these programs, attitude change is positively related to reduced alcohol consumption. They also found that those who improved in their use of decisionmaking concepts were more likely to show a shift toward less pro-alcohol attitudes. They concluded that "whereas behavioral outcomes are a desirable objective, they probably represent unreasonable expectations for interventions of such short duration, although they would continue to be significant long-term objectives and worthy of examination" (Goodstadt and Sheppard 1983).

College-age youth.--An effort to stimulate prevention and intervention programs has been made at the college level in recognition of both the paucity of such programs to this time and the differences in approach and strategy required for effective prevention and intervention with this population (Bryan 1982; Dean 1982).

Several interesting approaches to prevention with college-age youth have been reported. A survey of 457 Memphis State University students and 30 mental health professionals working in the university setting revealed that the students, unlike the professionals, considered substance abuse problems to be the most serious of 24 mental health problems experienced by students (Henggeler et al. 1980). Evaluations of alcohol education programs at the University of Virginia (Portnoy 1980) and Saint Mary's College (Leavy 1980) both demonstrated the apparent ease with which the student's information, based on al-

cohol knowledge, can be changed--and the real difficulty in changing actual drinking behavior. Similar results were obtained at Brown University by a "sons and daughters of alcoholics" group designed as a prevention and intervention strategy (Donovan 1980).

Between 1975 and 1980, a comprehensive alcohol education program for college students was developed, implemented, and evaluated at the University of Massachusetts (Kraft 1982) and somewhat later replicated and extended at the University of North Carolina (McCartney and Humbert 1983; Mills et al. 1983). These two projects were among the most carefully evaluated of all large-scale prevention projects directed at this population of drinkers. The University of Massachusetts project programmed mass media messages to raise awareness of drinking and driving, and sponsored group discussions and community development techniques designed to influence ways in which alcohol was consumed by college students. As a result of these efforts, knowledge and attitude changes took place; however, no positive changes in drinking behavior were observed (Kraft 1982). The evaluation design, however, was invalidated, since during the program the minimum purchase age was lowered in Massachusetts. It is impossible to know, therefore, if behavioral changes occurred as a result of the program.

Mass Media Campaigns

The consensus about mass media information campaigns is that: (1) such efforts are largely limited to increasing knowledge and reinforcing established attitudes and behavior patterns (Blane and Hewitt 1977), and (2) slight alteration on changing drinking patterns may occur, although this effect is indeterminate (Blane 1976). However, Wallack (1981) notes that:

A review on the effects of mass media would lead one to a healthy skepticism regarding the effectiveness of such a program implementation technique. This skepticism must be tempered because of the flawed evaluations which have generated the body of effectiveness data....It may be that determining the effects of mass media is a function of broadening research questions to gain better understanding of the wider view, rather than narrowing them further to assess individual exposure and reaction in greater detail (p. 230)

NIAAA's recent mass media campaign clearly indicates that such efforts can stimulate interest in both the prevention of alcohol problems and communitywide organization and development toward this end (NIAAA 1982).

Education Programs About Other Health Hazards

Recently, attention has been devoted to determining what can be learned about prevention of alcohol-related problems from carefully developed programs intended to reduce other health hazards (Hochheimer 1981). And some have concluded that the most promising prevention technique for the 1980s is the community-based model that blends a variety of approaches aimed at the host, agent, and environment to address the complexities and needs specific to the community (Hochheimer 1981; Wittman 1982; Holder and Blose 1983).

One design that combined mass media campaigns with intensive interpersonal training at the local level was the Stanford Heart Disease Prevention Three Community Study. This study had the following five goals: generate awareness about the program and its focus, increase the knowledge of the audience, motivate people to adopt new behaviors, teach people new skills, and reinforce the new skills and behaviors so they would be maintained (Hochheimer 1981). Their findings showed that dietary changes and exercise were learned through mass media alone (Maccoby and Farquhar 1975). However, other behaviors, such as smoking, changed only when interpersonal training (based on the social learning and behavior work of Bandura 1977, 1978, 1979, and Goodstadt 1978) was included (McAlister et al. 1979). After 2 years, the overall decrease in risk of heart disease was 16-18 percent in the experimental sites, compared with 6.5 percent for the control group.

In Project CLASP (Counseling Leadership About Smoking Pressures), which was based on previous smoking and health research, McAlister and colleagues (1979) trained several teams of five to seven high school students to lead classroom sessions for junior high school students. The objective of the sessions was to increase students' commitment not to start smoking and to psychologically "inoculate" them against pressures to smoke. Similar activities aimed at deterring the use of alcohol and other drugs also were added. When the experimental and control schools were compared following 21 months of longitudinal ob-

servation, the experimental school's students were found to be smoking and drinking significantly less than students in the control school, who had received an intensive course in health education but no peer-group sessions with high school students. Further evaluation is needed to see if this effect persists over time and to gain a better understanding of these efforts.

Other prevention programs based on social learning theory (Bandura 1977) emphasize strategies encouraging young people to refuse drugs. Significant positive results have been found in university-based research and demonstration programs aimed primarily at smoking prevention. Other research has supported the assumption that rehearsing responses to detrimental health influences increases the likelihood that these responses will be used in appropriate situations (McGuire 1969).

Most refusal programs incorporate three components: strategies that increase young people's knowledge by providing information about both the hazards of drugs and their effects, and about peer pressure and other influences to use drugs; techniques for expanding one's specific behavior repertoire for saying "no"; and training in life skills (e.g., values clarification, enhancing self-esteem, assertiveness training, problem solving, and coping abilities). As a wide body of research has shown, each plays a part in a person's choice to adopt either life-enhancing or health-risking behaviors (Bandura 1969; Rotter 1972; Jessor and Jessor 1977; Perry 1981).

In one such program--Project SMART at the University of Southern California--the principal objective has been to implement and study a school-based prevention program designed to offset peer pressure through the use of positive strategies to refuse drugs and thereby reduce cigarette smoking and alcohol and marijuana use in the sixth through ninth grades. In Project SMART, two curriculums have been developed; one focuses on social resistance skills training, and the other stresses self-management skills and values clarification. Both use peer leaders. Pretest findings indicate that it may be important to begin the intervention at a point where the target audience begins to perceive itself at risk, and that youth prevention programs are more effective when facilitated by peers (Johnson et al. 1981).

In the Life Skills Training Program, a similar program developed at the Cornell University Medical School, students are informed about myths and realities relating to smoking.

In addition, they are assisted in improving self-image, making informed choices, and resisting pressure to smoke. Advertising techniques, verbal and nonverbal communication skills, and appropriate and effective use of assertiveness also are examined. The material in each of these sessions is transmitted through discussion, demonstration, and role playing, and often is not smoking-specific, although connections are made where appropriate. Students also participate in a self-improvement program in which they identify a skill they want to improve or a behavior they want to change and then set long- and short-range goals for accomplishing this. Botvin and Eng (1982) have found that the program increased knowledge and reduced by 58 percent the onset rate of smoking. In addition, the experimental group expressed less social anxiety and were more resistant to influences than the control group.

Wellness programs may offer additional opportunities for alcoholism prevention at the worksite. Wellness programs--positive lifestyle change programs--offer healthy employees the opportunity to modify lifestyles that might, at a future date, cause health problems. Smoking cessation, exercise, stress management, weight control, and nutrition programs are typically offered in the effort to promote healthier lifestyles, improve quality of life at present, and reduce health risks in the future. A number of large corporations, including Johnson & Johnson and Control Data, have put wellness programs into place. These programs may, but typically do not, provide alcohol components, relying instead on existing employee assistance programs (EAP) to reach employees with alcohol problems. When no EAP exists to address stigmatized problems, its inclusion may greatly enhance the company's human wellness program. However, the existence of comprehensive positive lifestyle change programs may affect drinking practices and general awareness of healthy lifestyles, thereby preventing alcohol problems from developing in some individuals. Empirical studies of the impact of these programs on the prevention of alcohol problems are necessary before their role and widespread implementation in the worksite can be determined.

Programs for Populations at Special Risk

Women.--In the past, few prevention approaches have been designed specifically for women. These usually provided assertiveness training, exercises for enhancing self-esteem,

and stress management seminars (Irwin 1976; Sandmaier 1976). To date, neither the underlying assumptions nor the program effectiveness of these types of programs have been confirmed empirically.

One program currently being evaluated is the Alcoholism Center for Women, Los Angeles County, which recently developed a systematic community approach for identifying and delivering prevention activities to adult women at high risk for alcohol problems. Special attention was paid to developing a method to identify populations of high-risk women and their psychological and sociological predisposition to alcohol abuse. Using this information, the Alcoholism Center designed and implemented a multimedia, multifaceted prevention strategy, which is being tested.

Minorities.--For many years, accurate data on the nature and extent of alcoholism among ethnic and racial minorities within this country were unavailable, making the task of targeting prevention efforts within and between these groups difficult. Now, however, better data are available on alcoholism and problem drinking among black, Asian, Hispanic, and Native Americans. An important issue related to understanding variations among ethnic groups in use of alcohol and incidence of alcohol problems has been whether these variations derive more from sociocultural, biochemical, or physiological differences. Although current data attribute predominant influence to sociocultural differences (Schaefer 1982), the determination of significant genetic contributions to alcoholism risk indicates the need to more fully examine biochemical and physiological parameters. Thus, a final answer to the question awaits more sensitive bioassay methods and more sophisticated sociocultural survey designs.

While drinking behavior of youth and the elderly in the black community remains largely undocumented, the findings from a few prevention programs for black youth (Crisp 1980; Miranda 1981) and black women (Gaines 1976) have been reported. Such projects need to be studied in depth to empirically assess their effectiveness. Of particular concern for prevention programmers is the fact that young black males (aged 25-34) in metropolitan areas are as much as 10 times more likely than young white males of the same age to die from cirrhosis of the liver. This disproportionately increased rate remains higher for black males until age 65 (Malin et al. 1982). In addition, although it appears that blacks drink less than

whites, when they drink they drink more heavily, thus increasing the likelihood of certain negative consequences associated with heavy drinking (Roizen 1982).

Rates of alcoholism among Hispanic Americans appear to be higher than the national average (Garza 1979). Variables that may explain these elevated rates of alcohol abuse include sociocultural norms and values that encourage heavy drinking, especially by men, minority group status, problems of acculturation and their associated stresses, and possible biochemical and physiological factors. Causal relationship between these factors, singly or together, and alcoholism, in either individuals or groups of individuals, has to this time only been inferred. Hispanic Americans, like other minority groups, underutilize treatment and rehabilitation resources, and few prevention programs have been designed for them (Alcocer 1982).

Elderly persons.--Strong evidence exists that the percentage of drinkers begins to decline at age 50, and at age 60 to 65, the percentage of heavy drinkers among males drops even more sharply. From age 50 on, however, some older people who have been lifelong abstainers or moderate social drinkers become problem drinkers (NASADAD 1981; Brody 1982). But the negative attitudes of many mental health professionals toward older persons seeking help make the prevention and treatment of alcoholism in the elderly very difficult. One solution may be support for self-help groups--older persons helping those in need--as well as a greater emphasis on preretirement planning that may affect use and abuse of alcohol (Gomberg 1982). Loss, such as that reflected in retirement, appears to be an impetus for developing alcohol problems in this age group. In addition, isolation is of particular significance for prevention programmers, e.g., how to reach the large numbers of women in this age group who have experienced loss and who are living alone.

An assessment model to help plan treatment and prevention services for the elderly has been proposed (Kola and Kosberg 1981). Designed to consider the special needs of the elderly for prevention and treatment services, the model recognizes the importance of integrating these services into existing community treatment and prevention resources. An example of a self-help program effectively integrated into its community is located at the Eagleview Hospital and Rehabilitation Center, where it began drug and alcohol prevention

services to the elderly in Montgomery County, Pennsylvania, in 1978 (Leigh 1980).

Infants.—Alcohol consumption during pregnancy poses a significant risk for the unborn child, with consequences including increased spontaneous abortion, decreased birth weight, major and minor birth defects, and mental retardation.

These effects of alcohol on the developing human fetus are detailed elsewhere in this volume (see chapter 5). Some occur in children whose mothers, during pregnancy, consume alcohol in amounts consistent with a diagnosis of alcoholism. Some occur in children whose mothers drink moderately. All of them are completely preventable.

A variety of prevention approaches will be necessary to reach social or moderate drinking women and women who drink heavily. The Surgeon General's health advisory on the risks of alcohol consumption during pregnancy, issued in July 1981 (FDA), pointed up the need to prevent alcohol-derived birth defects. This advisory urged physicians and other health professionals to advise patients who are pregnant or considering pregnancy not to drink alcoholic beverages.

One of the best documented fetal alcohol syndrome (FAS) prevention programs is the Fetal Alcohol Syndrome Demonstration Program at the University of Washington (Little et al. 1980; McIntyre 1980). This program aimed at providing model public and professional education on FAS, clinical services for pregnant women, assessment services for FAS children, and ongoing evaluations of these activities. Compared with a control community, there was a significant increase in the number of women who agreed to become abstinent during pregnancy. However, the program was less successful in reaching the pregnant "excessive drinker" who was drinking at levels reported to pose a risk to the fetus. Thus, it may be indicated that some women who are exposed to programs on alcohol and pregnancy will decrease their drinking, while other women who drink more heavily may require specialized services such as those offered by the obstetrical community in terms of screening techniques for early intervention and subsequent referral for treatment (Little in press).

Other efforts—one in Goteborg, Sweden, and one in Boston—have reported highly successful outcomes utilizing a secondary prevention approach (Olegard et al. 1979; Rosett et al. 1980, 1983). During their first prenatal

visit to an obstetric clinic, women fill out a structured health history that elicits information on alcohol use as well as other items. Women who reported consuming more than 45 drinks per month were counseled in a nonconfrontative manner about the benefits of decreasing alcohol consumption for having healthier babies. Fetal outcomes were markedly superior for those heavy drinking women who decreased alcohol consumption or abstained during the remainder of the pregnancy to those for heavy drinkers who did not change their drinking behavior. Of particular interest, the Boston study tentatively concluded that either the later pregnancy period is most crucial for alcohol teratogenicity, or reversibility of injury is possible if heavy drinking is terminated early enough during the pregnancy course. Further work may clarify exactly when prevention efforts can be implemented for optimal effectiveness.

A recent cross-sectional study compared drinking patterns during pregnancy over a 6-year interval (Streissguth et al. 1983). Different samples of pregnant women were interviewed in 1974 and 1975 and in 1980 and 1981. In the interim period, the study population was exposed to public media and educational programs on alcohol and pregnancy. Although the proportion of women drinking during pregnancy was found to have decreased during the 6-year interval, the proportion of women drinking at least an ounce of absolute alcohol per day was relatively constant. Since it is precisely this limited proportion of the population that incurs the highest risk of alcohol-related birth defects, these results highlight the need for more intensive prevention efforts.

Law and Regulation

Since the publication of the Fourth Special Report to Congress (USDHHS 1981), a number of studies have been published, exploring the implications of legislative and regulatory approaches for the prevention of alcohol-related problems. The findings of these studies are summarized here.

International Studies

In collaboration with the World Health Organization (WHO), the International Study of

Alcohol Control Experiences (ISACE) reviewed historical patterns of alcohol consumption, problems, policies, and their effects in Finland, Ireland, the Netherlands, Canada, Poland, Switzerland, and the United States (specifically, California).

The study, entitled Alcohol, Society and the State (Makela et al. 1981), found that "growth in alcohol consumption was accompanied by an increase in a broad variety of problems related to drinking...This holds not only for consequences of prolonged drinking, but for social and health problems related to single drinking episodes as well." Noting that "from a policy perspective it is perfectly legitimate to be interested in the autonomous impact of alcohol control on consumption and problems," the authors state that "from a historical perspective, the notion of effect is problematic.... Most of the time, changes in policy measures have, in fact, been one facet of the same general social processes, and changes in consumption, another." (p. 109)

The report also concluded that (1) "a reorganization of responsibilities among branches of government may be required to achieve a successful combination of economic, health, and social considerations;" (2) "preventive alcohol policies should...be given a high priority as an alternative to the morally inspired control of problem drinkers;" (3) "policy changes on alcohol should be made with caution and a sense of experiment;" (4) "while control of availability should receive a high priority, it is important to consider governmental powers to manipulate the environment of drinking so as to lower the risk of adverse consequences" (e.g., traffic fatalities and alcohol-related violence); (5) "international organizations should give close attention to existing and future trade policies and arrangements with a potential for affecting the availability of alcoholic beverages" (Makela et al. 1981, pp.110-112).

In June 1982, the National Academy of Sciences published the proceedings of an Institute of Medicine (IOM)-supported inter-American workshop entitled Legislative Approaches to Prevention of Alcohol-Related Problems. The purpose of this workshop was to explore the issue of economic growth, alcoholism, and alcohol-related problems by reviewing available data and the experiences of other countries to help policymakers in the Americas understand the complexities governing public policy decisions. The consensus emerging from the conference participants was that: (1) the cost to society from episodic

alcohol intoxication in the Americas is the most neglected alcohol issue; (2) increased interest in group-oriented measures aimed at preventing alcohol-related problems should be developed; (3) available empirical evidence suggests that a number of prevention-oriented, legislatively based policies should integrate public health and economic development benefits and costs with the social, cultural, and political societal values; (4) public and governmental awareness must precede implementation of effective policies; and (5) while the experiences of one country can assist others, a country's alcohol-related prevention strategies must be shaped by its own unique social, cultural, political, and economic situations (Institute of Medicine 1982).

The ANSVAR Mutual Insurance Company, in connection with the 1981 annual conference of the International Council on Alcohol Addictions, published Alcohol in the World of the 80's: Habits, Attitudes, Prevention Policies and Voluntary Efforts (Armyr et al. 1982). This broad inquiry detailed conditions in 23 countries or groups of countries with respect to drinking habits, alcohol control policy, economic issues, and efforts from the voluntary sector. This review concurred with the findings of ISACE and also concluded that, in the coming 5 years, alcohol consumption was more likely to increase than decrease, along with many indicators of alcohol-related problems.

Taxation

As the ISACE study noted, there is a "relatively large econometric literature on the relation of alcohol taxes and prices to consumption" (Makela et al. 1981, p. 89). Recent studies attempt to address more clearly the relationship of alcohol taxes to alcohol-related problems.

In particular, Cook and Tauchen (1982) estimate that an increase in the liquor excise tax by \$1 (1967 prices) per proof gallon (viz., 64 ounces of ethanol) would reduce the liver cirrhosis mortality rate by 5.4 percent in the short run and perhaps by twice that amount in the long run. Cook also offers a historical example: When France imposed stringent wine rationing in 1942, the cirrhosis mortality rate in Paris fell from 35/100,000 in 1941 to 6/100,000 in 1945-46, only to return to its previous higher levels when rationing ceased in 1948. Other work (Mello 1972; Nathan and Lisman 1976) offers clinical evidence that

alcoholic persons do reduce their alcohol consumption as a function of the beverage costs.

Cook (1981) also found that increases in the tax rate of spirits will reduce both the auto fatality rate and the cirrhosis rate. Smith (1981) draws the same conclusion. Two reviews of these relationships worldwide (WHO 1979; Colon 1980) suggest that while alcoholic beverages behave as other market commodities do (in that consumption is affected by price), there are also complicated interactions between the availability of alcohol supplied by the distribution network and the pricing of alcohol.

Cook and Tauchen (1981) also note that the Federal tax on alcoholic beverages has remained constant in nominal terms since 1951. Thus, the real price of alcoholic beverages has actually declined in recent years, such that between 1960 and 1980 the real price of liquor declined 48 percent, beer 27 percent, and wine 20 percent.

On balance, the weight of opinion (Cook 1981) appears to be that consumption is affected by price, and data exist in support of this view. Moreover, the previously held view that reducing overall consumption does not affect consumption by the heaviest drinkers (Makela 1975) is being challenged and re-evaluated (Makela 1980; Smart 1982). Many countries already have adopted the view that increasing the price of alcoholic beverages is a viable control strategy. Sweden (Somervuori 1977), Finland (Koski 1977), Australia (Luey 1979), Poland (Malec 1980), and some European Common Market countries (Sulkunen 1978) employ some form of price policy as a consumption control.

Minimum Drinking Age

There is considerable evidence that increasing the legal age for possession, purchase, consumption, or sale of alcoholic beverages can be an effective measure to reduce drunk driving accidents among youth (Douglass et al. 1974; Wagenaar 1980; Wagenaar et al. 1981; Maxwell 1981; Williams et al. 1983; Hingson et al. 1983). A study by Wagenaar (1980) analyzed a random sample of 20 percent of all reported accidents (i.e., fatal, injury, and property damage) in Michigan from January 1972 to December 1979. It concluded that:

Controlling for trends, seasonally, and other patterns in the frequency of police-

reported 'had been drinking' (HBD) crash involvement among 18-20-year-old drivers, an [annual] reduction of 31 percent occurred in the first 12 months after the drinking age was raised from 18 to 21 in December of 1978. Analyses of late-night, single-vehicle crashes with a male driver, of which a majority have been consistently identified as involving a drinking driver, reveal a significant reduction of 18 percent among drivers aged 18-20 after the higher legal drinking age was implemented. (Wagenaar 1980, p. 148)

In another study (Maxwell 1981), made after Illinois raised its minimum legal drinking age in January 1980 from 19 to 21 years, data for single-vehicle, nighttime, male-driver involvements occurring between 8 p.m. and 3 a.m. were used as a surrogate for alcohol-involved accidents. This study, comparing 1980 with 1979 accident data, concluded that raising the legal drinking age law was effective in reducing the single-vehicle, nighttime, male-driver involvement for drivers aged 19 and 20. For 1980, the percentage of reduction attributable to the law change was 8.8 percent.

The Michigan and Illinois studies looked at all accidents—fatal, injury, and property damage. Another study (Williams et al. 1983), using data from the Fatal Accidents Records System (FARS) of the National Highway Traffic Safety Administration, looked at nine States that raised their legal minimum age. Eight of the nine States experienced a reduction in nighttime fatal crash involvement among drivers in the affected age group; the average annual reduction was 28 percent. A recent study (Wagenaar et al. 1981) on the effects of raising the minimum age in Michigan and Maine concluded that Michigan drivers aged 18-20 experienced a net reduction of approximately 20 percent in the frequency of involvement in alcohol-related injury-producing crashes due to a raised drinking age from 18 to 21; in Michigan, there was a 17-percent decrease in alcohol-related crashes damaging property for the same age group; and in Maine, 18- to 19-year-olds were involved in 20 percent fewer alcohol-related crashes damaging property.

There also is evidence that contiguous States with differential minimum drinking ages create problems with cross-border purchase of alcohol by youth (Lillis et al. 1982). Based on these studies, the National Transportation Safety Board recommended in 1982 that all States raise the minimum legal age for

drinking or purchasing all alcoholic beverages to 21. Several States have done so, and many introduced this kind of legislation in 1982 and 1983.

Highway Safety

In seeking to reduce injuries and fatalities from alcohol and highway traffic accidents, the legislative area has received increased attention. New laws are mandating stringent minimum penalties for intoxicated driving offenders and supporting more intensive enforcement of existing intoxicated driving laws. These legislative approaches are being taken to establish new limits on driving and drinking and to decrease the incidence of driving while intoxicated.

A recent review of intoxicated driving literature (Borkenstein 1981) suggested that the role of alcohol in traffic accidents can be curtailed by (1) reducing per capita alcohol consumption (by increasing price or raising drinking age); (2) constructing streets and highways that place fewer demands on drivers so the effects of alcohol are not as severe; and (3) increasing enforcement to bring about general deterrence of drinking and driving.

In Survey and Deterrence of the Drinking Driver: An International Survey, Ross (1981) reviewed the experience of various countries in adopting the Scandinavian model, which combines the principles of deterrence with laws related to drinking and driving. He concludes that "enactment and enforcement of drunk driving laws are most successful in their initial deterrence of drinking and driving. When certainty of punishment for drinking and driving violations is low, however, this initial deterrent effect disappears. A reasonable interpretation of the results of this review is that Scandinavian-type laws deter when initiated because of exaggerated perceptions of the risk of apprehension and punishment. Since they appear to increase the real risks much more moderately, the deterrent accomplishment rests not on a firm foundation, but rather on a temporary scaffold that becomes undermined through experience." Furthermore, Ross suggests that "research is needed to determine the function of the components of legal threat in affecting the behavior of drinking and driving, particularly the relation between actual and perceived certainty, severity, and celerity of punishment (Gibbs 1975); and the interaction between certainty and severity of punishment."

Current Activities

World Health Organization

In 1979, the World Health Assembly recognized that problems related to alcohol--excessive consumption, in particular--rank among the world's major health problems. A resolution was passed urging the member States to take all appropriate measures to deal with the increasing problem and requesting the director general to strengthen the World Health Organization's (WHO) capacity to this end (WHO 1979, 1980).

At the 1982 World Health Assembly, technical discussions involving 300 delegates from 100 countries were held on the topic "Alcohol Consumption and Alcohol-Related Problems: Development of National Policies and Programs." The need for national alcohol policies to overcome future problems was accepted by every discussion group, as was the need to mobilize "political will" to overcome the inevitable difficulties in developing national alcohol policies. Furthermore, this political will must spring from the grassroots and should represent the community as well as the Nation. A major recommendation emanating from these discussions was that alcohol-related problems be given their vitally important place in all strategies for reaching the goal of "Health for All by the Year 2000."

In 1983, the World Health Assembly passed a resolution reiterating the firm conviction that problems relating to alcohol consumption rank among the world's major public health concerns and constitute a serious hazard for human welfare. This resolution recommended that member States formulate explicit and comprehensive national alcohol policies with prevention as a priority within the framework of the strategy of "Health for All by the Year 2000." The resolution also asked WHO not only to continue but to intensify its program on alcohol-related problems as an integral part of the strategy and to strengthen its capacity to respond to requests from member States to support their efforts in dealing with alcohol-related problems.

National

Health promotion.--To achieve the 1990 health objectives, many agencies within the Department of Health and Human Services (DHHS) are increasing their collaborative activities. For example, the National Cancer Institute and NIAAA are in the process of de-

termining the appropriateness of and strategies for a public education effort designed to raise awareness about risks of alcohol-related cancers.

Furthermore, specific DHHS agencies are working with the Department of Defense (DOD) to introduce a comprehensive health promotion effort that includes preventing alcohol abuse and alcoholism within the military, as well as with DOD developers of a curriculum guide for use in dependent schools overseas.

Youth.—In 1982, the Secretary of DHHS undertook an Initiative on Teenage Alcohol Abuse to address the risks associated with the consumption of alcohol by youth. This effort, which was supported by the Departments of Transportation (DOT) and Education (DOE), included dissemination of model programs, joint initiatives with the private sector, regional prevention and treatment conferences, and a national conference held in Washington, D.C., designed to draw national attention to the work of a growing number of youth-directed drinking and driving programs. The Initiative will be continued through efforts directed at youth at high risk for development of alcohol problems.

In addition, DHHS and DOE are collaborating to explore the role of schools in preventing adolescent health problems. The Adolescent School Health Promotion Demonstration Program will focus on ways to improve the integration of alcohol, drug abuse, and mental and general health promotion/prevention principles into school management policies, health services, and curriculums.

Highway safety.—Subsequent to the initiatives of grassroots movements at the local and State levels, e.g., Mothers Against Drunk Drivers (MADD), Students Against Drunk Driving (SADD), Remove Intoxicated Drivers (RID), PARK IT, and Citizens for Safe Drivers, a number of activities have taken place at the national level. The Presidential Commission on Drunk Driving was created in April 1982 to heighten public awareness of the seriousness of the drunk driving problem, persuade States and communities to strengthen their law enforcement and trial process, and encourage State and local officials to accept and use the latest techniques and methods to solve the problems and to generate support for increased law enforcement. The Commission's final report will be completed by the end of 1983. Recently, the National Safety Council

developed a plan of action addressing this problem. The Council's program, consisting of six measures, including public education, is intended to continue the national momentum against intoxicated driving.

The Alcohol Traffic Safety and National Registration Act of 1982 (Public Law 97-364) authorizes incentive grants to the States to encourage the establishment of effective alcohol safety programs. Administered by the National Highway Traffic Safety Administration (NHTSA), the law provides a basic grant to States that meet a minimum set of standards and a supplemental grant when additional provisions are incorporated. National Drugged and Drunk Driving Awareness Week, also enacted by Congress, stimulated a sense of national priority about this problem during the week of December 12-19, 1982.

Within the Federal Government, several agencies and departments have joined to increase efforts to reduce alcohol-related highway accidents. For instance, NIAAA and NHTSA and the Department of Defense have strengthened interagency collaboration to prevent such accidents. NHTSA's approach vigorously supports a six-step countermeasure model.

Policy conference.—As noted earlier, in 1981, the National Academy of Sciences published a report on its study of alternative approaches to prevention to alcohol-related problems (*Alcohol and Public Policy: Beyond the Shadow of Prohibition*). In May 1983, under contract to NIAAA, NAS convened a cross section of national, State, and local leaders to examine the study's conclusions and to distill ideas for future research and effective prevention measures. Discussions centered on the supply of alcohol, communicating about alcohol, and community cooperation to reduce alcohol problems. The participants—representing Government, business, State legislatures, public interest organizations, and the scientific community—agreed that a "network of policies" and not a single-focus strategy could be effective in reducing alcohol-related problems. The proceedings of this conference will be published in late 1983.

State and Local

The Omnibus Budget Reconciliation Act of 1981 incorporated Federal resources for alcohol-related prevention and treatment services into the Alcohol and Drug Abuse and Mental Health Services Block Grant. Of the

amount used in any fiscal year for alcohol or drug abuse activities, the States must use not less than 20 percent for alcohol and other drug abuse prevention and early intervention. A report including information on services provided under this block grant will be submitted to Congress in January 1984.

Through the combined efforts of many State Alcohol Authorities and NIAAA, a public education campaign directed at women and youth was successfully implemented. It targeted alcohol problems among women, alcohol consumption during pregnancy, and alcohol problems among youth, including drinking and driving. Television, radio, and print materials were disseminated through a multilevel strategy designed to encourage both State and local collaboration and involvement from a wide consortium of groups interested in alcohol problems. The States provided resources for dissemination of the materials, organized the campaign efforts, and developed local programs. Furthermore, many States and local communities developed additional public service announcements and print materials.

Thousands of people became involved in this effort across the country, and process evaluation indicates that the campaign was cost-efficient and successful in reaching the target audiences. More than \$10 million was donated by the private sector for airing of alcohol-related television public service announcements during the first 11 months of the 1982 campaign, at a cost of less than 1.1 cent per person. Twenty percent of the public service announcements were broadcast between the hours of 5:00 p.m. and 11:30 p.m., one of the prime viewing times for women and youth (NIAAA 1983).

A national prevention network, composed primarily of State prevention coordinators, has been formed as part of the National Association of State Alcohol and Drug Abuse Directors (NASADAD). This group represents an important prevention resource for the Nation. It is now formalizing organization and developing operating procedures and agendas (NASADAD 1983).

Many States are particularly aggressive in their pursuit of preventing drinking and driving accidents and fatalities. During the 1983 legislative session in the States, 378 separate pieces of legislation related to alcohol and/or drugs and highway safety were introduced in 37 States, and 38 laws were enacted (NASADAD 1983).

A number of States have been active in gathering data on the problem and then or-

ganizing and expanding programs for persons arrested for driving while intoxicated (DWI). Some States and local communities have found that organizing a task force on intoxicated driving can be instrumental in eliciting cooperation. For example, the Governor's Alcohol and Highway Safety Task Force in New York was co-chaired by the director of the Division of Alcoholism and Alcohol Abuse and the commissioner of the Department of Motor Vehicles. This task force established one goal: "to reduce dramatically the incidence and tragic consequences of drunk driving in New York State." To achieve this goal, a comprehensive systems approach was recommended. The task force's recommendations resulted in legislative changes, the most significant being user funding for DWI programs.

The State of South Carolina also has been especially active in the area of drunk driving through the appointment of four regional task forces that will use community-based involvement to work on reducing the growing problem of drinking and driving in the State. The State Government also has introduced a comprehensive legislative package that would raise the legal drinking age to 21, prohibit the possession of an open container of beer or wine in a moving vehicle, provide that a blood alcohol concentration of .10 percent or more is legal evidence of impairment, and require that offenders render services to their community (NASADAD 1983).

Other States—Michigan, California, Maryland, Maine, Vermont, and Massachusetts, for example—have implemented campaigns and programs within their communities. The year-end figures for 1982 showed that more than 5,000 fewer fatalities were recorded than in 1981. This also represents a 14-percent decrease since 1980 (NHTSA 1983). NHTSA recently selected 10 cities, counties, and States to undertake special efforts to remove intoxicated drivers from the highways. The 10 sites have been designated "targets of opportunities," a concept developed to promote the adoption of comprehensive, community-based, general deterrence alcohol programs. The results of this effort hold potential for future prevention of public health problems.

An important point made about State and local prevention programs is that there is a lag in prevention programming and prevention outcome. According to the CONSAD Research Report on Prevention (McCartney and Humbert 1978), prevention must involve a large enough population base, over time, to actually affect social norms. An example that proves

the point, the Cottage Meeting Program (Boswell 1983), found that after 10 years it had changed community norms and attitudes, resulting in an increased demand for prevention services.

Private Sector

In 1982, the National Council on Alcoholism (NCA), which pioneered the concept of alcoholism as a disease and a major health problem, adopted a position paper on prevention (NCA 1982; Tofany 1983). This paper expresses NCA support for a wide variety of approaches, including education, information programs, regulatory measures including advertising curbs, an increase in Federal excise taxes on alcoholic beverages, a nationwide legal drinking age of 21, and a series of rotating health warning notices regarding the consequences of alcohol consumption.

Through the 1982 NIAAA public education campaign and outreach efforts conducted by the National Clearinghouse for Alcohol Information (NCALI), major voluntary associations embarked on alcohol abuse prevention for the first time. For example, the YWCA, with assistance from NCALI, developed a curriculum and provided technical assistance for the potential implementation of a prevention program in 900 local chapters. Many voluntary organizations included comprehensive prevention programs and activities on their agendas at the community level, thus increasing visibility for alcohol-related problems and prevention programming at the local level. Many businesses also joined this effort at the State and local levels.

In addition, in 1982, parent groups began to seek ways to prevent illegal and harmful alcohol use and misuse by youth. The previous focus of these groups was on the prevention of drug use; more recently, this attention has focused on alcohol and alcohol in combination with other drugs.

In the area of drinking and driving, other adult and youth community groups proliferated to prevent alcohol-related motor vehicle accidents. These various groups work to create a climate in which the community as a whole can be actively concerned about certain alcohol-related problems.

Probably one of the largest private sector initiatives to prevent alcohol and other drug problems among youth has been "The Chemical People," a two-part television broadcast aired in November 1983 in conjunction with the participation of more than 25 national organ-

izations and 200 local public broadcast stations (PBS) across the country. Each participating PBS station and community volunteers organized town meetings to watch the programs and discuss community solutions unique to the problems of particular communities. The project was produced and pilot-tested by WQED in Pittsburgh with funding by the Richard King Mellon Foundation and the Metropolitan Life Foundation (WQED 1983).

The private sector is becoming increasingly involved in national, State, and local prevention activities. Insurance companies, the motor vehicle industry, soft drink producers and distributors, tire manufacturers, sports teams, privately owned print and broadcast outlets, producers and distributors of alcoholic beverages, and a wide range of other private sector businesses have contributed to prevention efforts.

The message communicated by these efforts indicates that there is and must continue to be a shared responsibility for prevention among different groups at all levels. This environment of concern has the potential for generating additional enthusiasm, commitment, and resolve to address alcohol problems.

Summary

The intent of this chapter has been to describe current perspectives on prevention, to summarize available research, and to describe current prevention efforts and initiatives. While national, State, and local prevention activities—often stimulated by grassroots movements—may be implemented without a scientifically based and sound methodological design, description of these efforts does suggest ideas for future prevention research.

Clearly, advances have been made in prevention since the Fourth Special Report (USDHHS 1981) was published. Previous research efforts focused on measuring the impact of single variables to determine changes in behavior and often failed to demonstrate significant differences. A broader range of prevention approaches is now being tested, however, and evaluations are being designed to test models in which myriad factors and variables interact in contributing to alcohol-related problems.

Although definitive answers may not be forthcoming in the immediate future, research findings suggest that both educational approaches and laws and regulations contribute to a reduction of alcohol problems. For these

to be effective, however, public support must be generated and sustained.

Given the complexity and interaction of factors affecting drinking patterns, occurrences, and consequences, no singular prevention strategy is likely to be optimal or universally generalizable. What is becoming eminently clear from previous and recent research is that a combination of diverse strategies must be employed. In this regard, a report entitled Beyond the Shadow of Prohibition published by the National Academy of Sciences in 1981 states that researchers:

are convinced that the regulation of supply, legal and educational approaches to

drinking practices, and interventions in the environment mediating between drinking and certain of its consequences represent valid approaches with promise for sustained improvement. Each detailed element will fail or succeed only as it is implemented properly and thoroughly; tactics that are undertaken as part of a broad and coordinated approach are more likely to be effective than ones undertaken in isolation. (Moore and Gerstein 1981, p. 116)

It is hoped that these combined approaches will be effective in reaching the alcohol-related health objectives of the Nation.

References

- Alcocer, A.M. Alcohol use and abuse among the Hispanic American population. In: National Institute on Alcohol Abuse and Alcoholism. Special Population Issues. Alcohol and Health Monograph No. 4. DHHS Pub. No. (ADM)82-1193. Washington, D.C.: Supt. of Docs., U.S. Govt. Print Off., 1982. pp. 361-384.
- Alcohol, Drug Abuse, and Mental Health Administration. ADAMHA Prevention Policy and Programs 1979-1982. DHHS Pub. No. (ADM)81-1038. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981. 67 pp.
- Armey, G.; Elmer, A.; and Herz, U. Alcohol in the World of the 80's: Habits, Attitudes, Preventive Policies and Voluntary Efforts. Stockholm: Sober Förlags AB, 1982. 223 pp.
- Bandura, A. Principles in Behavior Modification. New York: Holt, Rinehart & Winston, 1969.
- Bandura, A. Social Learning Theory. Englewood Cliffs, N.J.: Prentice Hall, 1977.
- Bandura, A. The self system in reciprocal determinism. American Psychologist 33(4):344-358, 1978.
- Bandura, A. Self-Efficacy: An Integrative Construct. Stanford, Calif.: Stanford University, 1979.
- Bartlett, E.E. Contribution of school health education to community health promotion: What can we reasonably expect? American Journal of Public Health 71(12):1384-1391, 1981.
- Blane, H.T. Education and the prevention of alcoholism. In: Kissin, B., and Begleiter, H., eds. Social Aspects of Alcoholism. The Biology of Alcoholism, Vol. 4. New York: Plenum Press, 1976. pp. 519-578.
- Blane, H.T., and Hewitt, L.E. Alcohol and Youth: An Analysis of the Literature 1960-75. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, March 1977. NTIS No. PB268698/AS.
- Borkenstein, R.F. Problems of enforcement. In: Goldberg, L. ed. Alcohol, Drugs and Traffic Safety. Stockholm: Almqvist and Wiskell International, 1981. pp. 818-837.
- Boswell, B.N. The Cottage Program International, Inc. A Substance Abuse Prevention Program for the State of Utah. Final Report Analysis for Fiscal Year Ended June 30, 1983. Salt Lake City: The Cottage Program International, July 1983. 15 pp.
- Botvin, G.J., and Eng, A. The efficacy of a multi-component approach to the prevention of cigarette smoking. Preventive Medicine 11(2):199-211, 1982.
- Braucht, G.N. Problem drinking among adolescents: A review and analysis of psychosocial research. In: National Institute on Alcohol Abuse and Alcoholism. Special Population Issues. Alcohol and Health Monograph No. 4. DHHS Pub. No. (ADM) 82-1193. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 143-164.
- Brody, J.A. Aging and alcohol abuse. Journal of the American Geriatrics Society 30(2):123-126, 1982.
- Bryan, W.A. Administering a campus alcohol program. In: Dean, J.C., and Bryan, W.A., eds. Alcohol Programming for Higher Education. Carbondale, Ill.: ACPA Media, 1982. pp. 44-57.
- Colon, L. "Alcohol Control, Policies and Their Relation to Alcohol Consumption and Alcoholism." Ph.D. dissertation, Brandeis University, The F. Hellen Graduate School, 1980.
- Cook, P. Effect of liquor taxes on drinking, cirrhosis, and auto accidents. In: Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. pp. 255-285.
- Cook, P., and Tauchen, G. The effect of liquor taxes on heavy drinking. Bell Journal of Economics, 13(2):379-390, 1982.
- Crisp, A.D. Making substance abuse prevention relevant to low-income black neighborhoods. II. Research findings. Journal of Psychedelic Drugs 12:139-156, 1980.
- Dean, J.C. Alcohol programming: A conceptual model. In: Dean, J.C., and Bryan, W.A., eds. Alcohol Programming for Higher Education. Carbondale, Ill.: ACPA Media, 1982. pp. 15-29.

- Donovan, B. "Collegiate Group for the Sons and Daughters of Alcoholics." Paper presented at NADC Conference, Washington, 1980. 17 pp.
- Douglass, R.L.; Filkins, L.D.; and Clark, F.A. The Effect of Lower Legal Drinking Ages on Youth Crash Involvement. Final Report. Report No. UM-HSRI-AI-74-1-2. Washington, D.C.: National Highway Traffic Safety Administration, June 1974. NTIS No. PB 235-917.
- Evans, G.B.; Steer, R.A.; and Fine, E.W. Alcohol value clarification in sixth graders: A film-making project. Journal of Alcohol and Drug Education 24(2):1-10, 1979.
- Food and Drug Administration. Surgeon General's Advisory on Alcohol and Pregnancy. FDA Drug Bulletin 11(2):9-10, 1981.
- Fullerton, M. A program in alcohol education designed for rural youth. Journal of Alcohol and Drug Education 24(2):58-62, 1979.
- Gaines, J.J. Alcohol and the black woman. In: Harper, F.D., ed. Alcohol Abuse and Black America. Alexandria, Va.: Douglass Publishers, 1976. pp. 153-162.
- Garza, R. El alcoholico [the alcoholic]. Impact 9(3):4-5, 1979.
- Gibbs, J.P. Crime, Punishment, and Deterrence. New York: Elsevier, 1975. 272 pp.
- Gomberg, E.S. Alcohol use and alcohol problems among the elderly. In: National Institute on Alcohol Abuse and Alcoholism. Special Population Issues. Alcohol and Health Monograph No. 4. DHHS Pub. No. (ADM)82-1193. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 263-290.
- Goodstadt, M.S. Alcohol and drug education: Models and outcomes. Health Education Monographs 6(3):263-279, 1978.
- Goodstadt, M.S., and Sheppard, M.A. Three approaches to alcohol education. Journal of Studies on Alcohol 44(2):362-380, 1983.
- Goodstadt, M.S.; Sheppard, M.A.; and Chan, G.C. An evaluation of two school-based alcohol education programs. Journal of Studies on Alcohol 43(3) 352-369, 1982.
- Hames, L.N., and Petrucelli, E. Plan for influencing teenage drinking drivers. American Association for Automotive Medicine, October 1980. pp. 17-21.
- Henggeler, S.W.; Sallis, J.F.; and Cooper, P.F. Comparison of university mental health needs priorities identified by professionals and students. Journal of Counseling Psychology 27(2): 217-219, 1980.
- Hewitt, L.E. Current status of alcohol education programs for youth. In: National Institute on Alcohol Abuse and Alcoholism. Special Population Issues. Alcohol and Health Monograph No. 4. DHHS Pub. No. (ADM)82-1193. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 227-260.
- Hingson, R.W.; Scotch, N.; Mangione, T.; Meyers, A.; Glantz, L.; Heeren, T.; Lin, N.; Mucatel, M.; and Pierce, G. Impact of legislation raising the legal drinking age in Massachusetts from 18 to 20. American Journal of Public Health 73(2): 163-170, 1983.
- Hochheimer, J.L. Reducing alcohol abuse: A critical review of educational strategies. In: Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. pp. 286-335.
- Holder, H.D., and Blose, J.O. Reduction of Community Alcohol Problems: A Computer Simulation for Wake County, North Carolina, Washington County, Vermont, and Alameda County, California. Chapel Hill, N.C.: The Human Ecology Institute, 1983. 229 pp.
- Hundleby, J.D.; Carpenter, R.A.; Ross, R.A.J.; and Mercer, G.W. Adolescent drug use and other behaviors. Journal of Child Psychology and Psychiatry 23:(1)61-68, 1982.
- Institute of Medicine. Legislative Approaches to Prevention of Alcohol-Related Problems: An Inter-American Workshop. Pub. No. IOM-82-003. Washington, D.C.: National Academy Press, 1982. 285 pp.
- Irwin, K.C. "Women, Alcohol, and Drugs: A Feminist Course Focusing on Causes and Prevention of Abuse." Paper presented at the 104th Annual Meeting of the American Public Health Association, Miami, 1976.
- Jessor, R., and Jessor, S. Problem Behavior and Psychosocial Development. New York: Academic Press, 1977. 281 pp.
- Johnson, C.A.; Graham, J.W.; and Hansen, W.B. "Drug Use by Peer Leaders." Paper presented at the American Psychological Association annual meeting, Los Angeles, 1981.
- Kola, L.A., and Kosberg, J.I. Model to assess community services for the elderly alcoholic. Public Health Reports 96(5):458-463, 1981.
- Koski, H. Arbetsmarknaden och alkoholpriserna [Labor market and alcohol prices]. Alkoholpolitik 40:37-38, 1977.
- Kraft, D.P. "A Program to Prevent Alcohol Problems Among College Students: Successes and Failures." Paper presented at the American Psychological Association annual meeting, Washington, D.C., 1982.
- Leavy, R.L. First steps in campus prevention programs. CAFC News (Arlington, Va.), July-August 1980.
- Leigh, D.H. "Prevention Work Among the Elderly: A Workable Model." Paper presented at the National Alcoholism Forum, Seattle, 1980.
- Lillis, R.P.; Williams, T.P.; Chupka, J.Q.; and Williford, W.R. Highway Safety Considerations in Raising the Minimum Legal Age for Purchase of Alcoholic Beverages to Nineteen in New York State. Albany: New York State Division of Alcoholism and Alcohol Abuse, 1982. 15 pp.
- Little, R.E. Change in obstetrician advice following a 2-year community education program on alcohol. Journal of Obstetrics and Gynecology, in press.
- Little, R.E.; Streissguth, A.P.; and Guzinski, G.M. Prevention of fetal alcohol syndrome: A model program. Alcoholism: Clinical and Experimental Research 4(2):185-189, 1980.
- Lucy, P. Can alcohol taxes reduce consumption?

- Australian Journal of Alcoholism and Drug Dependence 6(4):119-122, 1979.
- Maccoby, N., and Farquhar, J.W. Communication for health: Unselling heart disease. Journal of Communications 25:114-126, 1975.
- Makela, K. Consumption level and cultural drinking patterns as determinants of alcohol problems. Journal of Drug Issues 5(4):344-357, 1975.
- Makela, K. Differential effects of restricting the supply of alcohol: Studies of a strike in Finnish liquor stores. Journal of Drug Issues 10(1):131-144, 1980.
- Makela, K.; Room, R.; Single, E.; Sulkunen, P.; and Wash, B. Alcohol, Society, and the State: A Comparative Study of Alcohol Control, Vol. 1. Toronto: Addiction Research Foundation, 1981. 146 pp.
- Malec, J. Metody zmniejszenia spożycia alkoholu [Methods of reducing alcohol consumption]. Problemy Alkoholizmu 27(4):3-4, 13, 1980.
- Malin, H.; Coakley, J.; Kaelber, C.; Munch, N.; and Holland, W. An epidemiologic perspective on alcohol abuse in the United States. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM)82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 99-153.
- Maxwell, D. Impact Analysis of the Raised Legal Drinking Age in Illinois. Washington, D.C.: National Highway Traffic Safety Administration, December 1981. 23 pp.
- McAlister, A.L.; Perry, C.; and Maccoby, N. Adolescent smoking: Onset and prevention. Pediatrics 63(4):650-658, 1979.
- McCartney, D.M., and Humbert, P. Evaluating the Prevention Demonstration: A Handbook for Grant Applicants. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, March 1978.
- McCarty, D.; Poore, M.; Mills, K.C.; and Morrison, S. Direct mail techniques and the prevention of alcohol-related problems among college students. Journal of Studies on Alcohol 44(1):162-170, 1983.
- McGuire, W.J. The nature of attitudes and attitude change. In: Lindzey, G., and Aronson, E., eds. Handbook of Social Psychology. Reading, Mass.: Addison-Wesley, 1969.
- McIntyre, C.E. "Evaluating Prevention and Education: Fetal Alcohol Syndrome." Paper presented at the National Council on Alcoholism annual meeting, Seattle, 1980.
- Mello, N.K. Behavioral studies of alcoholism. In: Kissin, B., and Begleiter, H., eds. Physiology and Behavior. The Biology of Alcoholism, Vol. 2. New York: Plenum Press, 1972. pp. 219-291.
- Mills, K.C.; McCarthy, D.; Ward, J.; Minuto, L.; and Patzynski, J. A residence hall tavern as a collegiate alcohol abuse prevention activity. Addictive Behaviors 8(2):105-108, 1983.
- Miranda, V.L. "Black Alcohol Prevention Programming: Past, Present, Future." Paper presented at NIAAA skills development workshop, Agenda for Black Alcoholism Programs. Jackson, Miss., 1981.
- Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. 463 pp.
- Mosher, J.F., and Mottl, J.R. The role of nonalcohol agencies in Federal regulation of drinking behavior and consequences. In: Moore, M.H., and Gerstein, D.R., eds. Alcohol and Public Policy: Beyond the Shadow of Prohibition. Washington, D.C.: National Academy Press, 1981. pp. 388-458.
- Nathan, P.E., and Lisman, S.A. Behavioral and motivational patterns of chronic alcoholics. In: Tarter, R.E., and Sugarman, A.A., eds. Alcoholism: Interdisciplinary Approaches to an Enduring Problem. Reading, Mass.: Addison-Wesley, 1976. pp. 479-522.
- National Association of State Alcohol and Drug Abuse Directors. Alcohol and drug abuse among the elderly: Out of sight, out of mind? NASADAD Alcohol and Drug Abuse Report, August 1981. 8 pp.
- National Association of State Alcohol and Drug Abuse Directors. Alcohol and Drug Abuse Report. Special Report. Washington, D.C.: NASADAD, May 1983.
- National Council on Alcoholism. "Prevention Position Paper." Unpublished NCA working document, June 11, 1982.
- National Highway Traffic Safety Administration. Fatal Accident Reporting System, 1981: An Overview of U.S. Traffic Fatal Accident and Fatality Data Collected in FARS for Year 1981. Pub. No. DOT HS-806-251. Washington, D.C.: NHTSA, January 1983.
- National Institute on Alcohol Abuse and Alcoholism. CASPAR Alcohol Education Program, Demonstration Curriculum Project. Final Report, 1979. Rockville, Md.: NIAAA, 1979.
- National Institute on Alcohol Abuse and Alcoholism. Here's Looking at You Alcohol Education Project. Final Report, 1981. Rockville, Md.: NIAAA, 1981a.
- National Institute on Alcohol Abuse and Alcoholism. Prevention of Alcohol Problems in Pre-Delinquent Youth. Final Report, 1981. Rockville, Md.: NIAAA, 1981b.
- National Institute on Alcohol Abuse and Alcoholism. Report to the United States Congress on Federal Activities on Alcohol Abuse and Alcoholism, FY 1980. Rockville, Md.: NIAAA, 1982. 154 pp. and appendixes.
- National Institute on Alcohol Abuse and Alcoholism. Implementing Public Health Education Campaigns: Lessons from Alcohol Abuse Prevention, An Evaluation of the National Institute on Alcohol Abuse and Alcoholism 1982 Alcohol Abuse Prevention Campaign. Rockville, Md.: NIAAA, January 1983. 37 pp. and appendixes.
- Olegard, R.; Sabel, K.-G.; Aronsson, M.; Sandin, B.; Johansson, P.R.; Carlsson, C.; Kyllerman, M.; Iversen, K.; and Hrbek, A. Effects on the child of alcohol abuse during pregnancy: Retrospective and prospective studies. Acta Paediatrica Scandinavica (Supplement No. 275):112-121, 1979.

- Perry, C.L., and Murray, D.M. "Peer Teaching in Smoking Prevention Research: Theoretical Considerations in Intervention Strategies." Paper presented at the American Psychological Association annual meeting, Los Angeles, 1981.
- Portnoy, B. Effects of a controlled-usage alcohol education program based on the Health Belief Model. Journal of Drug Education 10(3):181-195, 1980.
- Pringle, H.; Gregory, J.; Ginkel, K.; and Cheek, C. Alpha Centers: A viable prevention model for substance abuse agencies and public schools. In: Schecter, A.J., ed. Social and Behavioral Issues. Drug Dependence and Alcoholism, Vol. 2. New York: Plenum Press, 1981. pp. 183-189.
- Resource Alternatives Corporation. Prevention of Alcohol Abuse Among Pre-Delinquent Youth: An Overview of the Evaluation Results. Washington, D.C.: RAC, 1982.
- Roizen, J. Estimating alcohol involvement in serious events. In: National Institute on Alcohol Abuse and Alcoholism. Alcohol Consumption and Related Problems. Alcohol and Health Monograph No. 1. DHHS Pub. No. (ADM)82-1190. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 179-219.
- Rosett, H.L.; Weiner, L.; Zuckerman, B.; McKinlay, S.; and Edelin, K.C. Reduction of alcohol consumption during pregnancy with benefits to the newborn. Alcoholism: Clinical and Experimental Research 4(2):178-184, 1980.
- Rosett, H.L.; Weiner, L.; Lee, A.; Zuckerman, B.; Dooling, E.; and Oppenheimer, E. Patterns of alcohol consumption and fetal development. Obstetrics and Gynecology 61(5):539-546, 1983.
- Ross, H.L. Deterrence of the Drinking Driver: An International Survey. Washington, D.C.: National Highway Traffic Safety Administration, 1981. 121 pp.
- Rotter, J.B. Generalized expectancies in internal versus external control of reinforcement. In: Rotter, J.B.; Chance, J.E.; and Phares, E.J., eds. Application of Social Learning Theory of Personalities. New York: Holt, Rinehart & Winston, 1972.
- Sandmaier, M. "Women and Alcohol Abuse: A Strategy for Defense." Paper presented at the 104th Annual Meeting of the American Public Health Association, Miami, 1976.
- Schaefer, J.M. Ethnic and racial variations in alcohol use and abuse. In: National Institute on Alcohol Abuse and Alcoholism. Special Population Issues. Alcohol and Health Monograph No. 4. DHHS Pub. No. (ADM)82-1193. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp. 293-311.
- Smart, R.G. The impact of prevention measures: An examination of research findings. In: Institute of Medicine. Legislative Approaches to Prevention of Alcohol-Related Problems: An Inter-American Workshop. Pub. No. IOM-82-003. Washington, D.C.: National Academy Press, 1982. pp. 224-246.
- Smith, R. Relation between consumption and damage. British Medical Journal 283(6295):895-898, 1981.
- Somervuori, A. Prisskruven som alkoholpolitiskt medel [Pricing as an alcohol policy]. Alkoholpolitik 40:85-93, 1977.
- Staulcup, H.; Kenward, K.; and Frigo, D. A review of Federal primary alcoholism prevention projects. Journal of Studies on Alcohol 40(11): 943-968, 1979.
- Streissguth, A.P.; Darby, B.L.; Barr, H.M.; Smith, J.R.; and Martin, D.C. Comparison of drinking and smoking patterns during pregnancy over a six-year period. American Journal of Obstetrics and Gynecology 145(6):716-724, 1983.
- Stuart, R.B. Teaching facts about drugs: Pushing or preventing? In: Ward, D.A., ed. Alcoholism: Introduction to Theory and Treatment. Dubuque, Iowa: Kendall/Hunt, 1980. pp. 211-225.
- Sulkunen, P. Developments in the Availability of Alcoholic Beverages in the EEC Countries. Helsinki: Social Research Institute of Alcohol Studies, 1978. 93 pp.
- Tofany, V.L. The council's plan of action. Traffic Safety, March/April 1983.
- U.S. Department of Health and Human Services. Fourth Special Report to the U.S. Congress on Alcohol and Health. De Luca, J. R., ed. DHHS Pub. No. (ADM)81-1080. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1981.
- U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Prevention '82. DHHS (PHS) Pub. No. 82-50157. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. 168 pp.
- WQED Television. What Does It Take To Fight School-Age Drug and Alcohol Abuse? Pamphlet in The Chemical People series. Pittsburgh, Pa.: WQED, 1983.
- Wagenaar, A.C. "The Minimum Legal Drinking Age: A Times-Series Impact Evaluation." Ph.D. dissertation, University of Michigan, 1980.
- Wagenaar, A.C.; Douglass, R.L.; and Compton, C.P. Raising the Legal Drinking Age in Michigan and Maine. Report No. UM-HSRI-81-58. Rockville, Md.: National Institute on Alcohol Abuse and Alcoholism, 1981. 367 pp.
- Wallack, L.M. Mass media campaigns: The odds against finding behavior change. Health Education Quarterly 8(3):209-260, 1981.
- Weisheit, R. A.; Kearney, K.A.; Hopkins, R.H.; and Mauss, A.L. Evaluation of a Model Alcohol Education Project for the Public Schools: Phase II. An Overview of the First Year's Activities and Results. Pullman: Washington State University, 1979.
- Williams, A.F.; Zador, P.L.; Harris, S.S.; and Karpf, R.S. The effect of raising the legal minimum drinking age on fatal crash involvement. Journal of Legal Studies 12:169-179, 1983.
- Wittman, F.D. Current status of research demonstration programs in the primary prevention of alcohol problems. In: National Institute on Al-

cohol Abuse and Alcoholism. Prevention, Intervention, and Treatment: Concerns and Models. Alcohol and Health Monograph No. 3. DHHS Pub. No. (ADM)82-1192. Washington, D.C.: Supt. of Docs., U.S. Govt. Print. Off., 1982. pp.3-57.
World Health Organization. Alcohol control poli-

cies. In: Robinson, D., ed. Alcohol Problems. New York: Holmes and Meier, 1979. pp. 227-231.
World Health Organization. Problems Related to Alcohol Consumption. Report of a WHO Expert Committee. Technical Report Series 650. Geneva, Switzerland: WHO, 1980. 72 pp.

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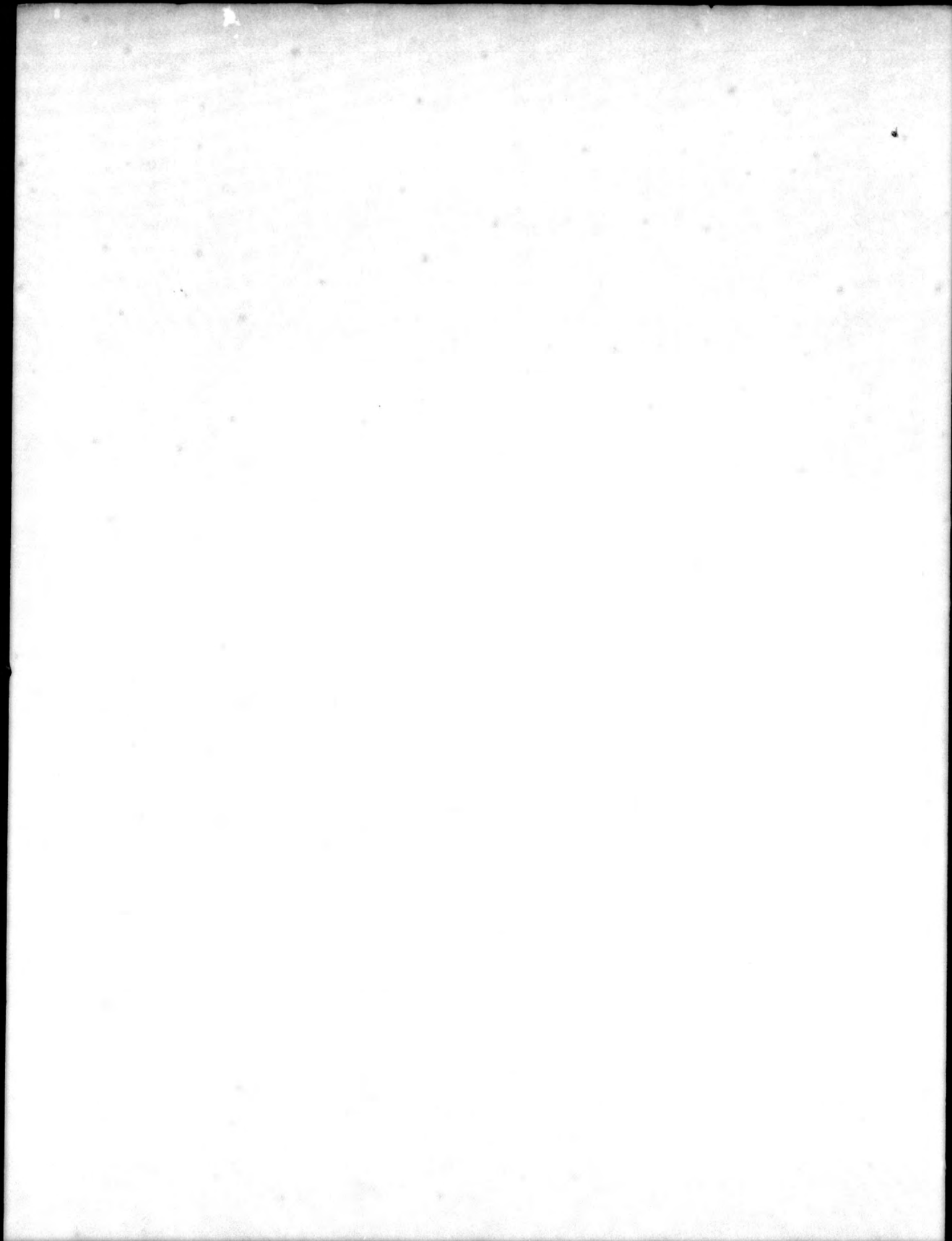
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Alcohol Consumption Patterns among American Indian and White College Students

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ABSTRACT. College students in Oklahoma completed a self-administered questionnaire to compare the drinking behaviors of culturally active American Indians ($N = 34$ men and 24 women) and Whites ($N = 181$ men and 250 women). Significantly more Indians were classified as drinkers, but they had begun drinking at a somewhat later age. Both groups indicated a preference for beer, and they were quite similar in quantity and frequency of beer consumption. White students reported drinking significantly more wine and distilled spirits, and drinking more often in public places, such as bars, pubs, restaurants and parked cars; Indians drank more in their own homes and in the homes of friends. White students tended to cite hedonistic reasons for drinking

whereas Indians reported escapist or social reasons and drinking to "get high." Drinking-related problems were reported somewhat more often by Indian students, notably so by Indian women. Indians were more inclined to report the more serious drinking problems of being arrested, blacking out, interference with school or work, and difficulties in human relationships. White students more often cited problems of nausea or vomiting, drinking and driving, doing something that was later regretted and damaging property. It was suggested that the higher Indian arrest rate could be indicative of police bias and that the reports of problem drinking among Indian women be investigated further. (*J. Stud. Alcohol* 45: 433-439, 1984)

NORTH AMERICAN INDIANS have received a great deal of attention in relation to how they perceive and use alcoholic beverages. From the earliest stages of the White man's settlement, historians have described Indians as having a craving for alcohol, being ill-equipped to handle it, and being inclined toward violent, destructive and antisocial behavior when drinking (MacAndrew and Edgerton, 1969). The literature seems to indicate that alcohol-related problems among Indians are becoming even more widespread. In 1960, for example, federal crime statistics showed that a greater proportion of Indians than of other ethnic groups were arrested for all alcohol-related offenses (Steward, 1964). In 1972, 21.3% of White arrests, but 61.8% of Indian arrests, were for drunkenness (Cockerham, 1977).

It has been suggested that statistics of this nature could be influenced by differential arrest rates. Forslund and Meyers (1974), for example, found the delinquency rate among reservation youth to be nearly five times the national average, but a majority of court appearances were for "relatively minor offenses," and a large proportion were alcohol-related. On the other hand, Indian health statistics paint a somewhat different picture. The death rate from cirrhosis of the liver increased from 14.2 deaths per 100,000 population in

1955 to 42.5 deaths per 100,000 in 1975 and was the fourth leading cause of death among Indians in that year (U.S. Health Services Administration, 1978). In 1975, accidents were the leading cause of Indian deaths (156.4 per 100,000 population); the majority of these were motor-vehicle-related, many involving alcohol.

Several theoretical explanations for Indian drinking behavior have been proposed, some of them substantiated by research. Early interpretations suggested that physiological differences made this group less tolerant of alcohol; in recent years, research along this line has been revived. Some investigators (Farris and Jones, 1978; Fenna et al., 1971) reported that Indians metabolized alcohol more slowly than Whites. Bennion and Li (1976), however, found no differences between the metabolic rates of the two groups. Wolff (1972) examined facial flushing as a result of drinking and concluded that some Indians exhibited vasomotor characteristics not found among Whites.

The psychological aspects of Indian drinking have also been investigated. Using the Minnesota Multiphasic Personality Inventory (MMPI), Kline et al. (1973) compared Indian and White alcoholics and found the Indians to have higher personality disturbance scores. Uecker et al. (1980), however, reported no differences in MMPI scores between Indians and Whites. Social variables such as stress, anomie or

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anxiety have also been implicated. For example, Maynard (1969) identified stress as a factor in Indian drinking, and Littman (1970) suggested that Indian alcoholism could be the result of anomie, or of conflicts arising from rural-urban transition. Lemert (1956) concluded that Indian drinking could be an expression of anxiety.

A third group of researchers has studied the cultural aspects of Indian drinking behavior. Some authors (DuToit, 1964; Lemert, 1956) have indicated that Indian drinking may have positive functions in that it helps integrate individuals into intimate groups or replaces lost social institutions. Lurie (1971) postulated Indian drinking to be a means of protesting against the White culture and of validating Indianness. Price (1975), however, related problem drinking among Indians to a breakdown of traditional cultural controls. Stratton et al. (1978) reported eastern Oklahoma tribes to have lower rates of both alcohol-related arrests and deaths than did those from western Oklahoma. They attributed this to differences in tribal culture and to the fact that eastern Indians had become farmers and merchants at an earlier stage in their history than western tribes, which more recently had experienced the culture shock of entering the White world. These authors maintained that drinking among western tribes could be a "retreatist or escapist response to acculturational stress." (p. 1171).

A great deal of variation in drinking incidence has been cited among American collegians. The initial study of Straus and Bacon (1953) found a range of from 20% among some women to 98% among some men. In a survey of 37 colleges across the U.S., Hanson (1977) reported an average drinking incidence of 83.5%. Wechsler and McFadden (1979) indicated that abstainers comprised less than 5% of their sample from 34 New England schools. Since methodologies often differed, comparisons must be viewed with caution, but it can be noted that some variables, such as region of the country or population density, appear to have an impact on drinking incidence.

Although many of the seminal studies on alcohol use (e.g., Straus and Bacon, 1953) have been with college populations, little research has been conducted on Indian students in this context. No known studies have examined only those Indian collegians who identify culturally with their native heritage and tend to practice its traditions. Strimbu et al. (1973) analyzed drug use among over 20,000 southeastern college students, 74 of whom were Indians, and found Indian students to report a high incidence of both drug and alcohol use. The degree of cultural identity among this group, however, was not known.

The literature concerning Indian drinking behavior has been inconsistent and does not lend itself to

predictive hypotheses. Explanations of Indian drinking have been varied, and have dealt with such topics as Indian youth, Indians on reservations, or those in alcoholism treatment programs. Few studies have examined Indian collegians, and the drinking patterns of Indian students who are involved in their native culture have been neglected. The purpose of this research, therefore, is to compare drinking behavior of culturally active Indian collegians with the activities of White students in the same environment.

Method

Data were collected from the student body of Oklahoma State University during the spring semester of 1981. The on-campus enrollment at the university was 20,739 (58.2% men and 41.8% women), and Indian enrollment was 329 (59.1% men and 40.9% women). The state of Oklahoma has one of the largest Indian populations in the U.S. (Bahr et al., 1979), but the proportion who identify with their native culture is not known. Indian students were identified for the present survey by an Indian who was able to separate the "cultural" Indians (those who considered themselves to be Indian, attended Indian tribal or cultural functions, and associated with other Indians) from those who identified primarily with the majority society. Sixty students were so designated and completed self-administered questionnaires. It was estimated that this group comprised nearly the entire known population of "cultural" Indian students at the university.

The tribes represented were largely from central Oklahoma (Pawnee, Ponca, Kawa, Tonkawa and Otoe-Missouria). All respondents were at least one-quarter Indian, and the majority had one-half or more Indian heritage. These tribes originated in the central part of the U.S. and in cultural orientation were considered Prairie Indians. They lived in areas in which a sedentary living pattern was possible, but would seasonally assume the lifestyle of the Plains Indians and follow the buffalo. In acculturation to the White society, they more closely resemble the Plains Indians from the western part of Oklahoma than tribes from the eastern part of the state (Howard, 1978).

The sample of White students ($N = 500$) was obtained from self-administered questionnaires completed in a random selection of 14 introductory sociology classes. The original sample included 46 non-White and foreign students but they were eliminated from the data analysis. Seven of the 46 were Indian students, and they were also excluded since it could not be determined whether they identified with the White or Indian culture.

The White cohort tended to be fairly young, with a mean age of 19.2, and was largely freshman. There were slightly more women (59.6%) than men. Most (96.8%) were single, almost half (46.9%) came from communities with populations of over 50,000, and 72.2% reported higher parental occupational prestige. The mean age for Indians was 23.4, and they indicated a more bell-shaped distribution over year in school. More than half (56.7%) of the Indians were men, 30% were married, 61.7% came from communities with populations of

under 50,000, and 78.3% reported lower parental occupational prestige.

Approximately three-fourths of both samples declared either business or arts and sciences as a major. Somewhat more Indian students than Whites were in agriculture and education and fewer were in engineering and home economics. In grade-point average, Indians were more inclined to be average students, a fewer number reporting both higher and lower grades. Descriptions of the sample apply to both drinkers and abstainers, but data analysis was conducted on drinkers only—431 White and 58 Indian students.

Since these Indians probably constituted most of the population of "cultural" Indians, it was concluded that some of the demographic differences, particularly those of age, were due not so much to sampling procedures, but to the fact that this group was essentially different from the more typical college student population. Indians tend to be rural in background and as a group are economically deprived (Brinker and Klos, 1976). Indian collegians enter college at a later age for a variety of reasons, perhaps for financial or family considerations.

Results

Quantity and frequency of drinking

Quantity and frequency information was elicited from a slightly modified form of the questionnaire used by Jessor et al. (1968). Students were asked six questions regarding their alcohol consumption—how often, on the average, they usually drank beer, wine and distilled spirits, and how many drinks of beer, wine and distilled spirits they usually had at any one time. Answers for quantity ranged from "none" to "over 6 drinks at any one time" and for frequency, from "never" to "daily." These were dichotomized at the 50% mark to obtain a chi-square statistic. With abstainers identified as those who answered "none" or "never" to all six questions, 86.2% of White and 96.7% of Indian students were classified as drinkers ($\chi^2 = 5.30$, 1 df, $p = .02$). For men, 89.6% of Whites and all Indians were classified as drinkers ($\chi^2 = 3.88$, 1 df, $p = .05$), and for women these figures were 83.9% of

Whites and 92.3% of Indians. The mean age for beginning to drink was 16.0 for Whites and 17.5 for Indians.

White and Indian students, as a whole, were not strikingly different in quantity and frequency of alcohol consumption, although in most cases, Whites reported drinking more alcohol and doing so more often than Indians (Table 1). The only exception to this trend was in both quantity and frequency of beer consumption, in which the percentages of Indians in the high categories exceeded those of Whites. Whites, however, had a significantly higher quantity and frequency of distilled spirits consumption.

When Whites and Indians together were examined by sex, several differences were noted, four of these significant: Although women drank wine more often than did men, men exceeded women by a significant margin in quantity and frequency of beer consumption and quantity of distilled spirits consumption. This seems to indicate that the Whites and Indians in this sample were similar to samples from other studies, both collegiate and nationwide, since almost all researchers have reported a higher rate of drinking among men (Cahalan and Cisin, 1968; Engs, 1977; Gallup, 1980; Straus and Bacon, 1953).

Comparisons between White and Indian men as well as between White and Indian women showed similar trends. In all but beer drinking, White students of both sexes indicated a higher quantity and frequency of consumption than Indians; the differences for distilled spirits being significant for quantity in both sexes and for frequency in men. A comparison of the percentages of students in the high categories indicated that beer was the most popular drink for both Whites and Indians and for both men and women; relatively few reported drinking wine.

Drinking locations

Frequencies of drinking in each of eight locations are shown in Table 2. Since these variables were dichoto-

TABLE 1. Drinking quantity and frequency by ethnicity and sex, in percent, with percentages given only for the high categories of each variable

	Ethnicity				Sex			Men only			Women only		
	Total (N = 439)	Whites (431)	Indians (58)	χ^2	Men (215)	Women (274)	χ^2	Whites (181)	Indians (34)	χ^2	Whites (250)	Indians (24)	χ^2
Quantity													
Beer	66.9	65.9	74.1	1.57	79.5	56.9	21.78*	79.6	79.4	0.00	56.0	66.7	1.02
Wine	25.6	26.9	15.5	3.49	27.0	24.5	0.40	29.3	14.7	3.09	25.2	16.7	0.86
Distilled spirits	48.4	51.8	22.8	16.88*	55.1	43.0	7.05*	61.1	23.5	16.33*	45.0	21.7	4.64*
Frequency													
Beer	55.2	53.8	65.5	2.83	69.3	44.2	30.80*	68.5	73.5	0.34	43.2	54.2	1.07
Wine	18.8	20.0	10.3	3.09	14.4	22.3	4.85*	16.0	5.9	2.39	22.8	16.7	0.48
Distilled spirits	32.6	35.1	13.8	10.58*	32.1	33.0	0.04	35.9	11.8	7.66*	34.5	16.7	3.16

* $P < .05$.

TABLE 2. Frequency of drinking locations by ethnicity and sex, in percent, with percentages given only for the high categories of each variable

	Ethnicity				Sex			Men only			Women only		
	Total	Whites	Indians	χ^2	Men	Women	χ^2	Whites	Indians	χ^2	Whites	Indians	χ^2
Residence halls	48.1	49.8	36.2	3.76	51.4	45.6	1.62	53.4	41.2	1.70	47.2	29.2	2.86
Own home	41.4	40.0	51.8	2.85	52.7	32.6	19.37*	53.2	50.0	0.12	30.6	54.6	5.26*
Night clubs, bars, etc.	45.4	48.7	20.7	16.18*	49.3	42.3	2.37	55.9	14.7	19.37*	43.6	29.2	1.86
Restaurants	65.8	67.7	51.7	5.78*	68.5	63.6	1.30	73.2	44.1	11.20*	63.7	62.5	0.01
Friend's home	60.6	59.7	67.2	1.21	65.7	56.6	4.15*	65.9	64.7	0.02	55.2	70.8	2.17
City parks	37.8	38.3	34.5	0.31	52.4	26.5	33.96*	54.5	41.2	2.03	26.6	25.0	0.03
City streets	27.3	28.4	19.0	2.29	39.2	18.0	26.83*	42.1	23.5	4.15*	18.6	12.5	0.54
Parked cars	54.0	56.7	34.5	10.15*	65.4	45.2	19.49*	70.6	38.2	13.22*	46.8	29.2	2.74

* $P < .05$.

mized at the midpoint (ranging from "never" to "very often"), percentages do not reflect the number of times that respondents drank in the given location but the frequency with which they drank there more often. Overall, a somewhat greater difference between White and Indian students was found when drinking locations were considered but, again, a higher percentage of White students reported drinking in most places. White students drank significantly more often in nightclubs or bars, in restaurants and in parked cars.

Again, drinking was more frequent among men and, when men and women were examined by ethnicity, some interesting differences emerged. White men drank more often than Indian men in all locations, four of which were significant—nightclubs or bars, restaurants, city streets and parked cars. Indian women drank significantly more often than White women in their own homes. In total reported frequencies, Indian women were closer to Indian men than White women were to White men. In no instance did White women report a higher frequency than White men, but Indian women drank more often than Indian

men in three locations—at home, in restaurants and in the homes of friends.

Reasons for drinking

Questions on reasons for drinking followed Cahalan and Cisin's (1968) model and were divided into three response areas: social reasons, hedonistic reasons and escapist reasons (Table 3). Cahalan and Cisin reported that the escapist reasons constituted a Guttman scale with a reproducibility of 0.96. A prior factor analysis with this questionnaire had indicated the same divisions, and all items were found to load well on the three distinct orthogonal factors. A twelfth item, drinking to "get high," was included in the questionnaire but not in the scales. These responses also ranged from "never" to "very often" and were dichotomized at the 50% point. The trend for White students to surpass Indians was considerably less apparent in reasons for drinking; for half of the reasons, a larger percentage of Indians appeared in the high categories. Whites reported drinking more often for hedonistic

TABLE 3. Reasons for drinking by ethnicity and sex, in percent, with percentages given only for the high categories of each variable

	Ethnicity				Sex			Men only			Women only		
	Total	Whites	Indians	χ^2	Men	Women	χ^2	Whites	Indians	χ^2	Whites	Indians	χ^2
Social reasons													
Get along better on dates or other social occasions	54.3	54.2	55.2	0.02	65.0	46.0	17.42*	66.1	58.8	0.67	45.6	50.0	0.17
Be sociable	49.0	50.0	41.4	1.52	53.7	45.2	3.48	57.2	35.3	5.53*	44.8	50.0	0.24
Friends drink	61.7	61.0	67.2	0.85	65.4	58.8	2.21	66.1	61.8	0.24	57.3	75.0	2.84
Adult thing to do	22.7	21.8	29.8	1.87	34.9	13.3	31.50*	35.2	33.3	0.04	12.2	25.0	3.14
Hedonistic reasons													
Celebrate	54.5	57.2	34.5	10.67*	54.7	54.4	0.00	60.6	23.5	15.32*	54.8	50.0	0.21
Enjoyment of taste	38.5	41.2	19.0	10.63*	47.9	31.3	13.86*	53.1	20.6	12.09*	32.7	16.7	2.61
Feel good	48.7	50.8	32.8	6.67*	53.6	44.9	3.60	57.6	32.4	7.33*	46.0	33.3	1.41
Get drunk	58.1	59.1	50.9	1.38	67.1	51.1	12.52*	69.5	54.2	2.82	51.6	45.8	0.29
Escapist reasons													
Help relax	61.7	63.1	51.7	2.79	65.4	58.8	2.21	68.9	47.1	6.02*	58.9	58.3	0.00
Relieve aches, pains or fatigue	30.0	29.2	36.2	1.19	38.3	23.5	12.46*	38.9	35.3	0.16	22.2	37.5	2.86
Improve appetite	21.4	19.7	34.5	6.65*	33.2	12.2	31.31*	32.2	38.2	0.47	10.5	29.2	7.11*
Get high	47.2	44.0	70.7	14.59*	61.1	36.4	29.18*	57.6	79.4	5.70*	34.3	58.3	5.47*

* $P < .05$.

reasons whereas Indians tended to cite social and escapist reasons. Whites reported drinking significantly more often to celebrate special occasions, for enjoyment of taste and for a sense of well-being or to feel good, whereas Indians reported drinking more often to improve appetite for food and to "get high."

Men reported drinking more often than women for every reason, significantly so for seven of the 12 reasons. The effects of ethnicity by sex remained somewhat similar to the effects of ethnicity in the sample. That is, White men reported drinking significantly more often than Indian men to celebrate special occasions, for enjoyment of taste, to feel good, to relax and to be sociable. Indian men, on the other hand, drank significantly more often than White men to "get high."

Although White men reported more frequent drinking than Indian men for most reasons, the opposite was true of women. Indian women indicated significantly more drinking than did White women only to improve appetite for food and to "get high."

Problem drinking

Questions regarding problem drinking were taken from research by Engs (1977), and responses again ranged from "never" to "very often." Respondents were asked how often their drinking had led to the 12 problems listed in Table 4. Variables were dichotomized into those who reported that they had never experienced the problem and those who reported that they had. Therefore, the percentages reflect frequencies with which the particular behaviors were actually reported.

In many areas of problem behavior, White and Indian students indicated quite similar responses, but

a somewhat greater percentage of Indians appeared in the high categories of seven of the 12 problems. The significant differences, though, were more often in areas in which Whites reported greater difficulties. Whites significantly more often reported nausea or vomiting, drinking and driving, and behavior that was later regretted. Indians significantly more often reported being arrested and thinking that they had a drinking problem.

Men indicated a higher frequency of problem behavior than did women, most differences being significant. White men generally reported more problem behavior than Indian men. Only in arrests and in being criticized by a date did Indian men indicate more problems than White men, and these differences were not significant. Reported significantly more often by White men were the problems of nausea or vomiting, interference with school or work, drinking and driving, damaging property and behavior that was later regretted.

Differences between White and Indian women, however, were mostly in the opposite direction. With the exception of nausea or vomiting, and drinking and driving, Indian women indicated higher frequencies than did White women. Two of these were significant: interference with school or work and concern about a drinking problem. White women reported fewer problems than White men in every area, but Indian women exceeded Indian men in over half of the problem areas. In total incidence of reported problems, White men indicated a mean of 47.7%, Indian women 39.9%, Indian men 36.3% and White women 31.4%.

Discussion

The present study was exploratory in nature and involved a limited number of Indian tribes in only one

TABLE 4. Problem drinking by ethnicity and sex, in percent

	Ethnicity				Sex			Men only			Women only		
	Total	Whites	Indians	χ^2	Men	Women	χ^2	Whites	Indians	χ^2	Whites	Indians	χ^2
Hangover	68.5	68.9	65.5	0.27	72.4	65.4	2.64	75.0	58.8	3.73	64.5	75.0	1.06
Nausea or vomiting	62.6	64.4	50.0	4.51*	66.7	59.6	2.56	71.0	44.1	9.28*	59.7	58.3	0.02
Blacking out	31.3	30.7	36.2	0.73	34.3	20.0	1.51	34.7	32.4	0.07	27.8	41.7	2.04
Interference													
with school or work	35.1	34.9	36.2	0.04	44.8	27.6	15.38*	48.3	26.5	5.49*	25.4	50.0	6.63*
Human relations	39.6	39.2	43.1	0.33	42.9	37.1	1.62	44.3	35.3	0.95	35.5	54.2	3.27
Drinking and driving	55.2	57.1	41.4	5.08*	67.6	45.6	23.26*	71.6	47.1	7.38*	46.8	33.3	1.59
Arrested for drunken driving													
or public intoxication	5.2	4.3	12.1	6.35*	10.5	1.1	21.17*	9.1	17.7	2.22	0.8	4.2	2.27
Criticized by date	24.3	23.1	32.8	2.58	32.9	17.7	14.92*	31.8	38.2	0.53	16.9	25.0	0.98
Fighting	31.0	30.3	36.2	0.84	40.0	24.0	14.19*	40.3	38.2	0.05	23.1	33.3	1.26
Damaging property	21.2	21.9	15.5	1.26	38.6	7.7	67.61*	42.1	20.6	5.54*	7.7	8.3	0.01
Regretting behavior	62.0	63.6	50.0	4.00*	67.9	57.4	5.62*	72.6	44.1	10.58*	57.3	58.3	0.01
Drinking problem	21.5	19.7	34.5	6.64*	32.1	13.3	24.68*	32.0	32.4	0.00	10.9	37.5	13.41*

* $P < .05$.

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es.

Another surprising finding was the frequency of problem drinking in this group are not those normative behavior. Indian women or Indian White women but not Indian men, and the difference was less often in bars, parks or other escapist reasons for drinking. In four of the 12 problem areas, this group indicated that men in three other areas. Of particular concern were the problems of blacking out at school or work, and difficulties of future alcoholism. These areas often considered more serious may also have had some perception they expressed the most concern that they might have a problem. Research is needed to explain particularly in regard to changing power.

they were largely under the legal age for purchasing these beverages. Whites reported drinking more often for hedonistic reasons, but Indians in homes or restaurants. Whites reported drinking more often for "get high." The incidences of problem behavior for Whites and Indians were fairly high and of about equal value. White men indicated the highest mean percentage of problems, followed by Indian women. Indian men and White women. It is not too surprising that over half experienced some of the behaviors (e.g., having a hangover) but the percentages of both Whites and Indians who reported blacking out from drinking appear excessive in light of the contention that this phenomenon could be indicative of future alcoholism (Straus and Bacon, 1953). This could only lend emphasis to recent concern over drinking patterns of our nation's youth.

The commonly held stereotype of the American Indian drinking in city parks was not upheld by this research since these Indian students appeared to be considerably less visible than Whites in their drinking locations. The Indian students were also less inclined to report drinking and driving, fighting after drinking or damaging property—all highly visible and socially censured behaviors. A surprising finding in this context, therefore, was the relatively high arrest rate for Indian students. Forslund and Meyers (1974) reported a disproportionate arrest rate among reservation youth, and our research supported these findings.

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References

- BAHR, H. M., CHADWICK, B. A. AND STAUSS, J. H. *American Ethnicity*, Lexington, Mass.: D.C. Heath Co., 1979.
- BENNION, L. J. AND LI, T. K. Alcohol metabolism in American Indians and Whites: Lack of racial differences in metabolic rate and liver alcohol dehydrogenase. *New Engl. J. Med.* 294: 9-13, 1976.
- BRINKER, P. A. AND KLOS, J. J. *Poverty, Manpower, and Social Security*, Austin, Tex.: Lone Star Pubs., Inc., 1976.
- CAHALAN, D. AND CISIN, I. H. American drinking practices: Summary of findings from a national probability sample. I. Extent of drinking by population subgroups. *Q. J. Stud. Alcohol* 29: 130-151, 1968.
- COCKERHAM, W. C. Patterns of alcohol and multiple drug use among rural White and American Indian adolescents. *Int. J. Addict.* 12: 271-285, 1977.
- DUTORT, B. M. Substitution: A process in cultural change. *Hum. Org.* 23: 16-23, 1964.
- ENGES, R. C. Drinking patterns and drinking problems of college students. *J. Stud. Alcohol* 38: 2144-2156, 1977.
- FARRIS, J. J. AND JONES, B. M. Ethanol metabolism and memory impairment in American Indian and White women social drinkers. *J. Stud. Alcohol* 39: 1975-1979, 1978.
- FENNA, D., MIX, L., SCHAEFER, O. AND GILBERT, J. A. L. Ethanol metabolism in various racial groups. *Can. Med. Ass. J.* 105: 472-475, 1971.
- FORSLUND, M. A. AND MEYERS, R. E. Delinquency among Wind River Indian Reservation youth. *Criminology* 12: 97-106, 1974.
- GALLUP, G. H. *The Gallup Poll*, Wilmington, Del.: Scholarly Resources, Inc., 1980.
- HANSON, D. J. Trends in drinking attitudes and behaviors among college students. *J. Alc. Drug Educ.* 22: 17-22, 1977.
- HOWARD, J. *North American Indian Cultures*. (Independent and Correspondent Study), Stillwater: Oklahoma State University, 1978.
- JESSOR, R., GRAVES, T. D., HANSON, R. C. AND JESSOR, S. L. *Society, Personality and Deviant Behavior*, New York: Holt, Rinehart & Winston, Inc., 1968.
- KLINE, J. A., ROZYNKO, V. V., FLINT, G. AND ROBERTS, A. C. Personality characteristics of male Native American alcoholic patients. *Int. J. Addict.* 8: 729-732, 1973.
- LEMERT, E. M. Alcoholism: Theory, problem and challenge. III. Alcoholism and the sociocultural situation. *Q. J. Stud. Alcohol* 17: 306-317, 1956.
- LITTMAN, G. Alcoholism, illness, and social pathology among American Indians in transition. *Am. J. Publ. Hlth* 60: 1769-1778, 1970.
- LURIE, N. O. The world's oldest on-going protest demonstration: North American Indian drinking patterns. *Pacif. Hist. Rev.* 40: 311-332, 1971.
- MACANDREW, C. AND EDGERTON, R. B. *Drunken Comportment: A Social Explanation*, Chicago: Aldine Publishing Co., Inc., 1969.
- MAYNARD, E. Drinking as a part of an adjustment syndrome among the Oglala Sioux. *Pine Ridge Res. Bull. S.D.* 9: 35-51, 1969.
- PRICE, J. A. An applied analysis of North American Indian drinking patterns. *Hum. Org.* 34: 17-26, 1975.
- STEWART, C. Questions regarding American Indian criminality. *Hum. Org.* 23: 61-66, 1964.
- STRATTON, R., ZEINER, A. AND PAREDES, A. Tribal affiliation and prevalence of alcohol problems. *J. Stud. Alcohol* 39: 1166-1177, 1978.
- STRAUS, R. AND BACON, S. D. *Drinking in College*, New Haven, Conn.: Yale University Press, 1953.
- STRIMBU, J. L., SCHOENFELDT, L. F. AND SIMS, O. S., JR. Drug usage in college students as a function of racial classification and minority group status. *Res. Higher Educ.* 1: 263-272, 1973.
- UECKER, A. E., BOUTILIER, L. R. AND RICHARDSON, E. H. "Indianism" and MMPI scores of men alcoholics. *J. Stud. Alcohol* 41: 357-362, 1980.
- U.S. HEALTH SERVICES ADMINISTRATION. *Indian Health Trends and Services, 1979 Edition*, Washington, D.C.: U.S. Government Printing Office, 1978.
- WECHSLER, H. AND MCFADDEN, M. Drinking among college students in New England: Extent, social correlates and consequences of alcohol use. *J. Stud. Alcohol* 40: 969-996, 1979.
- WOLFF, P. H. Ethnic differences in alcohol sensitivity. *Science* 175: 449-450, 1972.

References

- ALLMAN, L. R., TAYLOR, H. A. AND NATHAN, P. E. Group drinking during stress: Effects on drinking behavior, affect, and psychopathology. *Am. J. Psychiat.* 129: 669-678, 1972.
- CASH, T. F., KEHR, J. A., POLYSON, J. AND FREEMAN, V. Role of physical attractiveness in peer attribution of psychological disturbance. *J. Consult. Clin. Psychol.* 45: 987-993, 1977.
- FARINA, A., ALLEN, J. G. AND SAUL, B. B. B. The role of the stigmatized person in affecting social relationships. *J. Personality* 36: 169-182, 1982.
- FARINA, A., GLIHA, D., BOUDREAU, L. A., ALLEN, J. G. AND SHERMAN, M. Mental illness and the impact of believing others know about it. *J. Abnorm. Psychol.* 77: 1-5, 1971.
- FARINA, A., MURRAY, P. J. AND GROH, T. Sex and worker acceptance of a former mental patient. *J. Consult. Clin. Psychol.* 46: 887-891, 1978.
- GOFFMAN, E. *Stigma: Notes on the management of spoiled identity*, Englewood Cliffs, N.J.: Prentice-Hall, Inc., 1963.
- HAMILTON, F. AND MAISTO, S. A. Assertive behavior and perceived discomfort of alcoholics in assertion-required situations. *J. Consult. Clin. Psychol.* 47: 196-197, 1979.
- HAWKINS, R. AND TIEDEMAN, G. *The creation of deviance: Interpersonal and organizational determinants*, Columbus, Ohio: Charles E. Merrill Publishing Co., 1975.
- HIGGINS, R. L. AND MARLATT, G. A. Fear of interpersonal evaluation as a determinant of alcohol consumption in male social drinkers. *J. Abnorm. Psychol.* 84: 644-651, 1975.
- KILTY, K. M. Drinking status and stigmatization. *Am. J. Drug & Alcohol Abuse* 8: 107-116, 1981.
- KILTY, K. M. AND MEENAGHAN, T. M. Drinking status, labeling, and social rejection. *J. Soc. Psychol.* 102: 93-104, 1977.
- LANGER, E. J. AND ABELSON, R. P. A patient by any other name . . . Clinician group difference in labeling bias. *J. Consult. Clin. Psychol.* 42: 4-9, 1974.
- MARLATT, G. A. Craving for alcohol, loss of control, and relapse: A cognitive-behavioral analysis. In: NATHAN, P. E., MARLATT, G. A. AND LÖBERG, T. (Eds.) *Alcoholism: New Directions in Behavioral Research and Treatment*, (NATO Conference Series: III, Human Factors, Vol. 7), New York: Plenum Press, 1978, pp. 271-314.
- MILLER, P. M., HERSEN, M., EISLER, R. M. AND HILSMAN, G. Effects of social stress on operant drinking of alcoholics and social drinkers. *Behav. Res. Ther.* 12: 67-72, 1974.
- O'BRIEN, L. J., ROSSI, P. H. AND TESLER, R. C. How much is too much? Measuring popular conceptions of drinking problems. *J. Stud. Alcohol* 43: 96-109, 1982.
- POE, C. A. Development of a psychological effectiveness scale. *J. Psychol.* 85: 81-85, 1973.
- ROSENHAN, D. L. On being sane in insane places. *Science* 179: 250-257, 1973.
- SCHIEFF, T. J. *Labeling Madness*, Englewood Cliffs, N.J.: Prentice-Hall, Inc., 1975.
- TRICE, H. M. AND ROMAN, P. M. Delabeling, relabeling, and Alcoholics Anonymous. *Soc. Probl.* 17: 538-546, 1970.
- TWENTYMAN, C. T., GREENWALD, D. R., GREENWALD, M. A., KLOSS, J. D., KOVALESKI, M. E. AND ZIBUNG-HOFFMAN, P. An assessment of social skill deficits in alcoholics. *Behav. Assess.* 4: 317-326, 1982.

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Alcohol, Aggression and Assertiveness in Men: Dosage and Expectancy Effects*

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ABSTRACT. The effect of alcohol on aggression and assertiveness was examined in 54 men college students. A 2 (high vs low dosage expectancy) $\times$ 3 (0.0, 0.5 and 1.0 ml of 95% alcohol per kg of body weight) design was used. There was an increase in self-reported aggression at the moderate dosage

but an increase only in profanity at the high dosage. The expectancy manipulation also produced an increase in self-reported aggression. Actual dosage and dosage expectancy did not influence assertiveness. (*J. Stud. Alcohol* 45: 275-278, 1984)

EVIDENCE suggests that aggressive, violent and illegal behaviors are more likely to occur when individuals have consumed alcohol. Physiological and expectancy factors have been postulated as causal

elements. Several studies on humans (Bennett et al., 1969; Shuntich and Taylor, 1972; TAYLOR, S. P. AND GAMMON, C. B. The effect of type and dose of alcohol on human aggression. Presented at a meeting of the American Psychological Association, New Orleans, 1974) have reported an increase in aggression as a function of dosage. Similar findings have been reported with animals (Raynes et al., 1968). On the other hand, cross-cultural research (e.g., MacAndrew and Edgerton, 1969) has focused on the contribution of social, cognitive and expectancy factors. One study

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(Lang et al., 1975), directly comparing dosage and expectancy effects, found that aggression increased when subjects expected to receive alcohol under both provoked and nonprovoked contingencies. There were no differences as a function of actual consumption.

The present study is an attempt to estimate the contribution of psychological (expectancy) and physiological (dosage) factors to aggressive behavior following alcohol consumption. This study explores alternatives to the Buss aggression machine for measuring aggression to try to improve on previous research. The following hypotheses were made: (1) aggression will vary directly as a function of dosage, (2) subjects given a higher dosage expectancy will behave more aggressively than subjects given a lower dosage expectancy and (3) dosage and expectancy effects will combine additively.

Although the effect of alcohol on aggression was the primary focus of this study, the effect of alcohol on assertiveness was also tested. Research has suggested that assertiveness and aggression are different constructs and that they may therefore respond differentially to alcohol consumption. It was hypothesized that both high dosage and high expectancy manipulations would decrease appropriate assertive responses.

Method

A 3×2 random-group factorial design was employed. Independent variables were three alcohol dosages and dosage expectancy (high or low). Subjects were 54 men students in introductory psychology. Potential subjects had been administered a standardized questionnaire on their alcohol use, which allowed selection of a sample composed of social drinkers. Two potential subjects were eliminated because of possible alcohol dependency.

Informed consent was obtained and subjects were told that, following consumption of a dosage of alcohol based on their weight, they would be asked to complete several tasks designed to measure aspects of mental functioning such as decision making, hand-eye coordination and reasoning.

Prior to their arrival subjects were randomly assigned to one of the six treatment conditions. Subjects were informed that they were to receive approximately 24 oz of beer that was either one-half as strong (low dosage expectancy) or twice as strong (high dosage expectancy) as commercially available beer. Metbrew Near Beer, a nonalcoholic beer, was used as the basic beverage for all subjects. Dosage levels were either 0.0, 0.5 or 1.0 ml of 95% alcohol per kg of body weight. All subjects consumed 24 oz of beer. The particular dosages selected were commensurate with those used by other researchers (e.g., Shuntich and Taylor, 1972) on alcohol and aggression. A "floater" (1 ml of alcohol) was added to the surface of the nonalcoholic beverage to enhance placebo effectiveness.

Subjects were weighed, were brought a pitcher containing the beer mixture, and were told that they would have 20 min

to finish the beer and that they should pace themselves at a rate of about 8 oz of beer every 5 min. A 20-min waiting period following consumption was enforced to ensure alcohol absorption. The experimenter then joined the subject in the experimental room to administer the tests.

Several dependent measures were employed. A modified version of the Behavioral Assertiveness Test (Eisler et al., 1973) was given to subjects. An audiotaped series of ten hypothetical situations, modified for the college population, was presented to each subject. A narrator provided a description of each situation, followed by an initial statement made by an actor that called for a response from the subject. Subjects were asked to respond to the actor's statement as if the situation were really occurring. Subjects' responses were audiotaped for later scoring. Five measures of assertiveness were obtained for each of the 10 situations: response latency, affect, loudness, compliance and requests for new behavior. Reliability was established for the five assertiveness measures using two blind judges with 85% set a priori as the minimum level of interrater reliability. Subjects were also administered the self-report Assertiveness Scale designed by Rathus (1973). The Buss-Durkee Hostility Inventory (1957) was used as a self-report measure of aggression. The scores on the subscales Assault, Negativism, Verbal Hostility and Indirect Hostility were added together to yield the Total Hostility score. Verbal aggression was measured by recording the occurrences of profanity for each subject during administration of the Behavioral Assertiveness Test.

Subjects were administered the Buss-Durkee Hostility Inventory, the Rathus Assertiveness Scale and the Behavioral Assertiveness Test in a counterbalanced sequence. The experimenter was present only during the Behavioral Assertiveness Test in order to operate the tape recorder. To reinforce the belief that the study was primarily concerned with decision making, hand-eye coordination and reasoning ability, and to reduce the reaction to measures of aggression and assertiveness, subjects were also administered the Digit-Symbol subtest of the Wechsler Adult Intelligence Scale (1955) and the Shipley-Institute of Living Scale (1931), in a counterbalanced sequence. Instructions for all tests were presented via a tape recorder.

During debriefing, subjects were questioned on their perception of the amount of alcohol consumed, the purpose of the experiment and the expected results. Subjects remained in the laboratory until they were sober enough to leave.

Results

An overall multivariate analysis of variance (ANOVA) was performed with the two aggression measures serving as dependent variables, and dosage expectancy and actual dosage serving as independent variables. The ANOVA indicated main effects of dosage expectancy ($F < 3.5$, 1/48 df, $p < .05$) and actual dosage ($F = 3.9$, 2/48 df, $p < .01$) but no interaction effect. A subsequent univariate ANOVA revealed a main effect of dosage expectancy ($F = 7.0$, 1/48 df, $p < .05$) on Buss-Durkee Total Hostility scores, sub-

jects with high dosage expectancy yielding significantly higher scores (mean, 23.7) than those with low dosage expectancy (19.9). A main effect of actual dosage ($F = 4.0$, 2/48 df, $p < .05$) was also evident. Newman-Keuls analyses indicated that the moderate dosage produced greater hostility than the placebo dosage. None of the remaining comparisons was significant. Examination of the group means presented in Figure 1 suggests that the relationship between alcohol consumption and aggression may be curvilinear rather than linear. Furthermore, the effects of dosage expectancy and actual dosage appear to be reliable and additive.

A univariate ANOVA performed with occurrences of profanity during the Behavioral Assertiveness Test serving as the dependent measure revealed no main effect for dosage expectancy. However, there was a main effect for actual dosage ($F = 3.6$, 2/48 df, $p < .05$). Newman-Keuls analyses indicated that the high dosage group used significantly more profanity than either the placebo or moderate dosage group. The mean ($\pm$ SD) profanity scores for the placebo, moderate and high dosages were 0.15 ± 0.35 , 0.30 ± 0.70 and 1.15 ± 1.65 , respectively. The interaction was not significant.

Interrater reliabilities for the Behavioral Assertiveness Test measures ranged from .90 to .99. Consequently, a multivariate ANOVA was performed with the Rathus Assertiveness total score and Behavioral Assertiveness scores serving as the dependent measures. Results indicated no main effect for either actual dosage or dosage expectancy and no significant

interaction between main effects. Low correlations between measures of assertiveness and aggression reinforce the notion that these represent different constructs.

To assess the effectiveness of the placebo dosage, subjects were asked to estimate the amount of alcohol that they had consumed. For these estimates, main effects of both dosage and expectancy were significant. Newman-Keuls comparisons revealed no differences between the placebo and high dosages, demonstrating the effectiveness of the placebo. This analysis also suggests that the expectancy manipulation was effective since differences were in the expected direction. Answers to questions on the nature of the study revealed that subjects were unaware that aggression and assertiveness were being measured.

Discussion

These findings suggest that both dosage and expectancy affect aggressive behavior following alcohol consumption. The increase in Buss-Durkee Total Hostility scores with a moderate dosage of alcohol is consistent with Raynes et al.'s (1968) finding that there is a curvilinear relationship between alcohol dosage and aggression. The well-known depressant effect of alcohol may be responsible for the lack of increased aggression at high dosages. This finding is probably limited to laboratory studies using non-provocative methods. In contrast, a high, rather than a moderate, dosage of alcohol was found to increase profanity. Differential findings between self-report and behavioral measures may reflect the fact that self-report measures rely primarily on cognitive functioning, which may be more prone to the depressant effects of alcohol than are behavioral measures. Behavioral responses may be more reflexive in nature or controlled by disinhibitory factors. These findings are consistent with most previous research.

Expectancy factors were also found to contribute to increased aggression. Lang et al. (1975) reported that expectancy, but not dosage, was related to increased aggression. Lang et al. studied heavy drinkers rather than social drinkers and used a dosage 30% higher than the highest one used in the present study. These methodological variations may account for the differences in findings, especially if there is a curvilinear relationship between dosage and aggression.

In the present study, dosage and expectancy were found to affect aggression but not assertiveness. Although there is little agreement on how best to measure aggression in laboratory studies, self-report and behavioral-simulation approaches deserve further exploration. If a curvilinear relationship between alcohol dosage and aggression exists, then multiple dosage

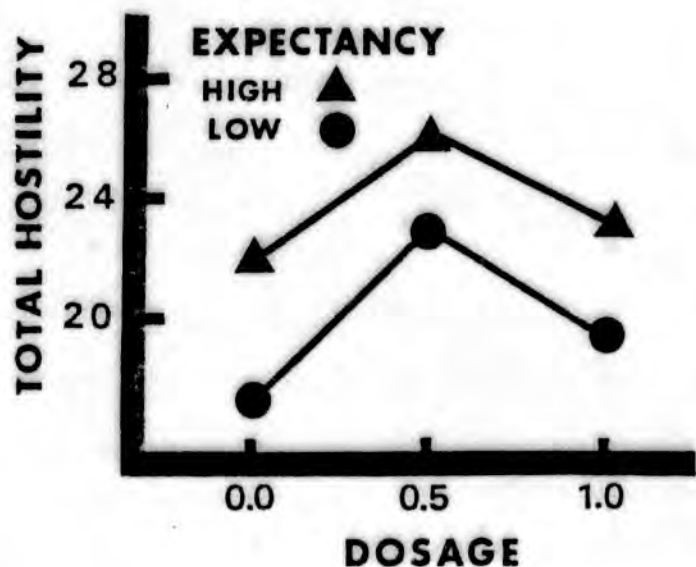


FIGURE 1. Total Hostility scores as a function of actual dosage and of expectancy.

levels, as well as an effective placebo control, seem necessary for subsequent research.

References

- BENNETT, R. M., BUSS, A. H. AND CARPENTER, J. A. Alcohol and human physical aggression. *Q. J. Stud. Alcohol* 30: 870-876, 1969.
- BUSS, A. H. AND DURKEE, A. An inventory for assessing different kinds of hostility. *J. Consult. Psychol.* 21: 343-348, 1957.
- EISLER, R. M., MILLER, P. M. AND HERSEN, M. Components of assertive behavior. *J. Clin. Psychol.* 29: 259-299, 1973.
- LANG, A. R., GOECKNER, D. J., ADESSO, V. J. AND MARLATT, G. A. Effects of alcohol on aggression in male social drinkers. *J. Abnorm. Psychol.* 84: 508-518, 1975.
- MACANDREW, C. AND EDGERTON, R. B. *Drunken Comportment: A Social Explanation*, Chicago: Aldine Publishing Co., Inc., 1969.
- RATHUS, S. A. A 30-item schedule for assessing assertive behavior. *Behav. Ther.* 4: 398-406, 1973.
- RAYNES, A. E., RYBACK, R. AND INGLE, D. The effect of alcohol on aggression in *Betta splendens*. *Commun. Behav. Biol.* 2A: 141-146, 1968.
- SHIPLEY, W. C. *Shipley-Institute of Living Scale for Measuring Intellectual Impairment: Manual of Directions and Scoring Key*, Hartford, Conn.: Institute of Living, 1931.
- SHUNTICH, R. J. AND TAYLOR, S. P. The effects of alcohol on human physical aggression. *J. Exp. Res. Personality* 6: 34-38, 1972.
- WECHSLER, D. *Manual for the Wechsler Adult Intelligence Scale*, New York: Psychological Corp., 1955.

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Alcohol and Aggression in Men: A Comparison of Brewed and Distilled Beverages*

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ABSTRACT. The effects of distilled and brewed alcoholic beverages on aggressive behavior were compared in a 2×2 factorial design with 16 subjects per cell ($N = 64$ men social drinkers). One half of the sample population was told that they would be consuming an alcoholic (distilled spirits) beverage and the other half that they would be consuming beer. Within each of these conditions, one-half of the subjects received the expected drug (alcohol) and one-half was given a

corresponding placebo beverage. Aggression was measured using a modified version of the Buss aggression machine. Subjects who consumed beer or believed that they were consuming beer were significantly less aggressive than subjects who consumed distilled spirits or believed that they were consuming distilled spirits. Differences between subjects receiving the drug (alcohol) and the placebo were not significant. (*J. Stud. Alcohol* 45: 278-282, 1984)

EVIDENCE from crime statistics and from anthropological studies points to a pervasive relationship between alcohol consumption and aggressive behavior (Pernanen, 1976). Studies generally indicate that alcohol is implicated in a high percentage of assaults, murders, rapes and suicides. In laboratory studies, increased aggressive behavior has been related to the type and dose of alcohol ingested (Boyatzis, 1974; Takala et al., 1957; Taylor and Gammon, 1975) and to the subjects' believed consumption of alcohol regardless of the actual content of the administered

drinks (Lang et al., 1975; Pihl et al., 1981). This latter expectancy effect may also account for some of the observed variations in behavior found as a result of administration of different types of beverages.

In an experimental study of interpersonal aggressive behavior in small group settings, Takala et al. (1957) reported increased aggression with increased alcohol intake, and significantly more aggression at the same blood alcohol level (BAL) in subjects consuming distilled spirits (brandy) as compared with brewed beverages (beer). The authors concluded that differences in physiological responses to the two types of beverages, possibly as a result of differences in congener content, most likely contributed to the finding. The effect of congeners found in certain alcoholic beverages on physiology and behavior is in itself a controversial

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issue. Congeners have been reported (Katkin et al., 1970) to have a variety of effects on behavior including increased risk taking and decreased performance on psychomotor tasks. However, in one study comparing the physiological effects of whisky, beer and wine (Kalant et al., 1975), no differences were observed as a result of beverage type on absorption or metabolism of alcohol or on performance on several sensorimotor tasks when the beverage was administered under conditions closely comparable with those in which alcohol is generally consumed. Furthermore, beer has significantly higher congener content proportionately than do distilled spirits (Greizerstein, 1981).

In another attempt to compare the effects of different types of beverages on aggression, subjects consumed either various types of distilled spirits or beer ad libitum in an experimental party situation (Boyatzis, 1974). Subjects in the distilled spirits condition were observed to be significantly more interpersonally aggressive than subjects in the beer condition. Problematically, this study employed several types of distilled beverages and the summary measure of aggression was broad; such behaviors as joking, dramatizing, expertizing and moralizing were included in the aggression measure.

In the present study, a comparison of the effects of distilled and brewed beverages on aggressive behavior is made using pure alcohol and beer. Aggression is measured in a controlled experimental situation, operationally defined as the intensity and duration of shock administered to a bogus partner.

Method

Subjects

Sixty-four men social drinkers, aged 18-35 (median age 23.8 yrs), were recruited through a newspaper advertisement as paid subjects. The advertisement stated "wanted male social drinkers, ages 18-35, for alcohol research study." All subjects were screened by means of a personal data questionnaire which requested demographic and drinking history information, and an interview to ascertain that the subject (1) did not have an alcoholism problem; (2) had never been treated or arrested in connection with excessive drinking; (3) did not have a hearing impairment; (4) had no previous familiarity with psychological experimentation; and (5) drank alcohol regularly but not in excess of seven beers per day. Because a high alcohol tolerance could attenuate the effects of alcohol ingestion in the laboratory, heavy drinkers were excluded from the study. Subjects were asked not to ingest any drugs or alcohol for at least 24 hr or food for at least 4 hr prior to the drinking session.

Procedure

A randomized, double-blind, 2×2 factorial design with 16 subjects per cell was used. The two independent variables

that were manipulated were drug and beverage type with subjects receiving a brewed or orange juice beverage with or without alcohol.

Task and instructions. The task, similar to the one used by Zeichner and Pihl (1979, 1980) was introduced to the subject as a reaction-time or pain-perception task in which the subject was to be tested for reaction time while his "partner" in the adjoining room was to be tested for perception of pain. The bogus partner was an operant-conditioning apparatus. The subject was told that both behaviors would be examined under the influence of alcohol in the following manner: Shortly after the appearance of a light on the subject's console, the partner would administer a 3000 Hz tone through the subject's headphones (stimulus tone). After a 5-sec exposure to this tone, a second light would indicate to the subject to press, as quickly as possible, one of the five buttons on his console. Pressing any of these buttons simultaneously terminated the stimulus tone and delivered an electric shock to the partner. It was stressed that although button number 1 delivered a barely perceptible shock, buttons 2-5 activated increasingly painful shock levels. The subject was led to believe that after having received the shock the partner communicated to him the degree of pain which he had felt by means of the tone level of his choice (contingency tone). This self-terminating contingency tone continued for 5 sec and was accompanied by a light signal indicating to the subject which loudness level was chosen by the partner. This constituted one trial.

Each subject participated in 22 consecutive trials spaced by 20-sec intervals to ensure clear differentiation between trials. The five contingency tone levels were meant to symbolically correspond to the five shock intensities that the subject could administer. Unknown to the subject, the level of each of the contingency tones was prearranged to correspond fully to the level of shock intensity chosen by the subject. During the task, two objective measures of aggression were recorded: the shock intensity that the subject selected and delivered to his partner, and the duration of time that the subject depressed each button.

Video deception. As the task instructions were communicated to the subject by the experimenter, the former could observe himself and the experimenter on a 15-cm television monitor. This was done by means of a closed-circuit television system. Following the instructions, the subject was advised that the monitor would enable him to observe his partner as the partner was receiving his own task instructions. After the experimenter left the subject's room, a prerecorded videotape depicting the experimenter instructing the "other" subject in another room was replayed on the monitor. This actor was given a sample of the tone to be administered to the subject as well as examples of shock intensities 1 and 2. The videotape concluded with a segment in which the experimenter, turning to face the camera, requested the subject to press button 1 in order to ascertain if the equipment was functioning properly. The subject's response, monitored in an adjacent room, provided proof of the deception's success, which was further investigated during debriefing.

Drug conditions. At this point in the experiment, subjects were randomly assigned to one of the four conditions: (1) Alcohol condition—subjects were administered 1.32 ml per kg of body weight of 95% United States Pharmacopoeia (USP) alcohol in a 1:5 (alcohol:orange juice) solution. (2) Placebo-alcohol condition—subjects were administered orange juice in proportions equal to those of the alcohol condition, the alcohol part being replaced by juice and 2 cc of 95% USP alcohol layered on the drink's surface. Each glass was encased in a terry-cloth holder sprayed with 95% alcohol. (3) Beer condition—subjects were administered 21.3 ml/kg of Brador (6.2% v/w). (4) Placebo-beer condition—subjects were administered 21.3 ml/kg of a mixture of 3 parts Texas Pride to 1 part Panther, both of which are nonalcoholic malt beverages which when mixed look and taste like typical Canadian brewed beverages. Pure alcohol rather than a more frequently consumed beverage such as bourbon was chosen for the alcohol groups in order to reduce congener content, to produce results which can be compared with an often studied drug and dosage, and to accommodate the need for a believable placebo.

Drinks were consumed in a carpeted, softly lit room decorated and furnished like an apartment living room, while the subject listened to the music of his choice. All four conditions were assigned a 20-min period in which to consume the beverage. Subjects in the alcohol and placebo-alcohol conditions were given three drinks of equal quantity, the first two to be consumed in 6 min each and the last in 8 min. Subjects in the beer and placebo-beer conditions were given their full allotment of beverage at once, and told to pace their consumption over a 20-min period. This practice was adopted because of the greater volume to be consumed by the beer groups. A waiting period of 20 min followed to allow the alcohol-drinking subjects to reach an elevated BAL and also to provide sufficient time to complete the experiment before reaching BAL peak. Following the waiting period, a BAL reading was taken by means of a Smith & Wesson Breathalyzer (Model 900A) and the subject was asked to compare his present level of intoxication with the highest level of intoxication he had ever experienced on an 11-point Likert-type "how drunk" rating scale.

At this time, the aggression task commenced. Throughout the task period the subject was alone in the room. After the task was completed a second BAL reading was taken and the "how drunk" scale was again administered. Those in the alcohol condition remained in the laboratory until their BALs had dropped well below the legal level.

Ethical considerations

For ethical reasons (1) all subjects were pure volunteers and were not coerced in any way to participate in the experiment; (2) the possibility of pressing only shock button number 1 (which delivered a barely perceptible shock) was clearly stated; and (3) during the debriefing discussion, the subject was assured that he had not administered a shock to anyone and that his behavior had been perfectly normal and comparable with other subjects' behavior under the same conditions.

Results

Alcohol manipulation

BAL. All subjects in the alcohol and beer conditions achieved BALs within the "social drinking" experience. Measurable BALs were observed in four subjects in the placebo-alcohol condition and their data were not considered for further analysis. Although BALs were not tested at the initiation of the study, it is believed that the modest readings in these four subjects (maximum .03%) were attributable to overzealous mixing of the placebo-alcohol drink. No identifiable BALs were observed in the remaining placebo-alcohol subjects or in placebo-beer subjects. BALs for each group are presented in Table 1.

"How drunk" scale. Scores on the "how drunk" scale are also included in Table 1. A three-way repeated-measures analysis of variance (ANOVA) performed on these scores revealed a significant drug main effect ($F = 15.77$, 1/56 df, $p < .001$). No other main effects or interactions were significant on the measure. Posthoc comparison (Tukey's HSO) indicated that for both pre- and posttask measures the alcohol and beer means were significantly higher than the placebo-alcohol and placebo-beer means.

Measures of aggression

Shock intensity. The shock-intensity score for each subject was obtained by computing the mean of all shock choices that had been made. A two-way ANOVA comparison of these scores revealed no significant main effects or interactions.

TABLE 1. Mean ($\pm$ SD) BAL readings and scores on the "how drunk" scale for each group, means with different superscripts significantly different at the .05 level

	Alcohol (N = 16)	Beer (16)	Placebo alcohol (12)	Placebo beer (16)
BAL pretask	.070 $\pm$.017	.071 $\pm$.016	0.0 $\pm$ 0.0	0.0 $\pm$ 0.0
BAL posttask	.072 $\pm$.023	.071 $\pm$.015	0.0 $\pm$ 0.0	0.0 $\pm$ 0.0
"How drunk" pretask	4.63 $\pm$ 2.03 ^a	4.06 $\pm$ 2.02 ^a	2.17 $\pm$ 1.75 ^a	2.63 $\pm$ 0.89 ^a
"How drunk" posttask	4.56 $\pm$ 2.13 ^a	3.94 $\pm$ 1.95 ^a	2.42 $\pm$ 2.19 ^a	2.75 $\pm$ 1.34 ^a

Shock duration. Mean shock-duration scores for each subject at each level of shock intensity were computed. A three-way repeated measures ANOVA performed on these scores revealed only a significant main effect for beverage type ($F = 5.46$, $1/56$ df, $p < .03$). The repeated-measures effect was not significant nor were there any other significant main effects or interactions. Mean scores for the four treatment conditions at each of the five levels of shock intensity are presented in Figure 1. The frequency of responses for each group at each intensity level was examined as the percentage of total responses. The average percentage of total response at the intensity levels 1-5 were 27.4, 22.2, 16.4, 18.5 and 12.3%, respectively.

Discussion

The significant increase in aggression observed in subjects consuming distilled spirits as opposed to beer both confirms and extends the previous results of Takala et al. (1957) and Boyatzis (1974). In particular, this difference has now been demonstrated in a laboratory situation and with a narrowly defined dependent variable. The relatively uncontrolled social situations of the previous work, as well as the exceedingly broad definitions of aggression which were employed, had mitigated the substance of the earlier studies. It may well be that considerable insight into the mechanisms of the alcohol-aggression relationship will be garnered by further exploration of this differential effect of various alcoholic beverage types. An uncontrolled factor in the present study which should be investigated in future work is the differing volume consumed by subjects in the two beverage groups. Subjects in the beer groups drank considerably more liquid, a factor which conceivably could affect performance, although no complaints were heard from the subjects in regard to this and "how drunk" ratings between groups were remarkably similar.

The fact that shock duration and not shock intensity was the differentiating measure is of import. Studies of racism using a similar paradigm (Donnerstein and Donnerstein, 1972, 1975; Donnerstein et al., 1972) have shown that White subjects repeatedly delivered lower shock intensities to Blacks than Whites, but higher shock durations. Higher shock durations are thus felt to represent indirect expressions of aggression. The elevated shock-duration scores of the alcohol-placebo group, illustrated in Figure 1, also represent a significant finding. It suggests that the differential effect between pure alcohol and beer is related to expectations concerning the type of beverage consumed rather than the actual content of the beverage. This study therefore provides some replicative support for the results of Lang et al. (1975) where expectation was the main determinant of alcohol-related aggression. Survey studies which assess the social meaning attributed to various alcoholic beverages would support this interpretation. Typically, subjects expect alcohol to enhance social and physical pleasures, power and aggression, and social assertiveness (Brown et al., 1980). However, these effects are also seen as dose-dependent, with heavy drinking leading to impairment. Further, the subject's familiarity with alcohol greatly affects his definition of heavy drinking (Southwick et al., 1981). One survey (Lang et al., 1983) which examined subjects' expectations relative to beverage type found that college students expect the most positive experience following wine, and then in order, mixed drinks, beer and distilled spirits.

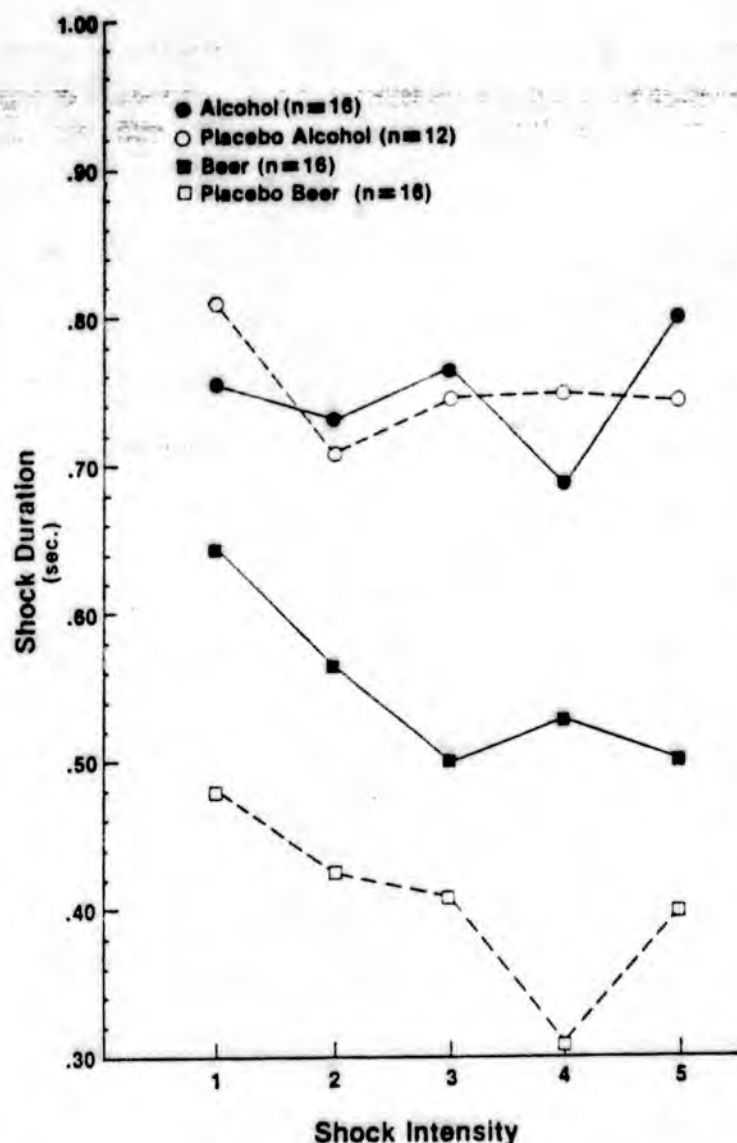


FIGURE 1. Mean shock duration scores at each of the five shock intensities for the alcohol, placebo-alcohol, beer and placebo-beer groups.

A speculative explanation for these findings would focus on an individual's differential drinking history associated with different beverage types. This history might take into account both chemical differences in the beverages as well as sociological-psychological differences surrounding their consumption. It may be, for example, that a differential conditioning history exists, in which alcohol consumption is associated with negative sociological events for some individuals, and that this association increases the likelihood of aggression. Furthermore, it is possible that beer consumption is associated with different events or situations which evoke a different set of behaviors. Conditioning models of drug misuse are well known (Wikler, 1973) but, in the case of alcohol, need to be explored further in terms of a specific drug-related response such as aggression.

References

- BOYATZIS, R. E. The effect of alcohol consumption on the aggressive behavior of men. *Q. J. Stud. Alcohol* 35: 959-972, 1974.
- BROWN, S. A., GOLDMAN, M. S., INN, A. AND ANDERSON, L. R. Expectations of reinforcement from alcohol: Their domain and relation to drinking patterns. *J. Consult. Clin. Psychol.* 48: 419-426, 1980.
- DONNERSTEIN, R. AND DONNERSTEIN, M. White rewarding behavior as a function of potential for black retaliation. *J. Personality Soc. Psychol.* 24: 327-333, 1972.
- DONNERSTEIN, R. AND DONNERSTEIN, M. The effects of attitudinal similarity on interracial aggression. *J. Personality* 43: 485-502, 1975.
- DONNERSTEIN, R., DONNERSTEIN, M., SIMON, S. AND DITRIDIA, R. Variables in interracial aggression: Anonymity, expected retaliation, and a riot. *J. Personality Soc. Psychol.* 22: 231-245, 1972.
- GREIZERSTEIN, H. B. Congener contents of alcoholic beverages. *J. Stud. Alcohol* 42: 1030-1037, 1981.
- KALANT, H., LEBLANC, A. E., WILSON, A. AND HOMATIDIS, S. Sensorimotor and physiological effects of various alcoholic beverages. *Can. Med. Ass. J.* 112: 953-958, 1975.
- KATKIN, E. S., HAYES, W. N., TEGER, A. I. AND PRUITT, D. G. Effects of alcoholic beverages differing in congener content on psychomotor tasks and risk taking. *Q. J. Stud. Alcohol, Supplement No. 5*, pp. 101-114, 1970.
- LANG, A. R., GOECKNER, D. J., ADESSO, V. J. AND MARLATT, G. A. Effects of alcohol on aggression in male social drinkers. *J. Abnorm. Psychol.* 84: 508-518, 1975.
- LANG, A. R., KAAS, L. AND BARNES, P. The beverage type stereotype: An unexplored determinant of the effects of alcohol consumption. *Bull. Soc. Psychologists Addict. Behav.* 2: 46-49, 1983.
- PERNANEN, K. Alcohol and crimes of violence. In: KISSIN, B. AND BEGLEITER, H. (Eds.) *The Biology of Alcoholism*, Vol. 4, Social Aspects of Alcoholism, New York: Plenum Press, 1976, pp. 351-444.
- PIHL, R. O., ZEICHNER, A., NIAURA, R., NAGY, K. AND ZACCHIA, C. Attribution and alcohol mediated aggression. *J. Abnorm. Psychol.* 90: 468-475, 1981.
- SOUTHWICK, L., STEELE, C., MARLATT, A. AND LINDELL, M. Alcohol-related expectancies: Defined by phase of intoxication and drinking experience. *J. Consult. Clin. Psychol.* 49: 713-721, 1981.
- TAKALA, M., PIHKANEN, T. A. AND MARKKANEN, T. The Effects of Distilled and Brewed Beverages: A Physiological, Neurological and Psychological Study. Finnish Foundation for Alcohol Studies, Publ. No. 4, Helsinki, 1957.
- TAYLOR, S. P. AND GAMMON, C. B. Effects of type and dose of alcohol on human physical aggression. *J. Personality Soc. Psychol.* 32: 169-175, 1975.
- WIKLER, A. Dynamics of drug dependence: Implications of a conditioning theory for research and treatment. *Archs Gen. Psychiat.* 28: 611-616, 1973.
- ZEICHNER, A. AND PIHL, R. O. Effects of alcohol and behavior contingencies on human aggression. *J. Abnorm. Psychol.* 88: 153-160, 1979.
- ZEICHNER, A. AND PIHL, R. O. Effects of alcohol and instigator intent on human aggression. *J. Stud. Alcohol* 41: 265-276, 1980.

"Serious Drinking," "White Man's Drinking" and "Teetotaling": Drinking Levels and Styles in an Urban American Indian Population*

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ABSTRACT. The differences between abstainers, moderate drinkers and heavy drinkers were examined in American Indians living in Los Angeles, California. Equal numbers of these three groups (total $N = 155$) were selected from four tribal groups: Siouan-speaking, Navaho, Five Civilized Tribes (of eastern Oklahoma origin) and indigenous California tribes. The relative predictive powers of sociostructural, cultural and psychological variables in accounting for current drinking levels were then assessed. The results indicated that, much as for non-Indian populations, heavy drinkers were more likely to have had heavy-drinking models in the

family of origin, to be men and to score high on psychophysiological stress indices. Socioeconomic status and traditionalism were found to be weaker predictors of drinking level. Differences in drinking styles over individuals' lifetimes and between tribes were also studied. Ethnographic observations, case vignettes and statistical summaries of the sample by tribe and by drinking level showed that tribal origins, age and socioeconomic status influenced drinking style and attitudes toward alcohol, even if they did not predict the current drinking level of the subjects. (*J. Stud. Alcohol* 45: 237-250, 1984)

AMERICAN INDIANS have a higher alcohol consumption than other ethnic groups or subgroups in the United States. Arrest statistics, random-sample surveys, national task force reports, professional and social-service-agency epidemiological studies, ethnographic monographs and Indians' own identification of drinking as their leading health problem all support this observation (Burns et al., 1974; Federal Bureau of Investigation, 1971; Ferguson, 1968; Graves, 1971; Indian Health Service, 1970; Kunitz et al., 1971; Reasons, 1972; Shore, 1974; Slater and Albrecht, 1972; Stewart, 1964; Suarez, 1971).

Indians have also suffered more cultural and economic upheaval than any other ethnic minority in this country. Indians who lost their traditional subsistence base because of successive waves of European immigration suffered prolonged, profound and systematic exploitation (Cook, 1976; Deloria, 1969; Fritz, 1963; Hamer, 1965; Harmon, 1941; Jorgenson, 1971; Kinney, 1937; Officer, 1971; Spicer, 1969). Several authors

have tried to account for differences in drinking levels between Indians and other ethnic groups (Bacon et al., 1965; Ewing et al., 1974; Farris and Jones, 1978; Fenna et al., 1971; Horton, 1943; Wolff, 1972; Zeiner et al., 1977) or differences in drinking levels among Indian tribal communities (Kunitz and Levy, 1974; Leland, 1981; Levy and Kunitz, 1974; Price, 1975; Stratton et al., 1978; Westermeyer, 1974) by referring to these profound social changes.

Given the high rates of alcohol consumption and related sequelae in Indians as a group, it is often overlooked that substantial numbers of them do not drink at all or drink in moderation. How do these Indians differ from the heavier drinkers? We are not asking why one tribe drinks more than another or why Indians as a group drink more than non-Indians but rather what characterizes intratribal differences in drinking levels. Are there common social or demographic characteristics, for example, among abstainers from Sioux, Navaho, California and Eastern Oklahoma regions? Do the antecedents of moderate drinking among the Sioux or Navaho differ from those among the Eastern Oklahoma and California groups? One goal of the present study is to compare several antecedent variables, in addition to tribal affiliation, for their ability to predict drinking levels within these four tribal groups.

We also explore the antecedent factors that differentiate Indians who had drunk heavily in the past but

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were abstinent at the time of interview from those who had never drunk at all or had drunk only briefly and experimentally long before. Our results indicate that predictors for classification into one of these groups are somewhat different from predictors for classification as either a current moderate or heavy drinker. Differences in the drinking pattern of the family of origin and tribal affiliation play a larger role in predicting classification for these currently abstaining subsets.

The second section of this article considers the drinking styles and life histories of the participants. Although tribal affiliation, socioeconomic status and degree of traditionalism were unrelated to current drinking levels, each of these variables did influence the ways, places and stages of life in which Indians chose to drink.

Method

Sample design

Our research design differed somewhat from most drinking-pattern studies in that we did not do a single-culture ethnography or generate a random sample to examine prevalence rates. Instead, we systematically and nonrandomly selected approximately equal numbers of heavy and moderate drinkers, and nondrinkers from each of the four most heavily represented tribal groups in Los Angeles: Sioux (including 11 individuals from other Siouan-speaking tribes—Winnebago and Omaha); Navaho; indigenous California tribal groups; and the "Five Civilized Tribes" of Eastern Oklahoma—Cherokee, Creek, Chickasaw, Choctaw and Seminole. All participants lived in urban areas of Los Angeles and Riverside counties, California, at the time of the interview. The sampling design and number of subjects in each cell are shown in Table 1.

The abstainer category included both lifetime abstainers and subjects who had consumed alcoholic beverages at moderate to heavy levels in the past but who, when interviewed, reported total abstinence for at least the previous 6 months. There was considerable variation across tribal groups in the ratio of lifetime abstainers to former drinkers. Slightly more than one-half of the Navaho ($N = 7$) and Eastern Oklahoma ($N = 8$) abstainers were former drinkers vs all of the Sioux ($N = 16$) and indigenous California ($N = 7$) abstainers. Of the 50 abstainers, 38 were former heavy drinkers. The sex of the subjects differed by drinking

category: 52% of the abstainers, 23% of the moderate drinkers and 71% of the heavy drinkers were men. Men comprised 60% of the Navaho and California subsamples and about 40% of the Siouan and Eastern Oklahoma tribal groups.

The sample was selected by means of network and snowball recruitment strategies. We contacted Indians who were known to Weibel from her previous work in the community (1978, 1981) or to members of our Indian field staff as exemplars of certain drinking levels. We initiated a wide range of social network nodal points and never interviewed more than three people in one network. This sampling strategy minimized the error of oversampling a few highly accessible but nonrepresentative "cliques." As indicated above, we were unable to locate the desired number of participants in some categories (for example, lifetime abstainers among the Siouan-speaking and California groups). Our ethnographic observations tend to support our impression that problems in obtaining equal cell sizes reflect real prevalence trends in these tribal groups. The sampling procedures have been discussed elsewhere (Weibel, 1978; WEIBEL, J. C. AND WEISNER, T. S. An Ethnography of Urban Indian Drinking Patterns in California. Unpublished ms., UCLA Alc. Res. Center, 1980; WEIBEL, J. C., WEISNER, T. S. AND LONG, J. Socioeconomic, Cultural and Psychological Influences on Drinking Levels in a Selected Sample of American Indians in Los Angeles: An Annual Statistical Analyses Report. Unpublished ms., UCLA Alc. Res. Center, 1980; WEIBEL, J. C., WEISNER, T. S. AND LONG, J. Socioeconomic, Cultural and Psychological Influences on Drinking Levels in Rural Indigenous California Indian Populations: A Statistical Analysis of Interview Data. Unpublished ms., UCLA Alc. Res. Center, 1981; WEIBEL, J. C., WEISNER, T. S., NORTHRUP, E. AND LONG, J. An Ethnography of Rural California Indian Drinking Practices. Prepared for the California State Office of Alcohol and Drug Abuse. Unpublished ms., UCLA Alc. Res. Center, 1981).

Measures of drinking level

The participants' self-reports of their quantity and frequency of alcohol consumption were used to classify their drinking in two ways: (1) a three-level categorization of abstainers, light to moderate drinkers and heavy drinkers; and (2) a six-point scale combining quantity and frequency. Both classification systems were based on quantity and frequency scales developed by Cahalan et al. (1969). The quantity scale was scored 0–4, the lowest score indicating no consumption of alcoholic beverages and the highest designating five or more drinks per occasion. The frequency scale was a 10-point measure ranging from abstinence at the lowest score to drinking an average of three or more times daily.

We used a three-level classification of current and former drinking levels for cross-tabulations with other characteristics of the sample. The descriptive profiles in this article are drawn principally from such cross-tabulations. Participants who were not currently drinking were included in the abstainer category. Those who drank no more often than twice weekly and consumed no more than four drinks on each occasion were classified as moderate drinkers. The

TABLE 1. Distribution of subjects by drinking level

Tribal group	Heavy drinkers	Moderate drinkers	Abstainers	Total
Navaho	10 (17%)	14 (30%)	13 (26%)	37 (24%)
Sioux	21 (36%)	6 (13%)	16 (32%)	43 (28%)
E. Oklahoma	12 (21%)	13 (28%)	14 (28%)	39 (25%)
California	15 (26%)	14 (30%)	7 (14%)	36 (23%)
Total	58 (100%)	47 (100%)	50 (100%)	155 (100%)

category, heavy drinkers, consisted of participants who had three or four drinks more than twice per week or five or more drinks per occasion.

The second, six-point measure of present and past drinking was constructed for use in the correlations and multiple regressions. To obtain this score we recoded the Cahalan et al. (1969) measures of drinking frequency and amount consumed per drinking occasion and summed the recoded scores. Each scale was recoded to a 4-point measure (0-3), with a score of 0 representing no drinking, 1 indicating up to two drinks per occasion and 3 designating five drinks or more per occasion. The recoded frequency measure ranged from once or twice per week (a score of 1) to daily or more often (a score of 3). When the two measures were combined into one, the range of scores was from 2 (up to two drinks, as often as twice weekly) to 6 (daily consumption of five or more drinks). Abstainers' scores of 0 were recoded to 1 to produce a 6-point measure. This procedure was employed for current drinking and, among the current abstainers, for previous drinking also.

The reliability of participants' self-reports on drinking was verified in several ways. First, we examined the internal consistency of subjects' comments about their drinking behavior in the interviews. When discrepancies with their initial self-reported drinking levels occurred, we elected to use the higher frequency and amount reported on both scales.

Second, we examined the external validity of these reports through ethnographic observations of interviewees in public drinking and nondrinking settings throughout Los Angeles. Of the 155 Indian participants interviewed in Los Angeles, Weibel (1982) observed 90 at one or more public events that Indians attend regularly. Of those 90 participants, 60% were observed drinking at a level that they had identified as their modal drinking level; 30% reported drinking heavily or moderately but when observed drank either moderately or not at all. Only 10% appeared to be drinking more heavily in public than they had self-reported. This subgroup consisted of nine subjects: two chronically heavy drinkers who were abstaining and in treatment at the time of the interview but had reverted to heavy alcohol use by the end of the fieldwork period and seven subjects who had described their drinking as "periodic" or "moderate" but who, when observed, appeared to be intoxicated and unable to maintain socially appropriate comportment. From these observations, we believe that our classification of the participants' current drinking levels at the time of the interview was broadly accurate and may have even slightly overestimated drinking levels in public places.

The theoretical model: Predictors of drinking level differences

Research on the origins of drinking patterns among Indians (as well as other ethnic groups) can be divided into four broad theoretical categories: sociocultural, cultural, psychological and genetic-physiological. Our selection of antecedent or predictor variables includes sociostructural, cultural, and psychological ones. We did not collect data that could be used to test genetic-physiological hypotheses. The small

number of studies focusing on genetic-physiological issues among Indians (Bacon et al., 1965; Ewing et al., 1974; Farris and Jones, 1978; Fenna et al., 1971; Wolff, 1972; Zeiner et al., 1977) mandates future expert, well-designed and procedurally sound analysis of the association between genetic predisposition, drinking level, and the behavioral and physical sequelae of drinking.

Sociostructural predictors of drinking level. Sociostructural theories of problem drinking have focused on the history of economic and political oppression among Indians on reservations and among those who have migrated to urban areas (Fenna et al., 1971; Graves, 1970; Hamer, 1965; Heath, 1981; Jorgenson, 1971; Officer, 1971). It is presumed that, like all other members of the lower classes and working poor in the U.S., Indians are exploited and suffer all the indignities of lowered esteem, insecurity and economic powerlessness. Unemployed men and women are more likely to drink and to get into trouble when they do drink (Cahalan and Cisin, 1976). Thus, Indians are thought to drink more than other ethnic groups because they are the quintessential victims of "externally induced cultural stress," as one Indian author phrased it (Deloria, 1969).

To assess whether Indians of a lower socioeconomic status drink more or with a different style than Indians who are economically better off, we analyzed the relationship between the respondents' occupational status, income, years of formal education, and drinking level. Age and sex were also included as sociostructural factors.

Cultural and historical predictors of drinking level. Among contemporary Indian societies, cultural differences persist in languages, ceremonial traditions, folk medicines, world view and, there is some evidence to suggest, drinking styles (Burns et al., 1974; Foreman, 1934; Grobsmith, 1981; Heizer and Whipple, 1967; Kluckhohn and Leighton, 1962; Kroeber, 1953; Kunitz and Levy, 1974; Kunitz et al., 1971; Leland, 1981; Price, 1975; Stratton and Zeiner, 1977; Stratton et al., 1978; Wax, 1971; Westermeyer, 1974).

Levy and Kunitz (1974) have suggested that loosely organized Indian societies, in which vision quests and the use of hallucinogenic drugs are integral to spiritual belief and ceremonial systems, will have institutionalized mechanisms for drinking. In these societies, patterned behavior, such as public intoxication, will not be negatively sanctioned. Drinking until psychic release is achieved will be the goal of drinking bouts (Mohatt, 1972). The Navaho and Sioux are the tribal groups in our study that closely fit this cultural model. The very public and "ecstatic" character of Navaho and Sioux drinking, however, is considered to be deviant behavior in non-Indian urban settings and often results in negative social sanctioning (Ferguson, 1968; Graves, 1970; Hurt and Brown, 1965; Maynard, 1969; Mohatt, 1972; Whittaker, 1963).

Levy and Kunitz (1974) also suggest that highly structured societies place more negative sanctions on drinking. In these societies, drinking is less public and more covert, involves more feelings of guilt and shame about excessive drinking

and is accompanied by adverse medical sequelae. The Five Civilized Tribes from Eastern Oklahoma are examples of more highly structured tribal groups (Foreman, 1934; Kunitz et al., 1971; Kunitz and Levy, 1974; Stratton and Zeiner, 1977; Weibel, 1978).

Whittaker (1963) hypothesized that high rates of drunkenness on the Standing Rock Sioux Reservation were attributable to poor living conditions, the breakup of Sioux culture, repressed aggression and the lack of social sanctions against drinking. Maynard (1969) saw problem drinking among Oglala Sioux as a means of dealing with acculturation stress. Kuttner and Lorincz (1967) found that feelings of group inferiority, anxiety over substance, acculturation stress, hostility or submissiveness to White authority, and frustration of ambition were neither characteristic of Sioux drinkers nor primary motivations for drinking. Rather, weakened family structures and familial predispositions to support and sustain drinking have been suggested (Hurt and Brown, 1965) as antecedent factors of Sioux drinking patterns.

Stratton et al. (1978), in their studies of alcohol-related problems within the diverse Oklahoma Indian populations, also noted the predominance of alcohol-related problems among Indians of the Great Plains. The Cheyenne and Arapaho of Western Oklahoma suffer a significantly higher mortality from alcohol-related causes than do eastern Oklahoma Indians (predominantly Cherokee, Cree, Choctaw, Chickasaw and Seminole). Stratton et al. suggested that profound social and historical differences between the two groups accounted for the differences in sequelae of excessive alcohol consumption. The eastern tribes were primarily horticulturalists whereas the western tribes were nomadic hunters. The eastern tribes were introduced to alcoholic beverages at least 100 yr before the western tribes. Also, the eastern tribes had used fermented drinks for ceremonial purposes before European contact. They, therefore, may have had a cultural disposition to use alcohol in more restricted and ritualized ways and may have had longer to assimilate the more secular use of alcoholic beverages into their own traditional use of ceremonial intoxicants. The influence of fundamentalist Protestant churches and their heavily negative sanctions on drinking may also account for the relatively rare incidence of alcohol-related problems among "Bible Belt" eastern Oklahoma Indians.

Unlike Plains, eastern Oklahoma and southwestern tribal groups, there are few ethnographic descriptions of California Indian drinking practices. Bushnell (1971) described the devastating effects of excessive alcohol consumption among the Hoopa Valley Indians and Colegrove (1977) documented the early onset of drinking and drug use in the same group. Fieldwork observations (WEIBEL, J. C., WEISNER, T. S., NORTHROP, E. AND LONG, J. An Ethnography of Rural California Indian Drinking Practices. Prepared for the California State Office of Alcohol and Drug Abuse. Unpublished ms., UCLA Alc. Res. Center, 1981) suggest that the binge drinking style exhibited by many Hoopa Valley men is modeled after the lumberjack, frontiersman, hard-working and hard-drinking ethos characteristic of that area during the initial period of contact with Europeans.

Burns et al.'s (1974) random sample of Los Angeles Indian drinking suggests some correlation between type of tribal

organization and drinking level. Sioux respondents drank more and were less likely to be abstainers than other tribal groups. The Navahos drank less than other tribal groups; Navaho women, in particular, had a very high rate of abstinence (56%) and were least likely to be current or past heavy drinkers. The eastern Oklahoma Indians also reported less heavy drinking than other tribal groups.

Our measures of cultural antecedents of drinking behavior included tribal affiliation, percentage of Indian ancestry, marriage endogamy, and the degree of adherence to traditional Indian customs such as participation in religious ceremonies and powwows, use of American Indian medicines and healing practices, and use or knowledge of the tribal language.

Psychological predictors of drinking level. Psychological factors designated (Brod, 1975; Bryde, 1971; Graves, 1970; Jessor et al., 1968; Jilek, 1974; Lemert, 1956; Littman, 1970; Shore, 1974; WEIBEL, J. C., WEISNER, T. S. AND LONG, J. Socioeconomic, Cultural and Psychological Influences on Drinking Levels in a Selected Sample of American Indians in Los Angeles: An Annual Statistical Analyses Report. Unpublished ms., UCLA Alc. Res. Center, 1980; WEIBEL, J. C., WEISNER, T. S. AND LONG, J. Socioeconomic, Cultural and Psychological Influences on Drinking Levels in Rural Indigenous California Indian Populations: A Statistical Analysis of Interview Data. Unpublished ms., UCLA Alc. Res. Center, 1981) as causes of heavy drinking include intrapsychic stress, psychodynamic conditions in personal history, and role modeling in the family of origin or procreation. Our measures of psychological antecedents included both the amount and types of drinking behavior models (e.g., peer binge drinking, heavy parental drinking, or abstinence in the home) to which the participants had been exposed during childhood. We also asked each informant about his personal psychophysiological stress level, as measured by the 50-item short form of the Cornell Medical Index (LEIGHTON, A. H. AND KENNEDY, D. A. Pilot Study of Cultural Items in Medical Diagnosis: A Field Report. Prepared for the U.S. Public Health Service, Division of Indian Health. Unpublished ms., Cornell University, 1957), a self-report inventory.

Results

Demographic profile

Participants had to be at least one-quarter Indian to be included in the study; 64.5% of the sample claimed full Indian ancestry. The subjects had a median age of 35.5 and were evenly divided by sex (78 men and 77 women). The median length of residence of immigrants in the city was 12.5 yr at the time of the interview; 10% of the participants had been born in Los Angeles. A total of 37% were currently married. Of those who were married or living with an unmarried mate, three out of four had an Indian mate. Over one-half of the sample had at least some postsecondary education but one-fourth had not completed high school. Of the

sample 37% were unemployed, and another 47% were unskilled laborers or semi-skilled and skilled technicians; 16% were employed in a professional, administrative or managerial capacity.

On variables such as length of time in Los Angeles, sex ratio, degree of Indian ancestry and drinking-level distribution, our sample closely paralleled the demographic profile of the Los Angeles Indians in Burns et al.'s (1974) random sample ($N = 512$). However, our sample was slightly older, had more education and included fewer unemployed. There was also evidence of less family stability in our sample (fewer marriages, and more divorces and separations). Since our sample was interviewed in 1978 and 1979, the demographic differences may reflect socioeconomic trends (inflation, a stable but aging urban Indian population and selective migration) in the Los Angeles Indian population between 1973 when Burns et al. collected their data and the time of our field work.

Because of our sampling strategy we reached fewer "invisible" Indians—those who live and work in Los Angeles without any regular participation in Indian activities. Therefore, the tendency to maintain and exhibit one's Indian identity was probably higher in our sample than it would have been in a random sample of the Los Angeles Indian population.

Demographic characteristics differed widely across the tribal groups in this study. Large majorities of Navahos and eastern Oklahoma Indians, for example, claimed full Indian ancestry but slightly less than one-half of the Sioux and less than one-fifth of the California Indians did. Mean ages across the tribal groups ranged from 30.5 for the Navahos to 41.6 for the Five Civilized Tribes. In two groups (the Navaho and California), women outnumbered men by a ratio of 3:2; the ratio was reversed in the other two groups. Most of the participants from the Five Civilized Tribes had migrated to Los Angeles before 1956, whereas most Navahos and Sioux came there in the mid-1960s. All but three of the California Indians had been born in the Los Angeles area. Marital patterns in the tribal groups were associated with age. One-half of the participants from the Five Civilized Tribes were married; the largest concentration of never-married participants (41.7%) was among the Navahos. In contrast, there was little tribal variability in education, job and income level.

Predictors of drinking level differences

Tribal differences as interaction effects. Before we could assess the relative impact of each variable and each class of variables on drinking level in our sample, we needed to account for the possible interaction effects of tribal background with our set of selected

antecedent factors. By design, our sample included fairly equal numbers of men and women at the three drinking levels in each of the four tribal groups. Tribal affiliation thus acted as a covariate for the other antecedent factors, not as an antecedent factor. Given this condition, we first performed an analysis of covariance to search for interactions between tribal group and our other independent variables.

The results indicated no consistent interaction effects between our set of antecedent variables and tribal status in their prediction of drinking level. There were also no significant correlations among the antecedent variables used in the regression analysis of the entire sample. This finding does not mean that tribal and cultural factors do not affect drinking level among Indians. On the contrary, tribal affiliation caused a major difference in prevalence rates, drinking styles and drinking levels among subsets of our population. The covariance analysis indicated no statistically significant interaction effects between tribal affiliation and other antecedent variables (degree of Indian ancestry, age, income, sex, stress level, etc.) that we used to predict current drinking levels in this sample. Thus, it was not necessary to control for tribal group in our subsequent analysis.

The entire sample. To determine the primary predictors of drinking level in our sample, we performed a series of stepwise multiple-regression analyses using the antecedent variables discussed above as independent predictor variables and the measure of current drinking frequency and amount as the dependent variable. One useful feature of multiple-regression analysis is that it yields an indication of the proportion of variance accounted for in the relationships tested. In our series of analyses, several variables did not increase the explained variance in drinking levels more than 1% and were thus successively eliminated from the analysis. These variables included: socioeconomic status (a compound measure of education, earned income and occupational status), degree of traditional Indian behavior (fluency in the tribal language, attendance at Indian rites and ceremonies and the use of traditional medical practices), exposure to tribal culture (years on a reservation and proportion of Indian childhood peers), years of residence in Los Angeles and drinking level in the household of origin. All failed to add even 1% to the amount of variance accounted for by the regression. Again, as with tribe as a covariate, these results do not indicate that these variables are not important influences on drinking behavior. In fact, some of them will reemerge in our analyses of drinking styles and in the studies of subsets of our population. The variables that remained as predictors of drinking level are shown in Table 2.

TABLE 2. Stepwise multiple regression of drinking level with selected predictor variables ($N = 142$)^a

	Multiple r	r^2	Simple r
Year of birth	.271	.073	.271
Cornell Medical Index score	.340	.116	.218
Sex (1 = male, 2 = female)	.382	.146	-.182
Proportion of Indian ancestry ^b	.407	.166	.203

^aThirteen cases were eliminated from this analysis because of missing data on the Cornell Medical Index.

^bIn Tables 2 and 3, the proportion of Indian ancestry was recoded so that full-blooded Indians (among whom drinking level is lowest) would have the lowest score on this measure. This recoding was done to illustrate more clearly the curvilinear relationship between this variable and drinking level.

Four variables accounted for 17% of the variance in this linear model: younger and middle-aged participants drank more than older ones; those with higher self-reported psychophysiological stress scores drank more than those with lower stress scores; men drank more heavily than women; and those who reported 50% Indian ancestry drank more than either those with 25% Indian ancestry (the minimum level for inclusion in our study) or more than 75% Indian ancestry.

Our findings are similar to those of a 1979 national survey of drinking patterns (Clark et al., 1981) with regard to the association of sex and age with drinking level. Our findings also correspond to the averaged results of six earlier national surveys as reported by Johnson et al. (1977). None of these surveys employed the Cornell Medical Index and, of course, our measure of Indian ancestry was not relevant. However, the indices of ethnicity and psychology that we found to be associated with drinking level were similarly identified in the 1979 national survey. We concur with Cahalan and Cisin (1976) that "ethnic background still plays an important part in determining patterns of American life, including drinking habits" (p. 87). Binge drinking is an expected feature of young male recreational behavior in many contemporary Indian communities, whereas heavy negative sanctions are placed on community elders who continue to drink "like young men." In this respect, too, more drinking by young Indian men appears to intensify existing American patterns.

Current drinking levels, excluding Indians "on the wagon." The terms "serious drinking," "White man's drinking," "teetotaling" and "on the wagon" are used indigenously to denote differences in both drinking style and level. Indians with a history of heavy

drinking who were abstinent at the time of the interview were clearly a different subset from those who had never drunk or those who reported current drinking. Indians on the wagon (abstinent at least 6 months at the time of the interview) were largely older men (between 45 and 60). Their drinking careers shared these features: they started drinking experimentally in their mid-teens; acceleration of consumption at 18 to 20 yr of age was often associated with an armed services drinking milieu; and drinking often accelerated to family-, job- and life-threatening proportions until middle age. Their conversions to lives of abstinence were often supported by renewed belief in traditional Indian or Christian spiritual dogma, membership in Alcoholics Anonymous, or involvement in an organized recovery home or treatment program.

In Table 3, we repeat the stepwise multiple-regression analysis illustrated in Table 2 but exclude these Indians "on the wagon" from the abstainer category. Both the rank order of entry and the relative power of the predictor measures now change. Self-reports of family drinking during childhood are now more strongly related to current drinking levels, and psychophysiological stress increases in predictive power. Age and percentage of Indian ancestry decrease to minor predictive importance. The results are interpreted as follows: Men drink more than women, those who report psychophysiological stress drink more than those who do not and those who report frequent heavy drinking in their families of origin drink more than those who report little or no drinking in their childhood homes. Older men and women drink less than younger ones and subjects reporting 50% Indian ancestry drink more than those with higher or lower percentages, but these predictors are less potent than when applied to the entire sample. Notwithstanding the smaller size of this subsample ($N = 116$), this analysis considerably improves the predictive power of the equation. The overall multiple correlation reaches .593, with over one-third of the variance in reported drinking level accounted for. Once again, factors

TABLE 3. Stepwise multiple regression of drinking level with selected predictor variables, excluding current abstainers who previously drank ($N = 106$)

	Multiple r	r^2	Simple r
Sex (1 = male, 2 = female)	.466	.218	-.466
Cornell Medical Index score	.528	.279	.331
Drinking in the household of origin	.570	.325	.277
Year of birth	.583	.340	.119
Proportion of Indian ancestry	.593	.352	.218

related to socioeconomic status are not important predictors of drinking level in this population.

The older men on the wagon appear to have produced most of the age effect for the entire sample. Indeed, when we subsequently regrouped these men with current heavy drinkers and reconducted our analyses, there was an interesting result: Neither age nor proportion of Indian ancestry appreciably affected drinking level once the effects of sex, stress level and drinking in the family of origin were accounted for.

Comparison of lifetime abstainers (teetotalers) and those currently on the wagon. Finally, we compared the two subpopulations exhibiting the most dramatic differences in lifetime drinking levels and styles. These two subgroups are especially interesting for both statistical and qualitative analyses, since they may provide clues to the similarities and differences in two major pathways to Indian sobriety: teetotaling and middle-age abstinence. We used the same multiple-regression techniques and variables employed in the previous analyses to find the major quantifiable features that differentiate the two kinds of abstinence. The multiple r is now .75 (Table 4). More than 54% of the variance in drinking levels was accounted for by these three variables (in descending order of influence): Indians who, as children, experienced heavy drinking in their own families are more likely to drink heavily as adults, after controlling for other circumstances; men drink more than women; and highly stressed individuals drink more than those reporting less stress. After the effects of these three variables were accounted for, age and proportion of Indian ancestry added only 1% each to the equation. Socioeconomic status, traditionalism and even age dropped out in favor of family experience, gender and stress effects.

Discussion

We have used drinking level as a dependent measure to be explained by quantitative analyses. However, this does not imply that the predictor variables that

we selected are causally antecedent or statistically independent. The relationships between drinking level and many of the antecedent variables are not unidirectional. For example, moderate and heavy drinkers may be under more actual environmental stress, and so report more psychic stress, than abstainers or light drinkers. Similarly, the physiological stress may be the result rather than the precipitating cause of chronic drinking. The stress measure, thus, is not necessarily an independent cause of drinking level; the two variables are likely confounded.

We do not believe that there is a confounding effect in the correlation between adult drinking levels and drinking in the family of origin. For example, we do not believe that heavy drinkers systematically report more drinking in the family of origin than in fact occurred or that abstainers cover up childhood remembrances of drinking in their families. We resort to the candidness of life history narratives for supportive evidence (pseudonyms are used):

Mary Smith, lifetime abstainer—"The only person I knew who drank at all was my grandfather and he only drank occasionally. I was always taught not to use it. And I taught that to my children. You see, my family was Christian." (Mary stopped at this point and gave the interviewer a knowing look as though identifying herself and her family as Christian were sufficient explanation for her lifetime abstinence from intoxicants.)

Dwayne Rochambeau, recovering alcoholic—"My family drank periodically. When they had enough money to buy it, they bought it—every 6 weeks or so. It took away the boredom of life, the frustrated feelings of not having a job. They thought of it as medicine. All of them would drink to excess when they could afford to buy it. They lost jobs, cars, husbands and wives because of it. They drank all the time in the house—sometimes for 3 or 4 days at a time. Lots of times we kids were fearful, but what could we do? They were the bosses; we had to go along with whatever they did."

We interpret drinking behavior modeled by the family of origin as socialization and role modeling that produce an adult drinker. It is also possible, however, that family drinking indicates an Indian genetic propensity for heavy drinking (Schuckit, 1980; Schuckit et al., 1972). Our data do not suggest this. If men and women with higher proportions of Indian ancestry also reported more drinking in the family of origin, then it might be plausible that some Indian genetic or physiological propensity to consume more alcohol or to react differently to alcohol consumption is confounded with reports of a family drinking model. However, our data indicate that participants with 50% Indian ancestry (a foot in each world, so to speak) drink more than those with either less or more Indian ancestry. The curvilinear

TABLE 4. Stepwise multiple regression of former drinking level with selected predictor variables, for current abstainers only ($N = 46$)

	Multiple r	r^2	Simple r
Drinking in the household of origin	.650	.423	.650
Sex (1 = male, 2 = female)	.719	.516	-.541
Cornell Medical Index score	.738	.544	.213
Year of birth	.747	.558	-.022
Proportion of Indian ancestry	.753	.567	.178

ear relationship between proportion of Indian ancestry and drinking level is more suggestive of an acculturation stress than a direct genetic effect. Of course individual family differences in genetic predisposition cannot be ruled out by our findings.

Furthermore, the proportion of Indian ancestry was confounded with age and sex. A majority of the heavy drinkers were not only of mixed parentage but also young and male. These men may exhibit normative drinking behavior for their age and social group, and they also deal with the stresses of obtaining an education in a novel urban environment, providing for their young families in a competitive employment market, and being separated from family and community norms. The decision to go to the city to "better oneself" is not without its ambivalences. "Getting a White man's education," and equipping oneself to "deal with the White man on his own terms" or even "to beat him at his own game" are encouraged by some as a survival strategy for contemporary Indians, but are also feared by both family and friends as "selling out" or the loss of one's "Indianness." Many Indian youths eventually reject this coping strategy and either return home or remain in the city and turn to heavy drinking and lives of marginality. The weekend drinking party with other urban Indians in the same circumstances can mitigate psychic stresses temporarily, as well as reestablish connectedness to Indian society and values (Burns et al., 1974; Lurie, 1979; Waddell, 1973; WEIBEL, J. C. AND WEISNER, T. S. An Ethnography of Urban Indian Drinking Patterns in California. Unpublished ms., UCLA Alc. Res. Center, 1980).

Although we have included both drinking models in the family of origin and level of stress under the heading of psychological factors, we do not mean to imply that stress (as measured by the Cornell Medical Index) is purely an intrapsychic phenomenon or that family drinking models represent only individualized socialization factors. On the contrary, as Stratton et al. (1978) and Levy and Kunitz (1974) have suggested, stress and family modeling effects are embedded in an environmental, social and cultural context that includes characteristics of reservation life, Christian influences and the degree of community affluence.

It is also evident that the Indian sample parallels findings (Clark et al., 1981) on drinking in the general American population: Drinking is heavier among younger than among older people; men in every ethnic group drink more at each stage of life than women; and the drinking pattern modeled in the family of origin is reproduced, to some degree, in the next generation. Drinking level and style may be unique in some respects among Indians and influenced by cultural heritage but the overall differences in drinking

levels and the life-time drinking patterns parallel the general drinking patterns in our society as a whole.

Profiles of drinking styles

Heretofore, we have discussed antecedents or predictors of drinking level. Drinking level is not synonymous with drinking style nor is it to be confused with prevalence levels. Unlike our findings on drinking levels, drinking styles and prevalence levels have been shown (Bacon et al., 1965; Burns et al., 1974; Kunitz et al., 1971; Stratton et al., 1978) to be affected by socioeconomic status, degree of Indian identity and tribal affiliation. By drinking style we mean not only quantifiable frequency, amount and variability of beverages consumed, but also the entire sociobehavioral matrix in which the drinking takes place. Drinking style takes into account where, with whom, and on what occasions one drinks, drinking patterns over time, the reasons for drinking and the types of behavior associated with drinking.

To elaborate on these features of drinking behavior, we turn to our ethnographic data elicited in life history interviews and observations of participants. Choosing participants who exemplify specific drinking styles or regional modalities, we reconstructed descriptive vignettes of their lives to illustrate characteristics of theirs that are representative of either their drinking style or cultural modality.

These data deal with the questions: Which Indians have successfully remained nondrinkers, or lifelong moderate drinkers? Are there similarities in their social histories? What are their present circumstances? And how might others do the same, given their personal and drinking history profiles? This ethnographic perspective complements the quantitative results derived from the analysis of current drinking levels. Age, sex, family drinking models during childhood and percentage of Indian ancestry (but not socioeconomic status or tribe) influence current drinking levels, but the same features may not be associated with longer-term histories of abstinence or heavy drinking, or with the quality of life and social supports that sustain different drinking patterns.

The following vignettes combine (1) qualitative data or portraits of characteristic drinking styles and (2) statistical and demographic summary profiles of informants who tended to cluster within each drinking style.

Teetotalers. There are two dramatically different kinds of urban Indian nondrinkers: lifetime teetotalers and former drinkers who are now on the wagon. The first group is composed primarily of older women of middle-level socioeconomic status who have never drunk or

who have drunk only briefly and experimentally during childhood. Lifetime abstainers tend to come from eastern Oklahoma and to have long-standing Indian Christian church affiliations. Mary Smith, a full-blooded Choctaw, exemplifies the lifetime non-drinking profile:

Mary Smith is 67 and speaks fluent Choctaw. She "never drank an alcoholic beverage" in her life. Her early socialization included strong prohibitions against alcohol consumption. Recently retired from her long-time employment as a commercial seamstress, her social life is centered on her family and church. Three of her four children live within a 5-mile radius of her comfortable home, which she owns. She attends church services, conferences and social affairs two or three times a week. Her involvement in urban Indian activities includes visits to other all-Indian Christian churches. Out of curiosity she attended a few Plains-type urban powwows. Her antipathy toward drinking is so complete that she refused to attend future powwows because of the drinking that she observed in those settings (minimal though it was). Her recommended solution to problem drinking among urban Indians is that they surrender to Christ as their personal savior.

The second group of current nondrinkers is more likely to be composed of older men from lower and lower-middle socioeconomic backgrounds who have had periods of heavy and problem drinking throughout their lives. These men are more likely to come from California and Siouan-speaking tribes, and are less likely to be married or to have had any formal church affiliations:

Dwayne Rochambeau is a 40-yr-old Sioux and Chippewa man whose alcoholism (he asserts) destroyed his family life—his wife died and his child was made a ward of the state. A product of an unstable home, he lived with his parents on a reservation until he was 12, when he moved to a midwestern city to live with an older sister. Dwayne began drinking at age 15. At the encouragement of an older sister (aged 19), already an alcoholic, he drank whisky until he passed out in the bathroom. After a short period of revulsion and abstinence he began to drink with the rest of his family in earnest. He drank moderately until he entered the Navy at age 20. During 3 yr in the Navy he drank excessively. After a dishonorable discharge due to infractions of Navy rules caused by his drinking, he abstained for approximately 1 yr. This was followed by approximately 15 yr of steadily increasing alcohol consumption, during which he quickly gravitated to the Skid Row street-drinking lifestyle. He finally entered an all-Indian alcoholism treatment program at the insistence of a physician who diagnosed liver cirrhosis. At that time the doctor estimated that Dwayne would survive only another year if he did not stop drinking. Through counseling received in the alcoholism program and deep involvement in A.A., Dwayne had maintained his sobriety for 4 yr at the time of the interview. His social life

consists of regular attendance at A.A. meetings and participation in Plains and pan-Indian activities in urban Indian centers. He is enrolled in an alcoholism counseling training program and interacts regularly with a circle of abstaining friends. His conversion to an abstinent lifestyle is perpetuated by his decision to present himself as a role model for his recovering Indian brothers.

Dwayne epitomizes successful recovery. He has been able to sustain his abstinence for an extended period of time. He is deeply committed to his chosen new lifestyle gained through a personally wrenching process not unlike spiritual conversion. He supports his belief in abstinence through involvement in A.A. and volunteer work in alcoholism treatment programs.

Other men and women in our sample who have had success in maintaining abstinence have employed similar support structures. Involvement in Indian alcoholism treatment programs as both patients and counselors, membership in an Indian Christian church, or A.A., and a reintensification of one's traditional belief system are among the most commonly mentioned vehicles for recovery and maintenance of abstinence.

Unfortunately, the majority of heavy drinkers who had attempted to curtail their drinking through similar means had returned, at the time of the interview—or eventually returned, subsequent to the interview—to heavy drinking. The most familiar heavy-drinking pattern includes periods of heavy drinking interrupted forcibly by incarceration or commitment to an alcohol or drug treatment program, an ambivalent "dry out" period, which is quickly aborted through abandonment of the treatment regimen, and a return to heavy drinking.¹

White man's drinking. Subjects who drank "like White men" tended to be middle-aged or older and from higher socioeconomic levels (more white-collar jobs, higher and more stable incomes and more formal education). Slightly more women than men were in this group, and the men tended to be older than the sample average; however these were not strong differences. They tended to be either full-blooded or one-quarter Indian rather than 50%. In other respects, however, they were not statistically different from the rest of the sample.

The following vignettes illustrate characteristic patterns of Indians who drink in moderation or, in Gusfield et al.'s (1981) terms, "manage competence" while drinking. From an Indian perspective, they drink "like White men":

¹This so-called revolving door phenomenon, which both frustrates and discourages alcoholism treatment personnel, is discussed elsewhere (Weibel-Orlando, in press).

Anna Sawyer, one-quarter Mono Indian from central California and a graduate student, was 29 and single at the time of the interview. When she was growing up she lived with her grandparents about half of the time and with her parents the other half. She never attended an Indian boarding school. She consciously maintains her Indian cultural heritage by acquiring tribal skills in food and clothing preparation, basketry and hunting. She still uses traditional medicines regularly, attends traditional ceremonies, speaks the language fluently and lives by the spiritual tenets of her ancestors. Her father, mother, brothers and sisters, grandparents, and aunts and uncles all drank moderately. There was only one uncle who drank heavily. "My parents greatly influenced how I live my life. I remember my uncle would call when he had been drinking. He'd be feeling so sorry for himself. We all were annoyed with him." Anna was 17 when she had her first drink. She currently drinks only on festive occasions. On rare occasions she drinks half a can of beer. "I'll have a glass of wine with dinner on occasion and a rare mixed drink at a disco or family gathering, but that's about it. I've experimented with marihuana, used uppers during exam time in college, peyote and a rare mind-altering drug, but only once or twice, mainly as an experiment. The drinking I do is mostly for social acceptance and correct social behavior—it's what's accepted in our society, to have a drink with a meal." She never drinks alone and has never "gotten high." "I limit myself to three drinks, as I want to be aware of myself and my environment at all times. I think my philosophical beliefs—those which stem from my people—have a lot to do with my level of drinking. We don't believe in overindulging in anything."

Sally Franklin is a 44-yr-old full-blooded Choctaw married to a 42-yr-old full-blooded Creek. She came to Los Angeles in 1955 and has a stable work record. They live in a comfortable two-bedroom house that they own in a solidly working-class Los Angeles suburb. Sally spent most of her childhood (14 yr) away from her family in Indian boarding schools. She had lived in a predominantly White area in eastern Oklahoma, had never learned any traditional skills or used traditional medicines. She speaks only a few words of her tribal language. She remembers that none of her relatives drank alcoholic beverages. She was 30 yr old and living in Los Angeles when she had her first drink during a celebration luncheon with co-workers, most of whom were non-Indian. She found a mixed drink at lunch relaxing. It made conversation flow more easily. She reports having a cocktail with a meal three or four times a year at most. Her present social circle consists mostly of Indians from the same area in southeastern Oklahoma. Her social activities include seasonal Indian church meetings, Choctaw classes, attendance at professional baseball games and an occasional movie or show. Her husband drinks as moderately as she does. They have never tried mind-altering drugs. Drinking is always associated with special celebrations and has never been a social or health problem for either of them. Sally has never been intoxicated: "I know how to control myself."

Serious drinking. Serious drinkers are younger, more often unmarried and from lower socioeconomic back-

grounds. The majority of subjects with 50% Indian ancestry fell into this category. Heavy drinkers were not likely to be active participants in Indian cultural activities such as powwow dancing, ceremonies, use of traditional medicines or use of tribal languages. Other demographic factors did not strongly differentiate heavy drinkers from the overall sample.

Wes Eagleman is half-Sioux, 22 yr old and single. He grew up in South Dakota exclusively among other Siouan-speaking people. He attended Indian boarding school for 8 yr and has tried his hand at radio communications and college for short periods of time. His Siouan experience includes learning the powwow dances and songs. He speaks Lakota and has practiced traditional Siouan spiritual ways. Although he does not involve himself in these activities in the city, he considers Fort Thompson his home and visits there three or four times a year. He reports that his father does not drink at all. Since his mother no longer lives with them, he does not recall her drinking pattern. Although his father does not drink, he appears to tolerate his son's use of alcoholic beverages: "You can drink beer as long as it doesn't become a problem." Wes had his first drinking experience at age 13 among a group of Mexican American friends in Los Angeles. He became intoxicated from one can of beer and was grounded by his father when discovered. Between the ages of 13 and 18 he drank sporadically at parties and never more than two cans of beer. Between 19 and 21, however, the frequency, amount and variability of his use of mind-altering substances increased: "I sometimes drank as much as two six-packs of beer a night or until I had passed out. I also drink a bottle of wine a night when it's available and I've gotten into weed in the last few years—everybody in my crowd is." When asked why his consumption has increased in the last few years, he mentioned no feelings of depression, anxiety or frustration. He matter-of-factly states: "I like to drink and am of age, so there's no reason not to. We're all partying and having a good time, so it doesn't matter if you get high—that's all part of the fun." Drinking constitutes his preferred recreational activity. Although he has already experienced three blackouts, has on occasion been embroiled in street-fights and arrested, has fought with his family frequently, has lost money while drinking and, he claims, has experienced delirium tremens at least once, he refuses to consider asking for assistance. "I don't need help. I have my drinking pretty well controlled."

Dixon Begay was a 30-yr-old full-blooded Navaho enrolled in an Indian studies program at a Los Angeles area college at the time of the interview. He grew up in an isolated area on the Navaho reservation and knew only Navahos and a few White traders during most of his childhood. He lived with his maternal grandmother most of his early years. He was sent to a variety of government and church-run Indian boarding schools before running away in tenth grade. In the ensuing years he managed to acquire a general education diploma and complete 2 yr of college. He had never lived in any city before his arrival in Los Angeles. He has acquired traditional Navaho skills such as silversmithing and sheepherd-

ing, uses traditional Navaho medicines, attends traditional ceremonies and speaks Navaho fluently. He describes his parents as "straight people." His father is a minister and neither parent drinks at all; nor, says Dixon, did his grandparents. His brother, aunts and uncles, however, drank "quite a bit," to the point of losing some of their sheep for the price of a bottle of wine. His older brother returned from Vietnam when Dixon was 15 and introduced alcoholic beverages to him for the first time. He drank moderately until he entered the army at age 17. During 3 yr of service, "I drank almost every day if I had the money, until I felt comfortable or blacked out. Sometimes I'd have 20 cans of beer in a night. My performance began to fail so my commanding officer finally ordered me to attend A.A. meetings." Dixon still drinks beer, mixed drinks and wine. He has experimented with marihuana only in the last 2 yr and only with his college friends. Dixon migrated to Los Angeles to go to school and to live with his older brother. Within a month after his arrival he was drinking regularly in the Los Angeles Indian bars with Indian friends. During his heaviest drinking period, Dixon drank every day and whatever was available. "I'd drink three or four jugs of wine, two cases of beer a week. Sometimes I'd spend \$20 to \$50 a night on the weekends. I think I drank more than any of the other people with whom I drank but I'm trying to slow down on my drinking." He reports currently drinking only two or three cans of beer almost every day.

Drinking styles and tribal affiliation

As is clear from our selection of vignettes, there is an association between life-long drinking styles and tribal origin. Findings of differences in drinking level by tribe are common in the literature (Burns et al., 1974; Kunitz et al., 1971; Leland, 1976, 1981; Levy and Kunitz, 1974; Stratton and Zeiner, 1977; Stratton et al., 1978; Zeiner et al., 1977; WEIBEL, J. C. AND WEISNER, T. S. *An Ethnography of Urban Indian Drinking Patterns in California*. Unpublished ms., UCLA Alc. Res. Center, 1980) and are clearly reflected in our own sample, even though we preselected our informants to cover a full range of drinking levels. Levy and Kunitz (1974) have pointed out that "Indian societies appear to react [to the contact situation] in ways that are determined by their own culture and values. There is a remarkable persistence of some cultural constellations over time. . . . [which] are of more value in explaining certain kinds of behavior commonly labeled deviant than has been usually thought" (p. 195).

Our ethnographic observations and life-history data also indicate a range of tolerable drunken comportment across tribal and regional groups. Obviously not every Indian from eastern Oklahoma is a teetotaler nor is every Sioux a serious drinker or on the wagon. However, certain demographic trends are apparent

from our sample, as well as certain proscriptive attitudes about alcohol consumption among some groups.

The eastern Oklahoma participants, for example, were older, with longer and more stable endogamous marriages. They were also more likely to be three-quarter to full-blooded Indian. Their socioeconomic levels were higher and were maintained through stable careers. The eastern Oklahoma participants were more often affiliated with fundamentalist Christian churches known for their long histories of prohibitionistic stands on alcohol consumption. This factor strongly influences familial attitudes toward drinking. Binge drinking is not the recreational norm for eastern Oklahoma young men that it is in other tribal groups in rural and reservation settings. The man who goes into town and "drinks up the seed money" is clearly deviant, suspected of "backsliding" and "flirting with the devil," and in need of community prayers and rededication to a "life in Christ." These beliefs, coupled with strong family commitments and the aforementioned cluster of cultural, familial and social characteristics, insulate the eastern Oklahoma youth, to some extent, from participating as heavily as other tribal youth populations in the drinking and drug culture.

Navaho participants were more often of full Indian ancestry, young, single, from low socioeconomic backgrounds and married endogamously. Both Navaho men and women had well-grounded personal experience in traditional tribal folkways and participated in reservation activities more often than the other tribal groups. They drank more heavily and more often than California or Oklahoma groups. Their drinking patterns were similar to those of the Plains groups.

The Siouan-speaking participants were more likely to be of mixed parentage, in tribally and ethnically exogamous marriages, and acculturated. Their reservation experience included extensive drinking during their youth. Heavy alcohol consumption among the Plains Indians generally and the Siouan-speaking people specifically has most commonly been related (Clark et al., 1981; Kuttner and Lorincz, 1967; Maynard, 1969; Mohatt, 1972; Stratton and Zeiner, 1977; Stratton et al., 1978; Whittaker, 1963; Zeiner et al., 1977) to their cultural, historical and psychological characteristics. Their high drinking level has been seen as a reaction to stress that has taken on epidemic proportions. Our ethnographic data (WEIBEL, J. C. AND WEISNER, T. S. *An Ethnography of Urban Indian Drinking Patterns in California*. Unpublished ms., UCLA Alc. Res. Center, 1980) support these findings. The Siouan-speaking participants tended to cluster at the extreme ends of the drinking continuum. The Skid Row and habitually heavy-drinking populations were composed of disproportionately large numbers of

Siouan-speaking Indians. A large majority of the Siouan-speaking participants who were abstinent at the time of the interview had formerly been heavy drinkers. Plains deculturation was rapid, profound and traumatic. With few remaining traditional avenues for validation of adulthood and particularly of manhood, alcohol consumption was and continues to be the salve of wounded warriors as well as the vehicle of protest (Lurie, 1971; Maynard, 1969; Mohatt, 1972).

The California Indians in Los Angeles come from a geographically dispersed collection of tribes and bands. They were more acculturated and reported a lower percentage of Indian ancestry than the other three tribal groups. A high proportion of their marriages were exogamous. Their drinking patterns were intermediate in many ways between those of the Sioux and Navaho on the one hand, and those of the Eastern Oklahoma participants on the other.

Conclusions

Even though the variety of Indian lifestyles and drinking patterns is too great to categorize any group, it is clear from our quantitative analyses and ethnographic reconstructions that sex, age, the models of drinking behavior provided by the family of origin and psychological stress best predict drinking level. However, it is important to note that which types of Indian drinkers (teetotalers vs those on the wagon, for instance) are compared makes a difference in the patterning of the antecedent factors predictive of drinking level. Only the sex difference (men consistently drinking more than women) retains its statistical and substantive importance throughout all the subset analyses.

Family drinking models and psychological stress appear to be the most powerful predictors of drinking level when comparing the extreme drinking patterns (lifetime abstainers vs those on the wagon vs heavy drinkers, for instance). Age and sex had stable effects on drinking levels across the entire sample, regardless of prior family experience or tribal affiliation. Family of origin and perceived stress played important roles in discriminating between heavy-drinking and teetotaling Indians, given the fact that men and youth drink more.

Indians in the teetotaling and moderate-drinking groups were more likely to have reservation experiences such as Christian religious training, to suffer from less economic marginality (especially in the Oklahoma groups) and to have a stake in an urban job. Paradoxically, traditionalism and concern for Indian ancestry and culture usually are associated with reduced drinking levels in conjunction with, not opposed to, upward mobility and Christian religious beliefs.

Acknowledgments

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References

- BACON, M. K., BARRY, H., 3d AND CHILD, I. L. A cross-cultural study of drinking. *II. Relations to other features of culture*. Q. J. Stud. Alcohol, Supplement No. 3, pp. 29-48, 1965.
- BROD, T. M. Alcoholism as a mental health problem of Native Americans: A review of the literature. *Archs Gen. Psychiat.* 32: 1385-1391, 1975.
- BRYDE, J. F. *Modern Indian Psychology*, Vermillion, S.D.: University of South Dakota, 1971.
- BURNS, M., DAILY, J. M. AND MOSKOWITZ, H. Drinking Practices and Problems of Urban American Indians in Los Angeles. *Part I. Study Description and Findings: Preliminary Report*, Santa Monica, Calif.: Planning Analysis and Research Institute, 1974.
- BUSHNELL, J. H. Lives in profile: A longitudinal study of contemporary Hupa men from young adulthood to the middle years. *Trans. N.Y. Acad. Sci.* 32: 787-801, 1971.
- CAHALAN, D. AND CISIN, I. H. Drinking behavior and drinking problems in the United States. In: KISSIN, B. AND BEGLEITER, H. (Eds.) *The Biology of Alcoholism*, Vol. 4, Social Aspects of Alcoholism, New York: Plenum Press, 1976, pp. 77-115.
- CAHALAN, D., CISIN, I. H. AND CROSSLEY, H. M. *American Drinking Practices: A National Study of Drinking Behavior and Attitudes*. Rutgers Center of Alcohol Studies, Monograph No. 6, New Brunswick, N.J., 1969.
- CLARK, W. B., MIDANIK, L. AND KNUFFER, G. Report on the 1979 National Survey, Berkeley, Calif.: Social Research Group, 1981.
- COLEGROVE, W. K. Hupa Alcohol Abuse Survey Program: A Study to Explore the Relationship between Occupational Selection and Usage of Drugs and Alcohol, Eureka, Calif.: Colegrove & Assoc., Inc., 1977.
- COOK, S. F. *The Conflict between the California Indian and White Civilization*, Berkeley, Calif.: University of California Press, 1976.
- DELORIA, V. *Custer Died for Your Sins: An Indian Manifesto*. New York: Macmillan Publishing Co., Inc., 1969.
- EWING, J. A., ROUSE, B. A. AND PELLIZZARI, E. D. Alcohol sensitivity and ethnic background. *Am. J. Psychiat.* 131: 206-210, 1974.
- FARRIS, J. J. AND JONES, B. M. Ethanol metabolism in male American Indians and Whites. *Alcsm. Clin. Exp. Res.* 2: 77-81, 1978.
- FEDERAL BUREAU OF INVESTIGATION. *Uniform Crime Reports for the U.S., 1970*. U.S. Department of Justice, Washington, D.C.: U.S. Government Printing Office, 1971.
- FENNA, D., MIX, L., SCHAEFER, O. AND GILBERT, J. A. L. Ethanol metabolism in various racial groups. *Can. Med. Ass. J.* 105: 472-475, 1971.
- FERGUSON, F. N. Navaho drinking: Some tentative hypotheses. *Hum. Org.* 27: 159-167, 1968.
- FOREMAN, G. *The Five Civilized Tribes*, Norman, Okla.: University of Oklahoma Press, 1934.
- FRIE, H. E. *The Movement for Indian Assimilation: 1860-1890*, Philadelphia: University of Pennsylvania Press, 1963.
- GRAVES, T. D. The personal adjustment of Navajo Indian migrants to Denver, Colorado. *Am. Anthropol.* 72: 35-54, 1970.
- GRAVES, T. D. Drinking and drunkenness among urban Indians. In: WADDELL, J. AND WATSON, M. (Eds.) *The American Indian in Urban Society*, Boston: Little, Brown & Co., 1971, pp. 274-311.

- GROSSMITH, E. S. *Lakota of the Rosebud: A Contemporary Ethnography*. New York: Holt, Rinehart & Winston, 1981.
- GUSFIELD, J. R., KOTARBA, J. AND RASMUSSEN, P. Managing competence: An ethnographic study of drinking-driving and the context of bars. In: HARFORD, T. C. AND GAINES, L. S. (Eds.) *Social Drinking Contexts. Research Monograph No. 7*, Washington, D.C.: U.S. Government Printing Office, 1981, pp. 155-172.
- HAMER, J. H. Acculturation stress and the functions of alcohol among the Forest Potawatomi. *Q. J. Stud. Alcohol* 28: 285-302, 1965.
- HARMON, G. D. *Sixty Years of Indian Affairs*, Chapel Hill, N.C.: University of North Carolina Press, 1941.
- HEATH, D. B. Determining the sociocultural context of alcohol use. *J. Stud. Alcohol, Supplement No. 9*, pp. 9-17, 1981.
- HEIZER, R. F. AND WHIPPLE, M. A. *The California Indians: A Source Book*. Berkeley, Calif.: University of California Press, 1967.
- HORTON, D. The functions of alcohol in primitive societies: A Cross-cultural study. *Q. J. Stud. Alcohol* 4: 199-320, 1943.
- HURT, W. R. AND BROWN, R. M. Social drinking patterns of the Yankton Sioux. *Hum. Org.* 24: 222-230, 1965.
- INDIAN HEALTH SERVICE. *Alcoholism: A High Priority Health Problem*, Section 2, Washington, D.C.: U.S. Public Health Service, 1970.
- JESSOR, R., GRAVES, T. D., HANSON, R. C. AND JESSOR, S. L. *Society, Personality and Deviant Behavior: A Study of a Tri-Ethnic Community*, New York: Holt, Rinehart & Winston, 1968.
- JILEK, W. G. Indian healing power: Indigenous therapeutic practices in the Pacific Northwest. *Psychiat. Ann.* 4 (11): 13-21, 1974.
- JOHNSON, P., ARMOR, D. J., POLICH, S. AND STAMBUL, H. U.S. Adult Drinking Practices: Time Trends, Social Correlates and Sex Roles. Prepared for the U.S. National Institute on Alcohol Abuse and Alcoholism, Publication No. PB294-044/AS, Springfield, Va.: National Technical Information Service, 1977.
- JORGENSEN, J. G. Indian and the metropolis. In: WADDELL, J. AND WATSON, M. (Eds.) *The American Indian in Urban Society*, Boston: Little, Brown & Co., 1971, pp. 66-113.
- KINNEY, J. P. *A Continent Lost—A Civilization Won*, Baltimore, Md.: Johns Hopkins Press, 1937.
- KLUCKHOHN, C. D. AND LEIGHTON, A. H. *The Navajo*, Garden City, N.Y.: Doubleday, 1962.
- KROEBER, A. L. *Handbook of the Indians of California*, Berkeley, Calif.: California Book Co., Inc., 1953.
- KUNITZ, S. J. AND LEVY, J. E. Changing ideas of alcohol use among Navaho Indians. *Q. J. Stud. Alcohol* 35: 243-259, 1974.
- KUNITZ, S. J., LEVY, J. E., ODOROFF, C. L. AND BOLLINGER, J. The epidemiology of alcoholic cirrhosis in two Southwestern Indian tribes. *Q. J. Stud. Alcohol* 32: 706-720, 1971.
- KUTTNER, R. E. AND LORINCZ, A. B. Alcoholism and addiction in urbanized Sioux Indians. *Ment. Hyg.* 51: 530-542, 1967.
- LELAND, J. *Firewater Myths: North American Indian Drinking and Alcohol Addiction*. Rutgers Center of Alcohol Studies Monograph No. 11, New Brunswick, N.J., 1976.
- LELAND, J. The context of Native American drinking: what we know so far. In: HARFORD, T. C. AND GAINES, L. S. *Social Drinking Contexts. NIAAA Research Monograph No. 7*, Washington, D.C.: U.S. Government Printing Office, 1981, pp. 173-205.
- LEMERT, E. M. *Alcoholism: Theory, problem and challenge. III. Alcoholism and the sociocultural situation*. *Q. J. Stud. Alcohol* 17: 306-317, 1956.
- LEVY, J. E. AND KUNITZ, S. J. (Eds.) *Indian Drinking: Navajo Practices and Anglo-American Theories*, New York: John Wiley & Sons, Inc., 1974.
- LITTMAN, G. Alcoholism, illness and social pathology among American Indians in transition. *Am. J. Publ. Hlth.* 60: 1769-1787, 1970.
- LURIE, N. O. The world's oldest on-going protest demonstration: North American Indian drinking patterns. In: MARSHALL, M. (Ed.) *Beliefs, Behaviors, and Alcoholic Beverages: A Cross-Cultural Survey*, Ann Arbor: University of Michigan Press, 1979, pp. 127-145.
- MAYNARD, E. Drinking as part of an adjustment syndrome among the Oglala Sioux. *Pine Ridge Res. Bull.* 9: 35-51, 1969.
- MOHATT, G. The sacred water: The quest for personal power through drinking among the Teton Sioux. In: McCLELLAND, D. C., DAVIS, W. N., KALIN, R. AND WANNER, E. (Eds.) *The Drinking Man: Alcohol and Human Motivation*, New York: Free Press, 1972, pp. 261-275.
- OFFICER, J. The American Indian and federal policy. In: WADDELL, J. AND WATSON, M. (Eds.) *The American Indian in Urban Society*, Boston: Little, Brown, & Co., 1971, pp. 8-65.
- PRICE, J. A. An applied analysis of North American Indian drinking patterns. *Hum. Org.* 34: 17-26, 1975.
- REASONS, C. Crime and the American Indian. In: BAHR, H. M., CHADWICK, B. A. AND DAY, R. C. (Eds.) *Native Americans Today: Sociological Perspectives*, New York: Harper and Row, Pubs, Inc., 1972.
- SCHUCKIT, M. A. Biological markers: Metabolism and acute reactions to alcohol in sons of alcoholics. *Pharmacol. Biochem. Behav.* 13: 9-16, 1980.
- SCHUCKIT, M. A., GOODWIN, D. A. AND WINOKUR, G. A study of alcoholism in half siblings. *Am. J. Psychiat.* 128: 1132-1136, 1972.
- SHORE, J. H. Psychiatric epidemiology among American Indians. *Psychiatric Ann.* 4 (11): 56-57, 61-63, 66, 1974.
- SLATER, A. D. AND ALBRECHT, S. L. The extent and costs of excessive drinking among the Uintah-Oray Indians. In: BAHR, H. M. AND CHADWICK, B. A. (Eds.) *Native Americans Today: Sociological Perspectives*, New York: Harper and Row, Pubs, Inc., 1972, pp. 358-366.
- SPICER, E. H. *A Short History of the Indians of the United States*, New York: Van Nostrand Reinhold Co., 1969.
- STEWART, O. C. Questions regarding American Indian criminality. *Hum. Org.* 23: 61-66, 1964.
- STRATTON, R. AND ZEINER, A. Variations in alcohol problems within the Oklahoma Indian population. *Alc. Tech. Rep.* 6 (No. 1): 5-12, 1977.
- STRATTON, R., ZEINER, A. AND PAREDES, A. Tribal affiliation and prevalence of alcohol problems. *Q. J. Stud. Alcohol* 39: 1166-1177, 1978.
- SUAREZ, E. Testimony of Eugene Suarez. In: *Hearings before a Subcommittee on Department of the Interior and Related Agencies of the Committee on Appropriations. House of Representatives, 92nd Congress, First Session, Part I*. Washington, D.C.: U.S. Government Printing Office, 1971.
- WADDELL, J. O. "Drink, friend!": Social Contexts of Convivial Drinking and Drunkenness among Papago Indians in an Urban Setting. *Proc. 1st Annu. Alcm. Conf. NIAAA*, Washington, D.C., 1973, pp. 237-251.
- WAX, M. L. Contemporary plains reservation communities. In: WAX, M. L. (Ed.) *Indian Americans: Unity and Diversity*, Englewood, N.J.: Prentice-Hall, Inc., 1971, pp. 65-87.
- WEIBEL, J. C. Native Americans in Los Angeles: A cross-cultural comparison of assistance patterns in an urban environment. *Anthropology* 2: 81-98, 1978.
- WEIBEL, J. C. There's a place for everything and everything in its place: Environmental influences of urban Indian drinking patterns. In: HARFORD, T. C. AND GAINES, L. S. (Eds.) *Social Drinking Contexts. NIAAA Research Monograph No. 7*, Washington, D.C.: U.S. Government Printing Office, 1981, pp. 206-227.
- WEIBEL, J. C. American Indians, urbanization and alcohol: A developing urban Indian drinking ethos. In: NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM. *Special Population Issues. Alcohol and Health Monograph No. 4*, Washington, D.C.: U.S. Government Printing Office, 1982, pp. 331-358.

- WEIBEL-ORLANDO, J. C. Indian alcoholism recovery program as flawed rites of passage. *Med. Anthropol. Q.* [In press.]
- WESTERMEYER, J. "The drunken Indian": Myths and realities. *Psychiat. Ann.* 4 (11): 29-31, 35-36, 1974.
- WHITTAKER, J. O. Alcohol and the Standing Rock Sioux Tribe. II. Psychodynamic and cultural factors in drinking. *Q. J. Stud. Alcohol* 24: 80-90, 1963.

WOLFF, P. H. Ethnic differences in alcohol sensitivity. *Science* 175: 449-450, 1972.

ZEINER, A. R., PAREDES, A., MUSICANT, R. H. AND COWDEN, L. Racial differences in psychophysiological responses to ethanol and placebo. In: SEIXAS, F. A. (Ed.) *Currents in Alcoholism*, Vol. 1, Biological, Biochemical and Clinical Studies, New York: Grune & Stratton, 1977, pp. 271-286.

1: *En studie i alternativ nykterhetspolitik*
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The birth of American alcohol control: prohibition, the power elite, and the problem of lawlessness

BY HARRY GENE LEVINE

Harry Levine is Associate Professor in the Department of Sociology at Queens College, City University of New York, Flushing, New York. His previous work with the Alcohol Research Group examined the emergence in the early nineteenth century of modern conceptions about alcohol as an addictive and criminogenic substance. He is currently completing a book on the history of ideas about alcohol problems in America.

Preface

The first two decades of the twentieth century in the United States are commonly referred to as the Progressive era. The name derives, in part, from a political perspective shared by much of the new professional middle class, the old entrepreneurial and small-business middle class, and the new, growing corporate elite. Progressivism declared that local, state,

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and federal governments should regulate, control, and administer economic and social activity so as to make new, industrial, twentieth-century America a more economically prosperous, healthy, and orderly nation. Progressivism signaled the end of laissez-faire as the dominant political vision of American government.

In the early twentieth century, reformers concerned with what the nineteenth century had called the "liquor problem" developed two political and policy programs in accordance with progressivist principles. The first and triumphant approach was prohibitionism. The nineteenth-century temperance movement, which Progressive-era prohibitionism replaced, had been a broad and assimilationist movement to get people to give up alcohol and stay off it. The Progressive-era prohibitionists created a single-issue campaign to pass laws restricting or eliminating the production and sale of alcoholic beverages. Prohibitionists argued that the new needs and conditions of twentieth-century industrial society—efficiency, rationality, order, and progress—required totally banning alcoholic drink.

The second new twentieth-century progressivist approach to the liquor question was alcohol control—sophisticated regulatory systems for the legal sale of alcoholic drink. The advocates of alcohol control shared many of the concerns and assumptions of prohibitionists, but they disagreed on one fundamental point: they did not believe that prohibition could ever be enforced, and they maintained that the ensuing law violation, public disrespect for the law, and corruption of the legal and criminal justice system would be extremely damaging and dangerous. Though proposals for alcohol control systems were formulated by important reformers, professionals, scientists, and representatives of the corporate elite, and were being adopted by some European countries, alcohol control was almost totally ignored in the United States until about the mid-1920s—until national constitutional prohibition proved to be unenforceable.

should regulate, control, and administrative activity so as to make new, by America a more economically orderly nation. Progressivism came to be as the dominant political movement.

In the early twentieth century, reformers concerned with what had called the "liquor problem" developed policy programs in accordance with these ideas. The first and triumphant approach was the nineteenth-century temperance movement.

Progressive-era prohibitionism was part of a larger movement to control and stay off it. The Progressive Era was a single-issue campaign to pass legislation restricting the production and sale of alcoholic drink. Prohibitionists argued that the new twentieth-century industrial society, with its demands for order, and progress—required such control.

The early twentieth-century progressivist approach to alcohol control—sophisticated regulation of the sale of alcoholic drink. The temperance movement shared many of the concerns of prohibitionists, but they disagreed on the means. Temperance advocates did not believe that prohibition was the best solution; they maintained that the ensuing social problems, such as corruption and crime, would be extremely difficult to deal with. Though proposals for alcohol control were advanced by important reformers, they were largely ignored by representatives of the corporate world and by some European countries, until the 1920s—until national prohibition was declared unenforceable.

This article is the second half of a larger work which discusses the political, ideological, and policy concerns of prohibitionists and of proponents of alcohol control policy in the United States from the turn of the century to the mid-1930s. The main points made in the first half of that paper can be summarized as follows.

1. In the early twentieth century, an upper-class-sponsored policy organization called the Committee of Fifty to Investigate the Liquor Problem had published five books and had articulated a new, distinct twentieth-century perspective on the liquor question. The Committee of Fifty argued that in most of the United States, prohibition could never be enforced. They proposed, instead, flexibly designed and administered governmental programs to reshape and reorganize public drinking and to eliminate the saloon from working-class life. Their ideas were almost totally ignored from 1900 to 1920 (Levine 1983).

2. Prohibitionism triumphed partly because it successfully persuaded many middle-class Americans that banning alcohol would eliminate or significantly reduce the major social problems in the country. The temperance movement in the nineteenth century had based its appeals on the concerns of the old middle class: protecting the family, aiding personal success, reducing or eliminating crime and poverty. The twentieth-century prohibitionists added a new series of concerns—prohibition was justified in terms of the needs of a new, complex, heterogeneous, class-stratified, industrial, efficiency-oriented society. In 1919, on the day that national constitutional prohibition went into effect, the famous preacher, Billy Sunday, repeated before 10,000 people and a radio audience the central fantasy of the temperance and prohibition campaigns: "The reign of tears is over. The slums will soon be a memory. We will turn our prisons into factories and our jails into storehouses and corncribs. Men will walk upright now, women will smile and the children will laugh.

Hell will be forever for rent" (quoted in Kobler 1973, p. 12). In short, prohibition was offered as a panacea for real social and economic problems.

Prohibitionism also triumphed for organizational and political reasons. The twentieth-century prohibition campaign was led by one organization—the Anti-Saloon League of America—the prototype of the modern political pressure organization. The Anti-Saloon League had paid organizers, backed candidates of either party who would vote as they wished, and drafted laws for their legislators to pass. The Anti-Saloon League successfully mobilized Protestant churches and, most significantly, received substantial support from businessmen and especially from the corporate elite. Although big business only turned strongly to prohibition after about 1913, its intervention made a decisive difference. If substantial elements of the corporate elite had opposed constitutional prohibition, it probably would never have passed. As the historian, James Timberlake (1970, p. 80), has noted, without the support of business, "national prohibition could never have attained such power and sweep." Shortly after ratification, the Reverend Harry Emerson Fosdick, pastor of the prestigious and wealthy Riverside Church in New York City, observed that prohibitionists "could never have put the law on the statute books had not the business motive become involved." "One of the basic facts necessary to understand the prohibitory campaign," he said, "is that American business found it impossible to run modern machines with drink-befuddled brains. . . . Canny, shrewd, businesslike America, knew that it would be a good financial bargain" (Timberlake p. 81).

3. After the Eighteenth Amendment to the Constitution and its enabling legislation, the Volstead Act, went into effect in 1919, the major utilitarian or instrumentalist criticism of prohibition was it was unenforceable. Tens of millions of people did not respect alcohol prohibition, refused to obey it, and there was in truth nothing effective that the federal or

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state governments could do to force obedience. Juries would often not convict bootleggers. Even if a huge force of federal agents had been hired, there never could have been enough to police every border, city, and town. And the agents could never have been paid so much that it would not have been profitable for them to look the other way at the crucial moment. All major cities in the United States, and a great many small cities and towns, flagrantly violated prohibition—drinking places were open all day and all night, drinking was common, and alcohol was plentiful.

4. Critics of prohibition argued that because prohibition was unenforceable it produced two extremely bad results: first, prohibition was an enormous financial drain in lost tax revenues; and second, mass violations of prohibition engendered widespread disrespect for all law—a condition usually termed "lawlessness." These two issues, the loss of tax revenues and lawlessness were the major ones in undermining support for prohibition among the corporate and political elite as well as among the middle class. The following discussion focuses on the issue of lawlessness, partly because lawlessness has usually been ignored or misinterpreted. But, more importantly, the problem of mounting disrespect for law was very much on the minds of those who proposed and designed the systems of alcohol control which replaced prohibition in 1933.¹

5. During the 1920s, law enforcement, law obedience, and lawlessness were the major preoccupations of prohibitionists. They held conferences, enlisted the support of politicians including all three U.S. presidents, and argued that because prohibition was part of the Constitution, the most sacred law in the land, national survival required it be obeyed. The increasing disrespect for all law resulting from disrespect for prohibition law, they warned, could lead to anarchy and communism. Many prominent officials also warned about the consequences of lawlessness. For example, in 1928 Her-

bert Hoover made the issue of lawlessness central in his campaign for president. He promised to appoint a commission—a "National Commission on Law Observance and Enforcement"—to fully investigate prohibition. In his inaugural speech Hoover declared: "Our whole system of self-government will crumble either if officials elect what laws they will enforce or if citizens elect what laws they will support. The worst evil of disregard for some law is that it destroys respect for all law." In July 1929, on the eve of the Great Depression, one news magazine (*Review of Reviews*, p. 64) summarized Hoover's position, noting that "He has sought to impress on the nation that disobedience of the law is the most malign danger that faces the United States today."

6. Just as one organization, the Anti-Saloon League, had dominated the making of prohibition, so did only one organization—the Association Against the Prohibition Amendment (the AAPA)—dominate the making of repeal. Not usually understood is that the AAPA was led, organized, and financed by some of the wealthiest and most conservative men in America. Headed by Pierre du Pont of du Pont Chemicals, and John J. Raskob of General Motors, the board of directors of the AAPA included the presidents and chairmen of the boards of many major corporations. It was commonly referred to at the time as a committee of "millionaires." There are two main reasons these men led the campaign for repeal. The original reason was economic: they believed that if liquor taxes were restored, their business and personal income taxes would be significantly reduced. A second reason the corporate rich turned against prohibition was their growing fear that disrespect for prohibition was producing widespread disrespect for all law, including property law. First pushed by prohibitionists, the argument that the widespread violation of prohibition law would lead to disrespect for all law was gradually adopted by those opposed to prohibition. By the end of the 1920s it had become part of the argument for modification of the Volstead Act, and for repeal.

ue of lawlessness central in his promised to appoint a commission on Law Observance and investigate prohibition. In his inaugural address, he declared: "Our whole system of government is based on the principle that either if officials elect what laws they will enforce or if citizens elect what laws they will disregard for some law is that it is not law." In July 1929, on the eve of the publication of the magazine (*Review of Reviews*, p. 10), in that position, noting that "He has shown that disobedience of the law is the greatest danger that faces the United States today."

the Anti-Saloon League, had been against prohibition, so did only one organization, the National Association Against the Prohibition of Liquor, dominate the making of repeal. The AAPA was led, organized, and financed by the wealthiest and most conservative members of the business community. By Pierre du Pont of du Pont, the son of General Motors, the AAPA included the presidents and many major corporations. It was at first a committee of "millionaires." The main reasons these men led the fight against prohibition was economic: they feared that if prohibition were restored, their business and income would be significantly reduced. A movement which turned against prohibition was based on disrespect for prohibition was disrespect for all law, including prohibitionists, the argument that prohibition law would lead to the eventual adoption of those opposed to prohibition. By the end of the 1920s it had become a justification of the Volstead Act,

I. The collapse of prohibition

By 1929, prohibition was becoming undone. Its unenforceability had become a national scandal. William Randolph Hearst, who had editorially crusaded for passage of the Eighteenth Amendment and who had supported prohibition, changed his mind by the late 1920s. He sponsored an essay contest on the question of what could be done. The prize of \$25,000 was awarded to a New York judge who proposed modifying the Volstead Act to allow sale of beer and wine. Hearst came out for complete repeal shortly thereafter. In April 1929, in his newspapers around the country, and then in a book (Tietz 1929), Hearst denounced Hoover and prohibition. "Of course, law enforcement machinery should be respected," he said. "Of course there should be respect for law in the abstract." But, he declared, "first there should be laws which deserve respect."

Hearst's defection from prohibition marked an important turning point: the argument that prohibition undermined respect for law had now fully become part of the argument for repeal. During the prosperous and relatively calm period of the 1920s, the threat of wider breakdowns of order, and of rebellion and revolt, was as Hearst said an "abstract" or theoretical issue. With the coming of the depression, however, it became a much more concrete problem.

During the 1920s, when prohibitionists warned about the consequences of widespread lack of respect for law, they would sometimes explicitly mention (and often hint about) Communists, anarchists, and industrial workers. During the first two decades of the twentieth century, socialists and radicals had become active in American politics and in union organizing; a number of them were jailed for opposing U.S. participation in World War I. The Russian Revolution, a wave of bombings in 1919, and a number of militant strikes, including a police strike in Boston (successfully broken by then-Governor Calvin Coolidge), further stimulated upper-

and middle-class fears of working-class revolt. However, for most of the 1920s, the danger of revolt was more hypothetical than real. The Great Depression made communism, working-class revolt, and mass disobedience to law relevant and significant issues again.

The Great
Depression:
real law-
lessness
comes to
America

Though the depression and its effects on mass consciousness and politics were not usually mentioned directly in discussions and debate about prohibition and alcohol policy, the political context of the depression provided the unavoidable backdrop to all concerns about alcohol policy. It is important, therefore, to briefly review the depression and its effects on American society to understand the full concerns of politicians, policy makers, and the corporate rich as they decided on repealing prohibition and on the policies which would replace prohibition.

From 1930 on, destitution, impoverishment, and homelessness were no longer marginal phenomena but inescapable and terrible facts of life for millions. In *The Perils of Prosperity 1914-32*, William Leuchtenburg (1958) described some of the consequences of what he called the "Smashup."

A year after the crash, six million men walked the streets looking for work. By 1932, there were 666,000 jobless in Chicago, a million in New York City. . . . In Cleveland 50 percent were jobless, in Akron 60 percent, in Toledo 80 percent. In Donora, Pennsylvania, only 277 of 13,900 workers held regular jobs. In the three years after the crash, 100,000 workers were fired on the average every week (p. 247).

In the 1920s wealthy businessmen had often claimed credit for the prosperity. Now they were blamed for the depression. "Many Americans who had never had a 'radical' thought before in their lives" came to question and distrust the entire capitalist system. The head of the Farmers' Union of Wisconsin told a Senate committee:

They are just ready to do anything. . . . The farmer is naturally a conservative individual, but you cannot find a conservative farmer today. . . . I am as conservative as any man could be, but any

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economic system that has in its power to set me and my wife in the
streets at my age—what can I see but red (Leuchtenburg 1958, p.
262).

The breakdown of order and the possibility of revolt were no
longer theoretical issues. Leuchtenburg points out that "as
the bread lines lengthened the mood of the country became
uglier," and "for the first time in history Lloyd's of London
sold large sums of 'riot and civil commotion insurance' to
Americans."

In July, 1931, 300 unemployed men stormed the food shops of
Henryetta, Oklahoma. An army of 15,000 pickets marched on
Taylorville, Illinois, and stopped operations at the Christian
County Mines in 1932. In Washington, D.C., 3,000 Communist
"hunger marchers" paraded From Bucks County, Pennsyl-
vania, to Antelope County, Nebraska, farmers banded together to
prevent banks and insurance companies from foreclosing
mortgages. When sheriffs attempted to carry out foreclosures,
mobs of farmers, brandishing pitchforks and dangling hangman's
nooses, persuaded the sheriffs to retreat. . . . In a national radio
broadcast, John A. Simpson, president of the National Farmers'
Union, denounced the wealthy as "cannibals that eat each other
and who live on the labor of the workers" (pp. 261-62).

By 1932 the United States was in the midst of an epidemic of
"lawlessness" that seemed like the nightmares of the 1920s
come true. With more than 15 million men out of work,
riots, revolts, and the use of the armed forces to control
hungry and angry men and women were becoming common
(Leuchtenburg 1958, p. 32; Manchester 1974, p. 10). In
Manchester's words, "the sound of famished men on the
march was heard from coast to coast."

In New York, thirty-five thousand men and women packed Union
Square to hear Communist party orators. Crowds in Oklahoma
City, Minneapolis, and St. Paul broke into groceries and meat
markets to rifle shelves. . . . In Lincoln, Nebraska, four thousand
men occupied the State House, another five thousand took over
Seattle's ten story County-City building, and five thousand Chi-
cago teachers, tormented beyond endurance, stormed the city's
banks. The sense of institutions, authority, and private property
. . . was showing signs of disintegration. . . . [There was] scat-

tered aimless rioting in Detroit, where relief had simply stopped . . . Here and there the starving were muttering violence. The mayor of a Massachusetts town, watching two thousand idle men milling around his city hall, wrote that "a spark might change them into a mob." Governor O. Max Gardner of North Carolina warned of the danger of "violent social and political revolution." Mayor Anton Cermak of Chicago, faced with the state's reluctance to appropriate funds for the city's six hundred thousand out of work men, told the legislature, "Call out the troops before you close the relief stations" (Manchester 1974, pp. 10-11).

The possibility that American citizens might take not only the law into their own hands, but also property and government, was not lost on the upper class. As Manchester put it, "the well-fed were edgy." Henry Ford, for example, "had always been a pacifist. Now he carried a gun." In some cities, businessmen formed committees to take control "should railroad and telephone lines be cut and surrounding highways blocked." New York hotels found that "wealthy guests who usually leased suites for the winter were holing up in their country homes." Some had machine guns mounted on their roofs. A number of observers at the time concluded that the fears of the wealthy were justified—revolution seemed a real possibility. The dean of the Harvard Business School, for example, declared that "Capitalism is on trial and on the issue of this trial may depend the whole future of Western civilization." The imminence of revolt was also debated by articles in *Yale Review*, *Scribner's*, *Harper's*, and *Atlantic* (Manchester 1974, p. 66).

The collapse
of upper-class
support for
prohibition

The depression had two major consequences for the debate about prohibition and repeal. Both were crucial in undermining support for the Eighteenth Amendment. First, the depression destroyed any credibility for the long-standing prohibitionist claim that prohibition brought prosperity, and it fueled the new fantasy that repeal would end the depression by putting men back to work by stimulating the economy. In fact, the same kind of panacea-seeking that had long been a

Detroit, where relief had simply the starving were muttering violence. In town, watching two thousand idle men, wrote that "a spark might change all." O. Max Gardner of North Carolina wrote of a "silent social and political revolution." In 1930, faced with the state's reluctance to send out its six hundred thousand out of state troops, "Call out the troops before you are too late" (Sinclair 1974, pp. 10-11).

In some cities, citizens might take not only the property and government, but also the class. As Manchester put it, "the Ford, for example, 'had always carried a gun.'" In some cities, citizens to take control "should be cut and surrounding highways found that 'wealthy guests who winter were holing up in their machine guns mounted on their roofs at the time concluded that the time had come—revolution seemed a real possibility."

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or consequences for the debate. Both were crucial in undermining the Eighteenth Amendment. First, the debility for the long-standing prohibition brought prosperity, and repeal would end the depression by stimulating the economy. In the meantime, a seeking that had long been a

part of prohibitionist arguments now became part of the repeal case. Congressman Linthicum, for example, claimed that with prohibition repealed, the "depression will fade away like the mists before the noonday sun" (quoted in Sinclair 1964, p. 398).²

Second, the depression intensified anxieties about prohibition undermining the legitimacy of all law. Speakers and writers now also linked discontent about prohibition with the rise of radicalism. In 1930, Congressman Robert Clancy warned the House Judiciary Committee that "this is a rather dangerous time, with the people in the cities, which hold 60 percent of the population, in great discontent because of prohibition, and also in great discontent because of the employment situation." Clancy reported that a few days before in Cleveland a mob of unemployed had attacked city hall and battled police. He said that "red" workers, socialists, and Communists were "taking advantage of the unemployment situation" in Cleveland and Detroit. Clancy warned that this was extremely dangerous,

because in my city 80 percent of the population are dissatisfied with the government because of its stand on the dry question, and that is true . . . as you know, in the large cities of the United States, and there is also quite a bit of discontent in the rural communities (quoted in Englemann 1979, p. 187).

In 1931, Matthew Woll, vice-president of the American Federation of Labor (AFL) and the sole labor member of the AAPA board, told the Wickersham Commission that workers were losing faith in the government's willingness to help them, and that prohibition was causing them to further distrust and resent government.

By 1932 a number of leaders and commentators were privately concluding, and some were publicly stating, that legalized beer would make workers feel better about the government and take their minds off their troubles. One AFL leader was quoted to Senate hearings in January 1932 as

saying: "Beer would have a decidedly soothing tendency on the present day mental attitude of the working men. . . . It would do a great deal to change their mental attitude on economic conditions" (quoted in Gordon 1943, p. 104). And by the end of that year, Walter Lippman argued that "Beer would be a great help in fighting off the mental depression which afflicts great multitudes" (quoted in Gordon 1943, p. 104). At Senate hearings on prohibition in 1932, Matthew Woll again blamed the growth of radicalism in the United States on prohibition, and former Michigan Governor Fred Green argued that the survival of the American government depended on immediate repeal (Englemann 1979, p. 187).

The enormous discontent engendered by the depression shaped the political environment in which decisions about repeal and about alcohol policy were debated and ultimately decided. Some speakers and writers explicitly mentioned the riots and marches making headline news. Most others just assumed them. No one wanted to say too often or too explicitly that legalizing alcohol would reduce discontent, but clearly many people thought it.

*The Wickersham
Commission*

The report of Hoover's National Commission on Law Observance and Enforcement played a significant role in the collapse of both elite support and public legitimacy of prohibition. Headed by George Wickersham, the U.S. Attorney General under William Taft, the commission was composed of trustworthy establishment representatives including "a former Secretary of War, a former state Chief Justice, a Circuit Judge, two District Judges, three practicing lawyers, the Dean of the Harvard Law School and the President of Radcliff" (Sinclair 1964). On January 20, 1931, Hoover released the report, announcing with approval that "the commission, by a large majority, does not favor the repeal of the Eighteenth Amendment. . . . I am in accord with this view." A two-page summary of conclusions signed by ten of the eleven commissioners did, in fact, oppose repeal and

a decidedly soothing tendency on the attitude of the working men. . . . It is to change their mental attitude on prohibition (quoted in Gordon 1943, p. 104). And Walter Lippman argued that "Beer is fighting off the mental depression induced by prohibition" (quoted in Gordon 1943, p. 104). In 1932, Matthew Josephson, a former Michigan Governor and a vocal opponent of the American government, argued for the repeal (Englemann 1979, p. 187).

It was engendered by the depression and the environment in which decisions about public policy were debated and ultimately made. Writers explicitly mentioned the headline news. Most others just wanted to say too often or too often that alcohol would reduce discontent, but not at it.

National Commission on Law Enforcement played a significant role in the support and public legitimacy of prohibition. George Wickersham, the U.S. Attorney General, Taft, the commission was composed of representatives including a former state Chief Justice, a Judge, three practicing lawyers, a law school and the President of the American Bar Association. On January 20, 1931, Hoover announced with approval that "the majority, does not favor the repeal of prohibition. . . . I am in accord with this majority of conclusions signed by ten of the members, in fact, oppose repeal and

oppose legalizing beer and wine, or creating government beer monopolies. However, the report's conclusions also said that some commissioners "are not convinced that prohibition under the Eighteenth Amendment is enforceable."

In contradiction to the two-page summary, the report found widespread disobedience to prohibition and seemed to conclude that national prohibition could never be enforced. In their personal statements attached to the report, most of the commissioners also expressed little hope that the Eighteenth Amendment could be enforced. Two of them wanted immediate repeal and return of liquor control to the states. Four favored a government monopoly based on the Swedish example, as proposed by Henry Anderson. Two more favored Anderson's plan if further enforcement attempts failed. George Wickersham wanted the Eighteenth Amendment resubmitted, and if repealed he favored the Anderson plan. Only one commissioner seemed to still support prohibition in the hope that public support could eventually be generated.

The Wickersham Commission report was immediately attacked by newspapers and critics, who pointed out the contradictions between the facts described in the body of the report and the summary which Hoover signed. Walter Lippman charged that Hoover and the commission had evaded "a direct, explicit official confession that federal prohibition is a hopeless failure" (quoted in Kyvig 1979a, p. 114). A columnist in the *New York World* wrote an often-quoted satirical poem called "The Wickersham Report," summing up a common view of the report's conclusions:

Prohibition is an awful flop.
We like it.
It can't stop what it's meant to stop.
We like it.
It's left a trail of graft and slime
It don't prohibit worth a dime
It's filled our land with vice and crime,
Nevertheless, we're for it. (Quoted in Sinclair 1964, p. 336.)

The text of the commission's report focused on the issue of law obedience and enforcement, and it was indeed very critical of prohibition. The commission warned of "the injury to our legal and political institutions" resulting from disobedience to prohibition. Arguing that it is "axiomatic" that "a law will be observed and may be enforced only where and to the extent that it reflects or is an expression of the general opinion of the normally law-abiding elements of the community," the commission concluded: "It is therefore a serious impairment of the legal order to have a national law upon the books theoretically governing the whole land, and announcing a policy for the whole land which public opinion in many important centers will not enforce" (p. 49).

The commission also warned that the "widespread and scarcely or not at all concealed contempt for the policy of the National Prohibition Act" was having a serious, detrimental effect on the attitude and consciousness of the working class. Under the heading "other sources of resentment and irritation," the commission pointed out explicitly the class character of the resentment produced by prohibition:

In the nature of things it is easier to shut up the open drinking places and stop the sale of beer, which was drunk chiefly by working men, than to prevent the wealthy from having and using liquor in their homes and clubs. Naturally . . . laboring men resent the insistence of employers who drink that their employees be kept from temptation. Thus the law may be made to appear as aimed at and enforced against the insignificant while the wealthy enjoy immunity. This feeling is reinforced when it is seen that the wealthy are generally able to procure pure liquors, while those with less means may run the risk of poisoning. Moreover, searches of homes, especially under state laws, have necessarily seemed to bear more upon people of moderate means than upon those of wealth or influence (pp. 54-55).

In their individual statements, some of the commissioners expressed concern about the consequences if this condition should continue. The strongest personal statement on this point was from William S. Kenyon, who worried about the

report focused on the issue of enforcement, and it was indeed very serious. The commission warned of "the social institutions" resulting from prohibition. Arguing that it is "axiomatic" that law may be enforced only where there is respect or is an expression of the moral law-abiding elements of the community, it concluded: "It is therefore a necessary condition to have a national law governing the whole land, and the whole land which public opinion will not enforce" (p. 49).

He noted that the "widespread and deep contempt for the policy of the government is having a serious, detrimental effect on the consciousness of the working class. Sources of resentment and irritation are being brought out explicitly the class characterized by prohibition:

It is easier to shut up the open drinking establishment, which was drunk chiefly by the wealthy from having and using money. Naturally . . . laboring men and women who drink that their employees are breaking the law may be made to appear as insignificant while the wealthy are reinforced when it is seen that the law is pure liquor, while those with money are poisoning. Moreover, searches of the poor, have necessarily seemed to bear more heavily upon those of wealth or

And, some of the commissioners noted the consequences if this condition continued. In a personal statement on this subject, Kenyon, who worried about the

effect on the general public of disobedience by "the 'upper crust' of society, meaning that portion of the very rich people of the nation constituting so-called fashionable society." Kenyon said:

It has been frankly stated before our commission that many of these people of great wealth and prominence will not obey the prohibition laws, do not intend to, and boast of that fact that they will not. . . . If that is to be the standard of law observance, our government will fail. The forger and the bank robber; the highwayman and the embezzler, do not believe in laws that restrain them. There is no more reason why what is termed the "upper crust" of society should choose the laws they will obey than the same privilege should extend to the "under crust" (p. 132).

In the midst of the depression, Kenyon explicitly reminded the wealthy that they need law obedience more than most people: "This government will continue to be a government of law" he said, "or it will cease to be a government at all." He continued: "The day may come in this country when representatives of great property interests will realize they need protection of the law for the properties they represent more than other people need it" (pp. 132-33). A year and a half later, in the worst year of the depression, as a tent city was created in Washington, D.C., by World War I veterans called the Bonus Marchers who were demanding their long-overdue bonus payment, and as scattered riots occurred throughout the country, one prominent member of the upper crust finally concluded that enough was enough.

*Rockefeller's
announcement*

On June 7, 1932, John D. Rockefeller Jr. made headline news across the country. On the eve of the Republican convention, Rockefeller released to the press a letter he had sent to Nicholas Murray Butler supporting repeal of the Eighteenth Amendment. "I was born a teetotaler," wrote Rockefeller. "All my life I have been a teetotaler on principle. Neither my father nor his father ever tasted a drop of intoxicating liquor, nor have I." He also repeated the well-known fact that "with my father, I for years supported the Anti-Saloon League in both state and national work," contributing \$350,000 from

1900 to the passage of the Eighteenth Amendment in 1919. He did not say, mainly because the extent of his contributions had become so controversial, that he and his father had contributed an additional \$487,000 between 1919 and 1929 (Memo to R. Fosdick, June 4, 1932, Box 52, Rockefeller Archives).³ After all these years of support, Rockefeller said that he had "slowly and reluctantly come to believe" that the benefits brought by prohibition "are more than outweighed by the evils that have developed and flourished since its adoption." Prohibition, therefore, should now be repealed.

Rockefeller's announcement was a bombshell. *The New York Times*, in one report from Washington, D.C., said that it "caused the greatest political sensation in the capital in years" (June 7, 1932, p. 1). Another *Times* article identified it "as perhaps the most dramatic single event bearing on the liquor question since the adoption of prohibition" (p. 12). Other stories reported "Drys are Resentful; Wet Chiefs Jubilant." The *Times* reported that "The wet forces generally accepted the conversion of Mr. Rockefeller as the greatest support that had been given to the repeal movement" (p. 1). *The New York Times*, like many other papers, carried the full text of the letter and for a number of days had major stories discussing reactions and consequences and quoting responses from other newspapers and prominent individuals around the country—most of which were extremely positive. Many people cited his courage and called on others to heed his advice. Governor Franklin D. Roosevelt hailed Rockefeller's statement; William Randolph Hearst declared that "this letter will do more than any document which has appeared in the whole discussion of the prohibition question" (*New York Times*, June 8, 1932, p. 1). In a long article on "The Rise and Fall of Prohibition" in *Current History*, the political scientist Louis Hacker (1932, p. 672) summarized the impact of Rockefeller's announcement:

The statement by John D. Rockefeller Jr., practically on the eve of the meeting of the Republican National Convention appeared exactly at the right psychological moment. . . . There can be no

Eighteenth Amendment in 1919. To use the extent of his contributions as evidence, that he and his father had given \$487,000 between 1919 and 1929. On June 4, 1932, Box 52, Rockefeller wrote to his sons of support, Rockefeller said "I instantly come to believe" that the arguments "are more than outweighed by the evils that have developed and flourished since its enactment, and therefore, should now be repealed."

It was a bombshell. *The New York Times*, Washington, D.C., said that it was a "cal sensation in the capital in 1932." Another *Times* article identified the "historic single event bearing on the question of prohibition" (p. 12). "Wet forces are Resentful; Wet Chiefs declared that 'The wet forces generally regard Mr. Rockefeller as the greatest obstacle to the repeal movement'" (p. 1). "In many other papers, carried the full number of days had major stories and sequels and quoting responses of prominent individuals around the country were extremely positive. Many had called on others to heed his advice. Roosevelt hailed Rockefeller's support. Hearst declared that 'this document which has appeared in the prohibition question' (*New York Times*) a long article on 'The Rise and Fall of Prohibition History, the political scientist' (p. 2) summarized the impact of

Rockefeller Jr., practically on the eve of the National Convention appeared at that moment. . . . There can be no

question of the influence Mr. Rockefeller's declaration had on the American public; that it must have borne real weight with the Republican and Democratic platform makers it is also hard to doubt.

Rockefeller based his case against prohibition on the problem of "lawlessness." He claimed that drinking and drinking places had increased under prohibition, and that temperance had not been furthered. He did not, however, mention the tax revenues to be gained from legalizing liquor, nor the idea that a revived liquor industry would put men back to work and stimulate the economy. For Rockefeller, the central issue was lawlessness. "When the Eighteenth Amendment was passed," he wrote, "I earnestly hoped with a host of advocates of temperance, that it would be generally supported by public opinion." But, he explained:

This has not been the result, but rather . . . that a vast army of lawbreakers has been recruited and financed on a colossal scale; that many of our best citizens, piqued at what they regarded as an infringement of their private rights, have openly and unabashedly disregarded the Eighteenth Amendment; that as an inevitable result respect for all law has greatly lessened; that crime has increased to an unprecedented degree—I have slowly and reluctantly come to believe.

Rockefeller noted that he was not "unmindful" of the "benefits that have resulted" from prohibition. But, he warned darkly: "It is my profound conviction, however, that these benefits . . . are more than outweighed by the evils that have developed and flourished since its adoption, evils which unless promptly checked are likely to lead to conditions unspeakably worse than those which prevailed before." Although he did not discuss any alcohol control policies, he implied their importance by noting that repeal of the Eighteenth Amendment will not by itself "end all these evils and restore public respect for law." However, he concluded, "repeal is a prerequisite to the attainment of that goal."

The main thrust of Rockefeller's argument about lawlessness was well understood. *The New York Times*, under the heading "For Obedience to Law," quoted previous statements he had made on the topic. The *Times* linked these previous concerns of Rockefeller's with his announcement, by noting: "In conformity with this view, Mr. Rockefeller in his letter to Doctor Butler declared that while the Eighteenth Amendment remains upon the statute books it should be obeyed" (June 7, 1932, p. 1).

On June 8, the day following Rockefeller's announcement, *The New York Times'* headline read: "Leaders of 2,500,000 Wets Unite to Fight for Repeal; Spurred by Rockefeller." A new organization, The United Repeal Council, with Pierre du Pont as chairman, was created to lead the fight. The *Times* also reported on that day that E. W. Edwards, a Republican delegate and president of "one of the largest banks in the Middle West followed John D. Rockefeller Jr. today in changing his viewpoint on prohibition." Like Rockefeller, Edwards had concluded that "this experiment in moral legislation has done more evil than good" (*New York Times*, June 8, 1932, p. 1). On the day after that, the *Times'* headline announced that Dr. John Mott, the world head of the YMCA, had come out for resubmission of prohibition to the states. Mott said he had made his announcement out of respect to the " 'many good men' like John D. Rockefeller Jr. who have changed their opinions on the issue." Later in June, another long-time prohibitionist, Alfred P. Sloan Jr., the powerful head of General Motors, came out for repeal. "It is my belief," said Sloan, "that our whole social and economic structure will in this manner be greatly strengthened." Sloan was now convinced that "the road toward greater temperance with a resulting better standard of ethics in our country is through repeal" (quoted in Englemann 1979, p. 187).

Repeal In June 1932, a week after Rockefeller's announcement, the Republican party convention adopted what some critics

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called a "wet-moist-dry" plank, favoring resubmission of prohibition to the states and, if repealed, retention of liquor control by the federal government. The Democratic party convention, meeting two weeks later, endorsed repeal and return of liquor control to the states. Franklin Roosevelt's backers had actually fought for a weaker statement, hoping not to alienate some dry supporters. But when Roosevelt made his acceptance speech he endorsed repeal. Roosevelt, however, did not make prohibition a major issue in the election; he campaigned on a relatively conservative program, promising to balance the budget and ensure fiscal integrity. There was during the campaign little hint of the dramatic changes Roosevelt would introduce and the hopefulness he would generate. In 1932 the U.S. was still in the absolute darkness of the depression.

In December 1932 the lame-duck Seventy-second Congress reconvened. Representatives of the AAPA and its women's affiliate, the Women's Organization for National Prohibition Reform (WONPR), visited almost every senator, urging adoption of a repeal resolution. If Congress did not act now, they said, it would be a year until the new Congress convened. Further, over 40 state legislatures were still in session, but most of them would also not meet for a year or more. The leaders of the repeal forces argued that if a repeal resolution was submitted to the states by February, in time for the state legislatures to act, two years could be saved (Kyvig 1979a). On November 16, 1933, the Senate voted to submit the Twenty-first Amendment to state conventions for ratification. The House approved it on February 20.

Just the promise of repeal seemed to take people's minds off the depression. Roosevelt was sworn in as president on March 4, 1933. On March 13, he asked Congress to modify the Volstead Act to legalize 3.2 percent alcohol beer, stressing the need for additional tax revenue. Congress quickly responded and on April 7 beer was legal in most of the country.

"A shiny new beer truck bearing a huge sign, 'President Roosevelt, the first real beer is yours,' delivered two cases to the White House at 12:04 A.M. on the morning of the seventh while a crowd of eight hundred cheered outside the gates." This was a striking contrast to the scene ten months earlier, when the White House gates had been chained shut against the thousands of protesting Bonus Marchers. Now there were celebrations in cities and towns throughout the country. *The New York Times* reported that "Everywhere one went, in hotels, restaurants, clubs, homes, even in some speakeasies, people were drinking the new beer and smiling" (quotes from Kyvig 1979a, p. 177). In the middle of the depression it had begun to seem, as the campaign song had promised, that "Happy Days Are Here Again."

Michigan voted repeal on April 3, and by the end of June, 16 states had passed repeal. By the end of October, 14 more states ratified, including several traditional prohibition strongholds. On December 5, 1933, Utah became the thirty-sixth state to pass the Twenty-first Amendment. The Eighteenth Amendment was repealed.

II. Designing alcohol control: The Rockefeller reports

In the months preceding and following repeal, the question facing the political elite was: What types of political and economic structures should be established to replace the Eighteenth Amendment? In the years before prohibition in the U.S., there had never been systematic control of the liquor industry. And the Eighteenth Amendment has not eliminated the industry, of course, but rather had profoundly altered its shape and constitution. In 1933 a huge, sprawling, and illegal industry for producing and distributing alcoholic beverages already existed. Composed of uncountable numbers of small, independent distributors and producers, and

bearing a huge sign, 'President is yours,' delivered two cases to 4 A.M. on the morning of the eight hundred cheered outside the contrast to the scene ten months ago. House gates had been chained shut protesting Bonus Marchers. Now cities and towns throughout the country reported that "Everywhere one after another, clubs, homes, even in some cities, were drinking the new beer and smiling" (p. 177). In the middle of the campaign, as the campaign song had it, "We Are Here Again."

April 3, and by the end of June, 16 more states. By the end of October, 14 more states. Several traditional prohibition states. By May 5, 1933, Utah became the thirty-first state to repeal the Eighteenth Amendment. The Eighteenth Amendment was repealed.

The Rockefeller reports

And following repeal, the question was: What types of political and economic control should be established to replace the system that had been in the years before prohibition in place of the systematic control of the Eighteenth Amendment has not been systematic control of the Eighteenth Amendment has not been course, but rather had profoundly affected the nation. In 1933 a huge, sprawling, producing and distributing alcoholic industry. Composed of uncountable numbers of distributors and producers, and

some larger ones, for 14 years this industry had, as everyone noted, kept the U.S. well supplied with alcohol. It was precisely the mass patronage of this industry—and the political and economic implications of that patronage—that had convinced prominent supporters of prohibition like Rockefeller of the necessity of repeal. Prohibitionists had always argued that unlike other industries, the liquor business was inherently unregulatable. Now the task was to show that was not true and to create structures to make the industry yield taxes and obey laws. In short, repeal posed a massive problem in social engineering and control.

During the 1920s and early 1930s, the AAPA and other opponents of prohibition had cited Canada and certain European countries, especially Sweden and England, as examples of control systems that worked. Many systems relied on government monopolies over retail sale. The AAPA in a number of publications seemed to support some version of this, but the problem remained how to transplant these foreign systems into American free-enterprise soil. There was also the additional difficulty of not establishing a system so restrictive that it too invited disobedience. Americans, after all, were by now quite used to disobeying liquor laws. The task, as the catchall title for all alternatives to prohibition stated, was "liquor control"—in the fullest sense of the term.

Rockefeller plans a report

In January 1933, a month before Congress sent to the states the proposed Twenty-third Amendment, John D. Rockefeller Jr. made another important decision. Governor Lehman of New York asked Rockefeller to serve on a committee to plan liquor legislation in the event of repeal. After discussing the matter with advisors, Rockefeller decided not to participate because the committee might be, in his words, "more or less political, that it might have on it members representing brewers, etc." Rockefeller, however, came up with a better idea, as he wrote enthusiastically to Raymond Fosdick at the time:

I should myself undertake through various competent associates a thorough and complete study of the various methods which have been tried in various countries, looking toward the control of the use of alcoholic beverages, bring together this information in a printed volume, giving the points in favor of and against each method, and seeking in conclusion to offer certain concrete suggestions as to what might seem to be the wisest method to adopt (Letter to R. Fosdick, January 19, 1933, Rockefeller Archives).

Rockefeller wrote to Fosdick (who was in Europe) to ask him to head up this study. "I am enthusiastic about this matter," said Rockefeller. "That I should sponsor something of this kind, following my letter to President Butler last spring, seems natural and to be expected, and the least that I can do to help make effective for good the outcome which that letter advocated." In fact, when Rockefeller came out for repeal, the strongest criticism he had received was that he had offered no plan to replace prohibition. He was now about to remedy that.

The man Rockefeller turned to, Raymond Fosdick, was tailor-made for the job. He was an attorney with a long interest in government policy. As a student of Woodrow Wilson at Princeton, he had been influenced by Wilson's progressivist vision of public administration working closely with business. Fosdick met Rockefeller in 1915 as a staff member of a prostitution study of which Rockefeller was a board member. Rockefeller then commissioned Fosdick to research European police systems and to write two books showing what the United States could learn from them. Fosdick's books, still classics and recently reprinted, compared the sloppy and politically controlled American police to the trained professional European police. Fosdick then helped design and administer the American boot camps for soldiers during the First World War—a major problem of social planning and control. During the 1920s, Fosdick regularly advised Rockefeller on his donations and relations to prohibitionists, and he probably drafted the famous letter to Nicholas Murray Butler. By 1933, Fosdick was on the

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boards of several Rockefeller family organizations; he was soon to become the head of the Rockefeller Foundation, a position he would hold for many years; and he would eventually write both the history of the Rockefeller Foundation and what is still the only biography of John D. Rockefeller Jr. Raymond Fosdick was truly Rockefeller's trusted advisor.

Fosdick himself immediately asked Luther Gulick, the head of the Institute for Public Administration (IPA), to help *him*. The IPA, a Progressive-era research organization, had been short of funds in the 1920s and Fosdick, who was on the board of directors, had convinced Rockefeller to endow it. Luther Gulick, in part because of his relationship with Fosdick, was also tailor-made for the job. Gulick was soon to become a major figure in the field of public administration. Dwight Waldo, the intellectual historian of the field of public administration, has called Gulick's approach "the classic theory" of public administration. Waldo summarized it as "the theory that, taking efficiency as the objective, views administration as a technical problem concerned basically with the division of labor and the specialization of function." Writing about the classic theory, Waldo (1966) said that since its major statement in 1937,

a generation of young students have demolished the classical theory, again and again; they have uprooted it, thrashed it, thrown most of it away. By and large, the criticisms of the new generation have been well-founded. In many ways the classic theory was crude, presumptuous, incomplete, wrong in some of its conclusions, naive in its scientific methodology, parochial in its outlook. In many ways it was the end of a movement, not the foundation for a science. Nevertheless, not only is the classical theory still today the formal working theory of large numbers of persons technically concerned with administrative-organizational matters, both in the public and the private spheres, but I expect that it will be around a long, long, time (p. 37).

This conception of public administration, which Gulick wrote, which Fosdick supported, and which Rockefeller

financed, was the one which shaped contemporary alcohol control policy.

Gulick assigned two IPA staff members, Leonard Harrison and Elizabeth Laine, to research liquor control. Rockefeller also asked Albert Scott, an engineer and a friend who had worked with him on a large missionary project, to head the study along with Fosdick. This constituted the core of what was called The Liquor Study Committee.

*Toward Liquor
Control*

In the summer of 1933, the group assembled near the Rockefeller summer retreat in Maine to draft the report. Gulick, who came up late, explained what happened.

I soon sized up the situation. *The Committee had already arrived at its conclusion before the study was started; the research was mostly window dressing to prove its case; what was needed was a good solid program to set up a new national system of liquor control to take the place of prohibition, and a report presenting the next steps in a clear and convincing form.* [Emphasis added.]

The first draft "had to be done by one man, with staff help at some minor points of course," felt Gulick.

Fosdick naturally agreed and asked me to undertake the job. Of course, I had to say yes, but did so only with a promise from him to really put some time himself on the final task after I had a skeleton in hand.

So I drew up first a set of major program points based on my talks with Fosdick, Scott, Harrison and Miss Laine. . . . Next I designed a "report" using pieces from various staff memoranda.

This framework was approved by Scott and RBF. Harrison, Laine, and I then went to work writing up the result. Finally, RBF went off by himself for a week and with a good deal of material which he then wrote, gave the report in its final form and life. The real style of the document and its statesmanlike quality is due to him. (Letter from Luther Gulick to Richard S. Childs, May 2, 1977, Rockefeller Archives.)

On October 6, 1933, two months before the final state would ratify repeal, Rockefeller again made front-page news, an-

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nouncing the release of *Toward Liquor Control* by Raymond Fosdick and Albert Scott. That day's press release contained the full text of Rockefeller's introduction to the book.

Rockefeller's statement began by reiterating his belief that total abstinence was best for the individual and society, and his conclusion that the attempt to enforce abstinence through the Eighteenth Amendment had been a "regrettable failure." Because of that failure, said Rockefeller, "*an evil even greater than intemperance resulted—namely, a nation-wide disregard for the law, with all the attendant abuses that followed in its train.*" As a result, Rockefeller's judgment was "[t]hat this intolerable situation should be done away with" and this task was "even more important for the moment than the promotion of temperance." He reminded people that "It was for that reason that I took a position more than a year ago in favor of the repeal of the Eighteenth Amendment." But repeal was only the first step in dealing with the problem. Therefore, he had commissioned Fosdick and Scott to study the question in order to develop "carefully laid plans of control." Rockefeller explained: "Rightly, *the first objective is the abolition of lawlessness.* Any program offered in lieu of the Eighteenth Amendment must make that its chief aim, *even if—and I weigh carefully what I say—the immediate result is temporarily away from temperance.* [Emphasis added.]" In his preface, Rockefeller stressed that generating respect for and obedience to the law should be the overwhelming concern of alcohol policy. "Men cannot be made good by force," he said. "In the end, intelligent lawmaking rests on the knowledge or estimate of what will be obeyed."

As Rockefeller indicated, the text of *Toward Liquor Control* made law obedience the central issue. In their recommendations, the authors declared that the first agenda was to rid the country of "the defiance of law that has grown up in the last fourteen years, the hypocrisy, the break-down in government machinery, the demoralization in public and private life, . . .

[and to] reestablish the integrity and dignity of the law" (p. 15). The introduction and conclusion to the book, both probably written by Fosdick, analyzed in some detail the issues involved in maintaining the legitimacy of the law. Under the heading, "The Limitations of the Law," it was observed:

Law does not enforce itself. Its machinery must be set in motion and kept in motion by human beings. As Dean Pound of Harvard has pointed out, there must be something more than the abstract content of the legal precept to move human beings to act. Certainly the only standard which the law has any hope of enforcing is the standard prevailing in the community as a whole. . . . In brief, to use the blunt phrase of "Golden Rule" Jones, former Mayor of Toledo: "Law in America is what the people will back up." Its authority is social acquiescence. Its life is in its enforcement. Victorious upon paper, it is powerless elsewhere. The test of its validity is the strength of the social reaction which supports it (p. 8).

The last pages of the book warned that law must be continually adjusted to the changing demands and customs of the population which it regulates and controls. "We need to be on our guard against any system of control that has outlived its usefulness and that no longer represents the prevalent ideas and attitudes of the community," wrote Fosdick and Scott.

In the last analysis, there is but one fundamental rule to be followed—and all other rules are corollaries: If the new system is not rooted in what the people of each state sincerely desire at this moment, it makes no difference how logical and complete it may appear as a statute—it cannot succeed (p. 152).

Though few at the time recognized it, *Toward Liquor Control* had taken as its central conclusions virtually all of the central policy recommendations made by the Committee of Fifty 30 years before. The Committee of Fifty's summary volume and the Rockefeller report both asserted that the legitimacy of the law must be of primary concern in liquor regulation. The reports agreed that the specific content of the

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law mattered less—far less—than that it be obeyed. Both reports urged that alcohol regulators adopt a flexible system, continually monitored and adjusted. And both reports advised that, if at all possible, government take over the selling of alcoholic beverages. Fosdick and probably the other study members had read the Committee of Fifty's books. The Committee was quoted at length on the corruption and lawlessness resulting from prohibition, now dramatically confirmed by national prohibition, and was listed seven times in the index, more than almost any other single item.

The specific plan for liquor control suggested by Fosdick and Scott, and their most controversial proposal, was that each state take over as a public monopoly the retail sale for off-premises consumption of spirits, fortified wine, and beer above 3.2 percent alcohol. "The primary task of the Authority would be the establishment of a chain of its own retail stores for the sale of the heavier alcoholic beverages by package only." The government-run outlets of Quebec and other Canadian provinces, Sweden, Norway, Finland, and the Carlisle State Management Scheme in England were cited as working examples of such a plan. This quickly became known as the "Monopoly plan" and was usually called the "Rockefeller plan."

For those states not willing to establish government liquor stores, Fosdick and Scott proposed a parallel system: "regulation by license." England was cited as the best example of a working license system. A single, nonpartisan board appointed by the governor would have statewide authority to issue liquor licenses and regulate the industry. There would be no "tied house" permitted, that is, no retail establishment owned directly by a distiller or brewer or under exclusive contract.

For both plans, *Toward Liquor Control* outlined a detailed set of matters over which the state agency would have jurisdiction. These included the power to acquire real estate

and other capital by purchase, lease, or condemnation; determine and change prices at will; establish a system of personal identification of purchasers; issue permits for and regulate the use of beer and wine for off-premises consumption and for on-premises consumption in "hotels, restaurants, clubs, railway dining cars, and passenger boats"; require alcohol manufacturers and importers to report on quantities; regulate or eliminate alcoholic beverage advertising; determine the internal design, visibility from the street, hours and days of sale, number, and locations of alcohol outlets.

Although it described guidelines for a licensing plan, *Toward Liquor Control* favored the monopoly plan over private businesses. The possibility of increasing profits, they said, would encourage private businesses to sell more alcohol, buy political influence and lax enforcement, and violate laws. The chief advantage of government-owned liquor stores was explained by Rockefeller in his foreword. "Only as the profit motive is eliminated," Rockefeller wrote, "is there any hope of controlling the liquor traffic in the interest of a decent society. To approach the problem from any other angle is only to tinker with it and to insure failure. This point cannot be too heavily stressed." The irony of a Rockefeller speaking about the dangers of the profit motive was not lost on observers at the time. Like others, Rockefeller and his associates had concluded, probably correctly, that government ownership of retail sale brought greater powers to regulate and control behavior, and ensure obedience to the law.

The sophisticated public relations operation run by Ivy Lee, which handled other Rockefeller announcements and projects, made sure that the report received maximum attention. After the October 6 announcement, the book was released in regular portions and sent out to newspapers and magazines as major press releases. Many newspapers, like *The New York Times*, published each portion of the book as it arrived.

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Two and a half weeks later, the last installment was released, the book was officially issued, and thousands of copies were sent out to the press, state legislators, policy makers, and influential citizens.

Not surprisingly, the response to *Toward Liquor Control* was very favorable. *The Chicago Daily News* told its readers that the book's conclusions "are well worthy of careful study by law makers. They are the conclusions of enlightened and liberal minds and are based on common sense." *The New York World Telegram* said that it contained "excellent general reminders and guides," and *The Baltimore Sun* reported that "The Rockefeller report has done a service to the country and to the cause of temperance." (All quoted in *Literary Digest*, October 28, 1933.) *The Nation* praised it twice. On November 8, in a full-page, signed editorial, the editor of *The Nation* declared: "John D. Rockefeller, Jr. Does a Fine Job." Rockefeller, he said, had "rendered a public service of great value in sponsoring the book," and "nobody should be allowed to discuss the problem hereafter unless he perused the volume." Two weeks later (on November 22), another article in *The Nation* urged that *Toward Liquor Control* "be placed immediately in the hands of all who will participate in liquor control legislation," and commended Rockefeller, "perhaps the outstanding pillar of the profit system," for recommending that the profit motive be removed from the liquor business. *The Commonwealth* said in November that "few, if any, more timely practically useful acts of public service have ever been performed in the field of social research than the study sponsored by John D. Rockefeller, Jr."

Rockefeller himself received a great deal of mail thanking him for the report. Edgar Allan Poe, Jr., one of the leaders of an antiprohibition organization, the Crusaders, told him that he intended to draw upon it "extensively in some speeches I have been asked to make in the City with relation to our Liquor Control Bill." The governor of Virginia wrote

to Rockefeller twice, first to praise him, and then to say he had asked Fosdick and Scott to consult with him about setting up Virginia's liquor control. (All letters are in the Rockefeller Archives.)

The most pertinent letter Rockefeller received (from the point of view of this article) was from Francis Peabody, the former secretary of the Committee of Fifty. Thirty years earlier, in 1904, Peabody had edited *The Liquor Problem*, the summary volume of the committee's conclusions. Now, Peabody, a personal acquaintance of Rockefeller, wrote to express his approval and to say "how much satisfaction I have found in the Report of your agents." Peabody said that he agreed completely with the plan for a government monopoly, and that he thought that the Fosdick and Scott report concurred with the aims of the group that "some thirty years ago I had the privilege of organizing." Peabody had every reason to be gratified: the Committee of Fifty's views had finally triumphed, validated in the most prestigious way imaginable.

The report also received some criticism. The *New York Daily News*, for instance, dismissed it, observing that Rockefeller's "contribution to the Anti-Saloon League did a lot to foist Prohibition on us." The *News* argued that "the people can solve their liquor problems better than John or any other reformed Prohibitionist can" (quoted in *Literary Digest*, October 28, 1933, p. 10). Perhaps the most trenchant criticism of the book was a review in *Christian Century* (November 11, 1933), titled "Fitting the Law to the Lawless," by a moderate prohibitionist (W. E. Garrison). "It seems fair to say," said the reviewer, "that the guiding principle in the report is the desire to construct a system which will be satisfactory to the people who want to buy and drink liquor that they will not find the legal hindrances annoying." While the author did not argue with that point, he did draw out some of its broader implications:

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In the very foreground of the Rockefeller report's argument is the wish to "reestablish the integrity and dignity of the law." Yet the assumption is that the people who want to drink alcoholic beverages have firmly determined that they will not respect any law which conflicts with their desire. . . . I find in all its pages not one glimmer of hope that anybody will obey, or ought to be expected to obey, any law that does not tell him to do what he was going to do anyway. The authors may be right. I am not arguing the point except to say that adjusting the law to the wishes of those who have shown themselves despisers of law (meaning patrons of the bootleggers) does not seem to me a procedure well calculated to "reestablish the integrity and dignity of the law." There is a story, so old that it may have been forgotten, of a man who orders his dog to go outdoors. Instead, the dog slunk under the bed. "Very well," he said sternly, "go under the bed. I will be obeyed."

This reviewer had correctly seen that the Rockefeller Commission's plan for alcohol control was concerned with creating the immediate *illusion* of authority and control. What he had not understood, however, was the importance of that illusion in the short- and long-term process of developing and maintaining real authority and control over the alcohol industry, and over the larger society. The legitimacy and authority of all law required that this alcohol law appear to be effective. For the sake of the whole system of state regulation, as well as the possibility of any future workable alcohol regulation, alcohol law did indeed have to be "fitted" around those who were willing to violate it. It had to be made to seem as if the masses of people were obeying the law.

Alcohol control laws

Fosdick decided that *Toward Liquor Control* would contain detailed guidelines but would not propose model laws. He understood that it was an important task, and he assigned Luther Gulick to write the sample law. Gulick, who eventually became something of an expert in drafting model laws, turned to another progressivist organization, The National Municipal League, to draft a model liquor control law. Gulick gathered what he called "a dozen close friends and members of our IPA board" with ex-governor Frank O. Lowden as chairman, and formed The Committee of Liquor

Control Legislation of the National Municipal League. Gulick served as secretary of the committee. On November 11, 1933, a month before repeal went into effect, at the National Municipal League convention, the committee endorsed the Rockefeller report and the Liquor Authority plan.

The National Municipal League Convention also heard an address on the principles of liquor control legislation by Pierre du Pont. As head of the AAPA, du Pont represented the only powerful group speaking on the topic of alcohol control. Besides having led the fight for repeal, the directors of the AAPA were the top executives and board members of many major corporations. Clearly, what they thought about alcohol control would be important. Without explicitly saying so, du Pont overwhelmingly supported the conclusions of *Toward Liquor Control*. In his address he listed what he called "the fundamentals of satisfactory liquor control," and the first two points he listed were the central conclusions of the Rockefeller report. First, he declared, "All operations concerning the manufacture, sale, transportation and use of alcoholic liquors must be brought into legal and honorable channels as quickly as possible *in order to restore respect for law* and respect for a national morale that has suffered much under prohibition." And second, he recommended, "The places and time for sale of alcoholic beverages must conform to popular demand, even though we may deplore the existence of this demand and may be tempted to turn to prohibitory law and forceful methods for its regulation or suppression." In short, du Pont ringingly seconded the general principles of alcohol control outlined by Fosdick and Scott.

The statement of the committee, the draft of the Model Law for a State Alcohol Authority, and the speeches were published in a supplement to the *National Municipal Review* in January 1934, the first month of Repeal, and widely circulated to legislators throughout the country. State legislators faced with difficult political choices, and with little

National Municipal League. the committee. On November repeal went into effect, at the convention, the committee endorsed the Liquor Authority plan.

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personal expertise in the subtle question of liquor regulation, turned to the authoritative and virtually unchallenged plans of the Rockefeller commission and the National Municipal League. Gulick estimated that the monopoly law was taken almost verbatim by 15 states, and the licensing law served as the text or draft for many more (Letter from Luther Gulick to Richard S. Childs, May 2, 1977, Rockefeller Archives).

After Repeal

In 1936, a second volume of the Rockefeller-sponsored Liquor Study Committee was issued. Written by Leonard Harrison and Elizabeth Laine, staff researchers on the first book, it included an introduction by Luther Gulick. The book, *After Repeal: A Study of Liquor Control Administration*, analyzed the results of liquor control after "a two year trial," and described the most important changes and innovations in liquor administration instituted since repeal.⁴

The overall thrust of the report was that, with some understandable exceptions, alcohol control worked extremely well. Still, the last pages of the book emphasized what the authors regarded as the central issue:

In a very real sense the whole administrative task of liquor control is one of law enforcement. . . . Two decades ago, the public believed that the evils connected with the liquor traffic would not or could not be corrected by public officials and that prohibition was the only remedy. A national revulsion against the breakdown of law enforcement was what caused prohibition's repeal. The liquor issues of the future will be decided, as always before, on the basis of success or failure in enforcement of whatever kind of control is attempted. [Emphasis added.]

III. Regulating daily life: the order of alcohol control

The two Progressive-era liquor reforms, prohibition and alcohol control, shared a common political and economic agenda. Both sought to alter the entire terrain of alcohol production, distribution, and consumption. They agreed that the state and law were appropriate mechanisms for doing

that. Both sought centrally directed efforts at transforming the industry and social life associated with drinking. The Anti-Saloon League, on the one hand, and the Committee of Fifty and the Rockefeller reports, on the other hand, rejected laissez-faire models of the state. Unlike the general thrust of nineteenth-century temperance and prohibition crusades which saw social change arising from a broad reformation of the character and morals of the American people as a whole, the Progressive-era efforts sought to impose laws primarily because of people's flawed characters and morals. In Gusfield's terms (1963), whereas the nineteenth-century reformers sought "assimilative" reform, the twentieth-century approach was "coercive" reform.

Because alcohol control has seemed so much more "permissive" than prohibition, alcohol control's coercive and interventionist character has been largely unnoticed—it has seemed "natural" and unproblematic. For example, in his recent study of the AAPA, David Kyvig (1979a) has argued that what really motivated the wealthy men who led the fight for repeal—in addition to reducing taxes and concern for lack of respect for the law—was a deeply felt conservative libertarianism: they were appalled by the intrusion of government, especially the federal government, into so personal a matter of daily life as drinking alcohol. Kyvig argues that the heads of the AAPA, including du Pont, Raskob, Sabin, and others, were motivated by a fear that government was, in effect, becoming an intrusive "big brother" interfering with all aspects of social and economic life. Kyvig's argument makes a certain amount of sense: these wealthy industrialists and bankers strongly disliked some kinds of government regulation. In the 1930s, after winning the fight for repeal, they constituted the core of the big business opposition to Roosevelt. Reorganizing themselves as the Liberty League, they opposed Roosevelt's programs for Social Security, the WPA, and other welfare and social service programs—they even opposed child labor laws (Wolfskill 1962; Kyvig 1979b). However, the leaders and board of the AAPA clearly were

directed efforts at transforming the associations with drinking. The one hand, and the Committee of ports, on the other hand, rejected state. Unlike the general thrust of temperance and prohibition crusades arising from a broad reformation of the American people as a whole, they sought to impose laws primarily on characters and morals. In contrast to the nineteenth-century reformers, the twentieth-century ap-

seemed so much more "permissive" control's coercive and intervention largely unnoticed—it has been problematic. For example, in his book David Kyvig (1979a) has argued that the wealthy men who led the fight against taxes and concern for the state was a deeply felt conservative reaction to the intrusion of government, into so personal a matter as alcohol. Kyvig argues that the leaders, du Pont, Raskob, Sabin, and others, feared that government was, in effect, a "big brother" interfering with economic life. Kyvig's argument is that these wealthy industrialists, in some kinds of government action, winning the fight for repeal, the big business opposition to themselves as the Liberty League, the Social Security, the social service programs—they (Volfskill 1962; Kyvig 1979b). The leaders of the AAPA clearly were

not against all government regulation. They were much more class-conscious and self-interested than Kyvig implies, and they sometimes found government intervention useful when it served their interests.

Alcohol control was not, in fact, the diminution of government control over daily life, and it aimed not to reduce government involvement in the production and distribution of alcohol. Rather, alcohol control aimed to substantially strengthen and enhance government power and involvement. The advocates of liquor control proposed to construct and administer a system of regulation far more pervasive, intrusive, and effective than anything before established in the United States. The leaders of the AAPA did not shy away from such government involvement—they welcomed it and encouraged it. After repeal, for example, Pierre du Pont sat on the Liquor Control Board of Delaware for a number of years, as did other leaders of the AAPA and its sister organization, WONPR, in other states. Some of the control systems they advocated, the Swedish model in particular, were remarkably intrusive. The Swedish system, usually known as the Bratt plan, required that each citizen be issued a passbook which would be stamped every time he or she bought alcohol, and each person would be rationed a specific amount each month.

A historical parallel helps to pinpoint the issue. Michel Foucault (1979) has observed that penal reformers at the end of the eighteenth century who sought to eliminate torture as a means of punishment are usually pictured as humanitarians seeking to eliminate the excesses and inhumanities of torture. They were, says Foucault, concerned with these things, but they were concerned with more. The reformers had become convinced that torture no longer worked as a system of control. Its excesses and arbitrariness enraged the very population it was meant to intimidate. Instead of standing in awe in front of the scaffold, angry mobs tore it down. Foucault argues that the penal reformers, middle-class fol-

lowers of the Enlightenment, had not only a negative aim, the elimination of the excesses and brutalities of torture, but also a positive aim, punishment and correction that really worked. In Foucault's words: "They sought to insert the power to punish deeper into the body politic." Similarly, I am suggesting that the advocates of alcohol control wanted more than the elimination of the problems created by the Eighteenth Amendment. The advocates of alcohol control also sought greater and more effective regulation of the alcohol industry as a way of further controlling a range of everyday behaviors, experiences, and thoughts associated with drinking. They sought to insert the power to regulate deeper into everyday life by intervening into every aspect of the production and distribution of alcoholic drink.

During Prohibition, the liquor business was wide open. Speakeasies closed when they wished or not at all; they sold whatever they wanted, to whomever they wanted, at whatever price they wanted. They decorated as they wished, provided whatever food or entertainment they wished. Producers made alcohol in any strength they wanted, in any way they wanted, using whatever products they wanted. Neither producers nor distributors paid any taxes (except for payments to police and politicians) and they were not regulated by any government agency. During Prohibition the liquor industry was probably the freest big business in American society.

Alcohol control, on the contrary, was premised on massive government intervention into every aspect of the liquor business. Controversial issues, such as whether food must be served, women admitted, music and games banned, bars and barstools allowed, all had to be settled. The number, types, and locations of on-premises and off-premises outlets, and their hours of sale, had to be determined. Producers had to be regulated to ensure that products were safe and of a uniform alcohol content. In order to eliminate untrustworthy or disreputable persons, both producers and distributors had

had not only a negative aim, and brutalities of torture, but not and correction that really "They sought to insert the body politic." Similarly, I am alcohol control wanted more problems created by the Eighteenth Amendment of alcohol control also gave regulation of the alcohol controlling a range of everyday pleasures associated with drink—power to regulate deeper into every aspect of the production of the drink.

For business was wide open. Unrestricted or not at all; they sold whatever they wanted, at whatever price they wished, wherever they wished. Prohibition length they wanted, in any way products they wanted. Neither did any taxes (except for pay-ment) and they were not regulated. During Prohibition the liquor business was the biggest business in American

history, was premised on massive regulation of every aspect of the liquor industry such as whether food must be served and games banned, bars and restaurants settled. The number, types, and locations of off-premises outlets, and the hours were determined. Producers had to ensure their products were safe and of a certain quality to eliminate untrustworthy producers and distributors had

to be screened and licensed, and they had to be made to pay taxes. Moreover, this all had to be done so as not to upset the abstaining portion of the population—about half the adults. And it had to be done without making regulation so tight, or taxes so high, that drinkers preferred to patronize their bootlegger or speakeasy—to return to patterns of law disregard. Constructing alcohol control, in fact, involved issues of government regulation so large as to make some of the classic Progressive-era concerns—regulating meat-packing, for example—seem paltry in comparison. Except for national prohibition, alcohol control is probably the most striking twentieth-century example of government force used to fundamentally reshape an entire industry and the way its products are consumed.

Transformations

Postrepeal regulation transformed the alcohol beverage production industry. Other nations, notably Finland, had nationalized production of spirits, but such proposals were not seriously discussed in the United States. Instead, production was monopolized by a relatively few corporations. Alcohol control transformed production even more dramatically than had prohibition. Before the Eighteenth Amendment, liquor was produced by many small distillers and a number of larger ones. During Prohibition, the distilling industry was further divided: many more small and individual distillers operated profitably. However, within four or five years of repeal, 75 to 90 percent of all distilled liquor in the United States was manufactured by only four corporations (Culver and Thomas 1940). The beer industry, while more diverse nationally because beer required local and quick distribution, was monopolized by region or area. Both federal and state regulatory agencies preferred to deal with a few large corporations—they were easier to police, to make agreements with, and more likely to be concerned with keeping the image of the industry clean and respectable. This pattern of monopolization was not unique, of course: most major American industries—steel, automobiles, soft drinks, chemicals, for example—were increasingly dominated by three or four

corporations. Alcohol production was exceptional only in the speed of monopolization.

While production was monopolized, distribution was splintered and scattered. Perhaps the most important long-term innovation in post-Prohibition alcohol regulation was that it distributed the legal selling of alcohol to a wide variety of sites. Before Prohibition the saloon had been a single, all-purpose institution—there one drank beer, wine, or spirits, and there one purchased for off-premises consumption a bottle of spirits or a bucket of beer (beer canning and bottling had not yet developed on a large scale; most pre-Prohibition and Prohibition-era beer was keg beer). After repeal, alcohol control created several different types of establishments to sell alcoholic beverages. In most states special stores were created to sell only distilled liquor—often they could not sell any food at all, or even cigarettes. Beer, on the other hand, was made relatively widely available in bottles and cans—grocery stores and small markets were licensed to sell it. In other words, after Prohibition, alcohol was increasingly separated from the public drinking place; drinking became increasingly privatized. Whether alone or with others, drinking became something more commonly done at home. By 1941, off-premises consumption accounted for the majority of alcohol sales (Kyvig 1979a, p. 189). Finally, distribution was severed from production: “tied house” laws in most states banned or restricted economic connections between producers and retailers.

The character of public drinking was significantly altered by these legal and regulatory changes. A new class of licenses for on-premises consumption of beer only, or of beer and wine, was established and liberally issued to restaurants, diners, cafeterias, and greasy spoons. This separated out the bar-room selling distilled liquor and beer as a distinct institution. Many state alcohol control laws made provision for a local option whereby a county government could prohibit specific kinds of liquor selling within its borders. This option has

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been widely exercised. As late as 1973, of the 3,073 counties in the United States, 672 prohibited sales of distilled liquor by the drink for on-premises consumption, and 545 totally prohibited sales of distilled spirits (Joint Committee of the States 1973).

Under alcohol control, all establishments for on-premises consumption of spirits were specifically restricted and shaped in a multitude of ways. In some areas, spirit sales were limited to bona fide restaurants with laws specifying how many feet of kitchen space and how many food preparation workers there must be. All or most states have restrictions on the following: the number of entrances and their locations—back entrances are usually prohibited; the times of day and days of the week a place selling alcohol may be open; what decorations are prohibited; the degree of visibility of the interior from the street; the numbers and uses of other rooms; distance of the establishment from churches, schools, and other alcohol outlets; whether customers may sit at a long bar—a counter with a foot railing—or whether they must sit at tables; the ratio of chair seating to bar seating. Many states prohibit dancing or live music except under special license. Most gambling or betting is prohibited, and other games are restricted as well—for many years, New York and other states did not allow barrooms to have pinball machines. Many states specifically ban the use of the word "saloon," others the use of the word "bar," and some prohibit all words to indicate a drinking place. In California until recently the only sign allowed other than the name was a symbol, a tilted glass with a stirrer.

The effect of all these changes was to eliminate the saloon as *it had been*. For the Committee of Fifty, the most noxious feature of the saloon was not that it sold drink, but that it was the center of working-class social life—the "workingman's club." From this stemmed its subversive character. As the committee explained, "liquor dealers are not the proper persons to have charge of the social life of our

American working people." Because it was the saloon's "social functions" and not its beverages which were most problematic, the committee did not call for prohibition. Rather, the committee sought what it called bluntly "the legislative repression of the social side of saloon life." This agenda was shared by prohibitionists, of course: in 1933 wets as well as dries agreed that suppression of the traditional working-class saloon was one of prohibition's truly valuable achievements. Alcohol control has extended and made permanent the transformation begun during Prohibition. The barroom is no longer *the center* of (male) working-class social life, and working-class leisure has been systematically integrated into traditional middle-class patterns, especially into homelife. New amusements and activities, many of which include drinking, have become "substitutes for the saloon" as the Committee of Fifty proposed. The barroom has ceased to be the primary meeting place for union organizers and leftists, and mainstream politicians no longer find it a major recruiting agency. One does not go there to get a job or help from a politician. In certain carefully controlled forms, the working-class *drinking* place remains, but it exists in an entirely different context from the old saloon, and with none of its political and economic functions.

From a pre-Prohibition or Prohibition-era perspective, there are two other surprising characteristics of postrepeal alcohol control. First, virtually all laws and regulations are obeyed. Drinking places, for example, scrupulously stop serving and collect glasses at the required hours; and they observe the regulations about tables, dancing, decorations, signs, entrances, and so on. Overwhelmingly, alcohol law is obeyed.⁵ Second, postrepeal alcohol regulation is usually not perceived as especially restrictive by customers. The many laws and restrictions are rarely noticed or are completely taken for granted.

A third, less surprising characteristic of postrepeal alcohol control is that policy is not aimed at maximizing what

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nineteenth-century and Progressive-era reformers called "temperance"—meaning, above all, reducing habitual drunkenness or repeated heavy drinking as a way of reducing other social problems, especially poverty, crime, individual failure, family dissolution, violence, and illness. Those are not the aims of alcohol control. In his preface to *Toward Liquor Control*, Rockefeller maintained that such things could not be the concerns of liquor regulation—they would have to be taken up by other agencies and incorporated as part of broader educational efforts. In effect, alcohol control carved out a separate space or a distinct series of tasks, apart from the issue of liquor regulation, for public education and health. And since repeal these tasks have been taken up by a number of independent and government groups, notably Alcoholics Anonymous and the National Council on Alcoholism, various state alcoholism agencies, and in 1970 a federal agency—the National Institute on Alcohol Abuse and Alcoholism. Alcohol control is concerned with public order and not public health.⁶

For nearly 50 years, alcohol control has smoothly, quietly, and effectively organized and managed not only the production, distribution, and sale of alcoholic beverages, but also much of the social life associated with drinking. What once seemed impossible was actually achieved very quickly and efficiently. It is a coercive system, but one designed in a particular way—as *Christian Century* (Garrison 1933) noted: to some extent regulation has been shaped around the preferences of drinkers and the desires of the alcohol beverage industry. As the Committee of Fifty recommended, alcohol regulation has been concerned above all with promoting "order, quiet, and outward decency" and has sought "all external, visible improvements." It has successfully done this by working to keep friction and conflict between individuals and the system to a minimum. The social environment of drinking—how it is sold, where, when, in what kinds of places—all this has been subject to intense manipulation. Yet unlike the situation under contemporary marijuana prohibi-

tion, for example, individual consumers are rarely cited for violating liquor laws: they do not come in conflict with the state; normally they do not even feel it impinge. Alcohol regulation is not a part of everyday consciousness and therefore it does not raise any broader political questions about law or government.

A general, comprehensive set of principles has been articulated from the Committee of Fifty, to both Rockefeller-sponsored books, to the most recent report by the organization of alcohol control administrators (Joint Committee of the States 1973). Since the early twentieth century, all authorities have agreed that the regulation of this particular area of daily life requires minimizing political, ideological, and cultural resistance to the regulatory system while maximizing social control and order. The system is organized so that the task of administrators is to maintain social order and discipline by flexibly using the powers contained in a battery of potential interventions. The image and example of "lawless" unregulated prohibition are available as a sort of rhetorical and ideological resource, and are cited even in present-day publications: "We don't want to bring back the situation of prohibition when there was no law or regulation" is the warning. The alcohol beverage industry now has much to lose by disorder; the handful of corporations which monopolize production have a major stake in keeping the system functioning without resistance. The underlying logic of alcohol control has become institutionally, ideologically, and administratively hegemonic: the most important tasks are maintaining orderliness, obedience to law, and the acceptance of the broader government system of regulating daily life.

Despite frequent claims to the contrary, alcohol control has of course sought to legislate morality—not the morality of the nineteenth-century Victorian middle class, but the morality of the new business and professional middle class, of the corporate elite, and to some extent of the twentieth-century working class. Production has been monopolized, distribu-

al consumers are rarely cited for not coming in conflict with the law, even feel it impinge. Alcohol is part of everyday consciousness and raises any broader political questions

of principles has been articulated. Fifty, to both Rockefeller-sponsored report by the organization of the Joint Committee of the Twentieth Century, all authorities on this particular area of daily life, political, ideological, and cultural system while maximizing social order is organized so that the task of maintaining social order and discipline by a battery of potential examples of "lawless" unregulated behavior as a sort of rhetorical and cited even in present-day publications to bring back the situation of "no law or regulation" is the reason the liquor industry now has much to lose. The underlying logic of alcohol control is that the most important tasks are to maintain obedience to law, and the accepted system of regulating daily

life. In contrast, alcohol control has been a morality—not the morality of the middle class, but the morality of the professional middle class, of the extent of the twentieth-century economy has been monopolized, distribu-

tion scattered, and the working-class "saloon" stripped of its political and economic functions. Unlike other "vices" or "pleasures"—prostitution, gambling, marijuana, heroin, or cocaine—alcohol is not an outlawed industry in the hands of independent entrepreneurs paying off police and government officials. The heads of the major alcohol-producing corporations are members of the economic establishment with an investment in maintaining order and obedience to law. All of this happened, I am suggesting in this article, not because people voted for it, or because it was "natural," but for specific historical and sociological reasons.

IV. Conclusion

The passage of the Eighteenth Amendment was not inevitable, nor was the creation of alcohol control when and in the form it was instituted. National prohibition would not have been enacted without a mass movement organized around the idea that alcohol was a destructive and demonic substance. But the existence of that movement, and of the public support it gathered, was not enough to pass an amendment to the Constitution. Many issues with a majority of public support—the recent Equal Rights Amendment, for example—do not get written into the Constitution. The Eighteenth Amendment was ratified in part because no significant elements of the corporate elite opposed it, and a number of important corporations and individuals supported it. Wealthy businessmen supported prohibition because they thought it would reduce crime and other social problems; make workers more efficient, productive, and disciplined; and eliminate the saloon as a center for working-class social and political life. To many businessmen at the time, prohibition seemed like a good deal.

However, the Eighteenth Amendment and the Volstead Act created new problems that members of the corporate elite came to regard as detrimental and threatening to their economic and political interests. Some, notably the leaders of

the AAPA, believed that prohibition was costing them a great deal of money because of lost liquor tax revenue. They and others also believed that prohibition was turning the working class and much of the middle class against the government and law. Prohibitionists in the 1920s were the first to argue that mass disobedience to prohibition was creating disrespect for all law, including property rights, and helping the cause of Communists and radicals. Increasing numbers of those opposed to prohibition became convinced of that during the 1920s, but it took the upheavals and devastation of the Great Depression to fully make the threat of mass revolt seem real to significant elements of the political and economic elite.

Respect for capitalism and the legal order it required did indeed appear to be coming undone in the 1930s. To many people, law and government in the United States no longer seemed to be concerned with the interests or the welfare of most people. The Great Depression created the most serious crisis of the legitimacy of the state and of law in twentieth-century America, and most establishment observers concluded that prohibition was making things worse. The Wickersham Commission found that prohibition was inducing law disobedience, undermining respect for law and government, and fostering resentment among the working class. During the 1930s, a number of wealthy businessmen who had originally supported prohibition turned against it because they became convinced that the lack of respect for law generated by prohibition, coupled with the conditions of the depression, had created an explosive situation. As Sinclair (1964) has pointed out:

This lawlessness, spawned by prohibition, now threatened to spread with mass unemployment, and shake the roots of society. The same employers who had supported the Eighteenth Amendment a decade earlier to benefit themselves and their workers now advocated repeal to protect themselves from their workers. They hoped that legal beer would relieve some of the social tensions of the time and lessen class hatred (p. 384).

prohibition was costing them a great deal of lost liquor tax revenue. They and prohibition was turning the working middle class against the government in the 1920s were the first to argue prohibition was creating disrespect for property rights, and helping the cause of criminals. Increasing numbers of those who became convinced of that during the upheavals and devastation of the Great Depression, the threat of mass revolt seemed real to the political and economic elite.

And the legal order it required did not seem undone in the 1930s. To many in the United States no longer aligned with the interests or the welfare of the country, the depression created the most serious crisis of the state and of law in twentieth-century history. Most establishment observers concluded that prohibition was making things worse. The fact that prohibition was undermining respect for law and law, and resentment among the working class, and a number of wealthy businessmen had turned prohibition against it convinced that the lack of respect for law, coupled with the conditions of the depression, had created an explosive situation. As Sin-

cey prohibition, now threatened to undermine the roots of society. It supported the Eighteenth Amendment and it themselves and their workers now freed themselves from their workers. They believe some of the social tensions of the 1920s (p. 384).

Nineteen thirty-two was probably the worst year of the depression. There seemed to be nothing that anyone could do to affect the economy: the presidential campaign produced no new economic proposals or program—Roosevelt campaigned on fiscal conservatism and balancing the budget. But significant elements of the elite had concluded that at least something could be done to remove one major source of public resentment. The Repeal Amendment was speeded through Congress and then the states—it was made available for people to vote on—because Rockefeller and the directors of the AAPA, and those they represented, were convinced that something had to be done to restore some degree of public confidence in law and government and to reduce public resentment of government.

The state policy for regulating liquor which was established at that time sought to carve out an area of social life where law was obeyed, where the system worked. As late as 1931, repeal had still seemed impossible. As a result many people, especially representatives of labor, proposed not repeal but modification of the Volstead Act to allow for legal sale of beer and wine. This was unacceptable to Rockefeller and the AAPA because it still left a large area of drinking behavior illegal and thus did not speak to the central issue of developing respect for law. Only by creating a situation where every aspect of drinking could be legal would a fully "lawful" environment be established. And the alcohol control policy which was established did help to affirm the legitimacy of the state and its law. The rebelliousness and discontent associated with liquor restriction were eliminated almost immediately: the taxes were collected and the laws obeyed.

It is important to recognize, however, that the institutionalization of alcohol control was by no means a complete victory for the corporate elite. Many of them, perhaps most, had supported prohibition and only reluctantly gave up on it. Those like Rockefeller who changed their minds in the 1930s had repeal forced upon them by the very population they

sought to control. Unable to end the depression, and unwilling to make significant economic changes, they felt obliged to make symbolic and cultural changes—to make it cheaper and easier for working people to drink. Legal sale of alcohol did not, of course, quell riot and revolt. That was largely accomplished by Franklin D. Roosevelt and the symbolic and real measures of the New Deal. But elite and upper-class support for repeal, set in motion before Roosevelt took office, was the first and perhaps final unified response to the depression of nearly the whole capitalist class.

The often-cited lesson of prohibition—that one cannot legislate morality—is actually a way of addressing in a kind of shorthand code a more complicated problem. It is what Rockefeller and Fosdick spoke to directly in *Toward Liquor Control*: the problem of how an elite can regulate society in its interests while obtaining the consent of the governed. Rockefeller and Fosdick were acutely aware that they were attempting to use the state to control a volatile population which could nullify, by individual and collective action, *any law*—not just alcohol prohibition. However, even during the depression the upper class was not willing to alter fundamental property laws or even change the distribution of wealth. These were the essentials which had to be preserved. But if necessary, concessions could be made on lifestyle and cultural issues.

Alcohol control was a compromise made by the political and economic elite: they found it necessary and desirable to make concessions partly because so many people were willing to engage in “lawless” behavior. The presumed costs of increased alcohol consumption were no longer as important to them as increases in tax revenue, the continued legitimacy of law and government, and working-class good will. Perhaps the largest specific concession to the working class was making beer widely available—in groceries, delicatessens, and markets of all sorts, as well as in inexpensive restaurants, diners, and cafes. In most places where workers bought food

to end the depression, and unwilling economic changes, they felt obliged to make cultural changes—to make it cheaper for people to drink. Legal sale of alcohol was a riot and revolt. That was largely the work of Roosevelt and the symbolic and political Deal. But elite and upper-class opposition before Roosevelt took office was perhaps final unified response to the role of the capitalist class.

Prohibition—that one cannot legislate a way of addressing in a kind of complicated problem. It is what we have to do directly in *Toward Liquor* how an elite can regulate society in the absence of the consent of the governed. They were acutely aware that they were unable to control a volatile population through individual and collective action, any prohibition. However, even during the 1920s, they were not willing to alter fundamental economic change the distribution of wealth. The status quo had to be preserved. But if change had to be made on lifestyle and cultural

promise made by the political and economic elite necessary and desirable to make it so many people were willing to accept it. The presumed costs of income were no longer as important to the elite, the continued legitimacy of the working-class good will. Perhaps opposition to the working class was the elite—in groceries, delicatessens, as well as in inexpensive restaurants, places where workers bought food

they could buy beer. (It is worth noting that the class politics of beer have not changed much since then: in 1982 the Reagan administration, searching desperately for new tax sources, considered and then abandoned the idea of raising taxes on beer—the White House concluded that beer was still the “workingman’s drink” and Reagan did not want to further alienate workers. Fifty years earlier, beer was even more the worker’s drink.) But at least some workers were also whiskey drinkers and besides making off-premises consumption legitimate, most large and medium-sized cities were obliged to legalize the whiskey barroom. After repeal, some places with substantial numbers of industrial workers tried to outlaw on-premises spirits consumption. Most, like Connecticut and San Francisco, for example, when confronted with continued “lawlessness” abandoned the attempt almost immediately.

In short, the political and economic elite’s concern with maintaining the appearance and illusion of voluntary law compliance meant that working-class men and women, in conjunction with the increasing numbers of middle-class drinkers, were able to force the authorities to make alcohol far more available than would otherwise have been the case. Alcohol control laws and regulations were, in effect, codifications of the political, economic, and cultural struggles between the elite and the people they sought to control.

Notes

1. Virtually every commentator at the time and since has pointed out the importance of the economic argument in winning support for repeal. Davis, writing in 1933, noted that “government is going to need a great deal of money in the next few years,” and that “one of the most powerful arguments in the repeal campaign was the possibility of substituting liquor taxes for others that have proved more burdensome” (p. 284). In an article called “Wall Street Cheers Repeal,” *Business Week* claimed in 1933 that “the promise of tax revenue more than anything else turned the country toward repeal,” and concluded that “there is dancing and singing in The Street.” Hacker (1932) noted the “widely expressed belief that the revival of liquor manufacturing under proper restraints would provide the

necessary new opportunities for the investment of capital and the employment of labor" and that "liquor would provide new sources of tax revenue." Jones (1961) concluded that in the thirties, "government finances and respect for the law, either singly or in combination, became the pervasive arguments for repeal" (p. 275). In this article, I have concentrated on the concern with lawlessness, as I say in the text, because the economic argument has been mentioned frequently, because the concern with law legitimacy was neglected or misinterpreted, and because the concern about lawlessness was absolutely central to the construction of alcohol control policy. For discussion of the economic arguments for repeal and the economic interests in it, see Dobyns (1974); Jones (1961); Englemann (1979); and Gordon (1943).

2. From the mid-1920s on, the leaders of the AAPA had argued that liquor taxes could virtually replace personal and corporate income taxes. The AAPA issued and widely distributed pamphlets arguing that liquor taxes could generate one billion dollars a year and could, for example, pay the entire federal deficit. In the 1930s they found a new constituency for their economic arguments. State and local elected officials, among others, faced with severe drops in tax revenue, and huge demands for services, found the economic arguments very attractive. Further, during the depression many people claimed that the revived liquor industry would put men back to work and stimulate the economy.
3. This and other correspondence to and from John D. Rockefeller Jr., his father, and his close advisors, cited or drawn upon for this article, is from the Rockefeller Family Archives, Rockefeller Center, New York.
4. *After Repeal* reported that "Chief among these" postprohibition regulatory innovations were:
 - "1) the generally adopted practice of separating the package sale of liquor from sale for consumption on the premises,
 - 2) the arrangement for the sale of nonintoxicating beer on a different basis from that governing the sale of wines and spirits,
 - 3) the provision that the on-premises sale of the heavier alcoholic beverages be limited to bona fide restaurants and hotels,
 - 4) the requirement that licensed places be open to the view of the public without the screening devices common to the saloons of preprohibition days and to the speak-easies which later took their place,
 - 5) the recognition of the state's responsibility for supervising the sale of liquor as contrasted to the earlier practice of placing the entire responsibility in the hands of municipal and county officials."
 In addition, they reported that 15 states had already adopted public monopolies for the sale of liquor, and another 25 had established central licensing and regulating authorities.

the investment of capital and the liquor would provide new sources of revenue. It was concluded that in the thirties, "government law, either singly or in combinations for repeal" (p. 275). In this concern with lawlessness, as I say, the argument has been mentioned with law legitimacy was neglected or the concern about lawlessness was the main concern of alcohol control policy. For arguments for repeal and the economic impact; Jones (1961); Englemann (1979);

Members of the AAPA had argued that the liquor industry would provide personal and corporate income. They widely distributed pamphlets arguing that the industry would bring in one billion dollars a year and could, in fact, eliminate the federal deficit. In the 1930s they found a number of economic arguments. State and local governments, faced with severe drops in tax revenues for services, found the economic arguments. Further, during the depression many in the liquor industry would put men back to work.

and from John D. Rockefeller Jr., who was cited or drawn upon for this study. Family Archives, Rockefeller Center,

chief among these" postprohibition

the idea of separating the package sale of liquor from the premises,

the sale of nonintoxicating beer on a different level of wines and spirits,

the direct sale of the heavier alcoholic beverages at restaurants and hotels,

places be open to the view of the devices common to the saloons of the 1920s, the "speakeasies" which later took their

responsibility for supervising the sale of liquor. The earlier practice of placing the entire responsibility on municipal and county officials."

5 states had already adopted public control, and another 25 had established local authorities.

5. By and large, this is achieved through careful policing coupled with the power to revoke or suspend licenses. Operating a liquor-selling business is usually quite profitable, compared to other kinds of retail establishments, and owners guard their money-making licenses carefully. The one obvious, flagrant exception to the usual successful enforcement of and obedience to liquor regulation is on minimum drinking age laws—which, it should be noted, are one of the remaining forms of prohibition.
6. Though some state alcohol control laws composed around 1934 paid lip service to furthering "temperance," alcohol control systems have never pursued such an aim. Indeed, it is not even clear whether state alcoholic beverage control agencies (ABCs) are capable of incorporating public health concerns in any way but as an issue of business regulation. The system turns all questions into administrative matters with a focus on public order. The Rockefeller reports discouraged earmarking any alcohol tax revenues for educational or public health purposes, especially alcohol-related ones, and nearly all state legislatures took the advice. Further, the alcohol beverage industry, led by the corporate manufacturers which support the trade organizations, exercises substantial power in state governments and in Washington. Thus, for example, the industry has been able to stop all efforts to place warnings on bottles, or even to get producers to list ingredients.

- References**
- Asbury, Herbert. *The Great Illusion: An Informal History of Prohibition*. Garden City, N.Y.: Doubleday, 1950.
- Association Against the Prohibition Amendment (AAPA). Various pamphlets. Washington, D.C., 1929-31.
- Baden, Anne L., comp. *Selected List of Writings on the Control of the Liquor Traffic in the United States and Foreign Countries*. Washington, D.C.: Library of Congress, Division of Bibliography, 1933.
- Billings, John S.; Eliot, Charles W.; Farnam, Henry W.; Greene, Jacob L.; and Peabody, Francis G. *The Liquor Problem: A Summary of Investigations Conducted by the Committee of Fifty, 1893-1903*. Boston: Houghton, Mifflin and Company, 1905.
- Business Week*. "Wall Street Cheers Repeal." July 22, 1933, p. 20.
- Cassidy, James E. "Liquor Control." *The Commonwealth*, February 9, 1934, pp. 406-08.
- Catlin, George E. G. *Liquor Control*. New York: Henry Holt and Company, 1931.
- Cherrington, Ernest H. *The Evolution of Prohibition in the United States of America*. Westerville, Ohio: The American Issue Press, 1920.

- . *Standard Encyclopedia of the Alcohol Problem*. 6 vols. Westerville, Ohio: American Issue Publishing Company, vol. 1, 1923; vol. 2, 1924; vol. 3, 1926; vol. 4, 1928; vol. 5, 1929; vol. 6, 1930.
- Childs, Randolph W. *Making Repeal Work*. Philadelphia: Pennsylvania Alcoholic Beverage Study, Inc., 1947.
- Clark, Norman H. *The Dry Years: Prohibition and Social Change in Washington*. Seattle: University of Washington Press, 1965.
- . *Deliver Us From Evil: An Interpretation of American Prohibition*. New York: W.W. Norton & Company, 1976.
- Committee on Liquor Control Legislation of the National Municipal League. "Liquor Control: Principles Model Law." Supplement to *National Municipal Review* 23 (January 1934):47-78.
- The Commonwealth*, "The Rockefeller Report." October 27, 1933, pp. 603-04.
- Culver, Dorothy Campbell, and Thomas, Jack E. *State Liquor Control Administration: A Statutory Analysis*. Berkeley, Cal.: University of California, Bureau of Public Administration, 1940.
- Davis, Elmer. "How the Wets Won." *Current History*, December 1933, pp. 276-84.
- Dobyns, Fletcher. *The Amazing Story of Repeal: An Expose of the Power of Propaganda*. 1940. Reprint. Evanston, Ill.: Signal Press, 1974.
- Domhoff, G. William. *The Higher Circles: The Governing Class in America*. New York: Vintage Books, 1970.
- Dorr, Reta Childe. *Drink: Coercion or Control?* New York: Frederick A. Stokes Company, 1929.
- Engelmann, Larry. "Organized Thirst: The Story of Repeal in Michigan." In *Alcohol, Reform and Society: The Liquor Issue in a Social Context*, edited by Jack S. Blocker Jr. Westport, Conn.: Greenwood Press, 1979.
- Fosdick, Raymond B. *John D. Rockefeller, Jr.: A Portrait*. New York: Harper & Row, 1956.
- Fosdick, Raymond B., and Scott, Albert L. *Toward Liquor Control*. New York: Harper & Brothers Publishers, 1933.
- Foucault, Michel. *Discipline and Punish: The Birth of the Prison*. New York: Vintage Books, 1979.
- Garrison, W. E. "Fitting the Law to the Lawless." *Christian Century* 50 (Nov. 11, 1933):1505-06.
- Gordon, Ernest. *The Wrecking of the Eighteenth Amendment*. Frances-town, N. H.: Alcohol Information Press, 1943.

f the Alcohol Problem. 6 vols. Western Publishing Company, vol. 1, 1923; vol. 4, 1928; vol. 5, 1929; vol. 6, 1930.

Repeal Work. Philadelphia: Pennsylvania Inc., 1947.

Prohibition and Social Change in University of Washington Press, 1965.

Interpretation of American Prohibition. Company, 1976.

Legislation of the National Municipal Principles Model Law." Supplement to 23 (January 1934):47-78.

eller Report." October 27, 1933, pp.

Thomas, Jack E. *State Liquor Control Analysis*. Berkeley, Cal.: University of Administration, 1940.

" *Current History*, December 1933, pp.

Story of Repeal: An Expose of the Power nt. Evanston, Ill.: Signal Press, 1974.

her Circles: The Governing Class in e Books, 1970.

n or Control? New York: Frederick A.

rst: The Story of Repeal in Michigan." *Society: The Liquor Issue in a Social* Blocker Jr. Westport, Conn.: Green-

ockefeller, Jr.: *A Portrait*. New York:

Albert L. *Toward Liquor Control*. New blishers, 1933.

Punish: The Birth of the Prison. New

o the Lawless." *Christian Century* 50

the Eighteenth Amendment. Frances- ation Press, 1943.

Gulick, Luther. Letter to Richard S. Childs, copy to Laurance S. Rockefeller. Rockefeller Archives, May 2, 1977.

Gusfield, Joseph R. *Symbolic Crusade: Status Politics and the American Temperance Movement*. Urbana, Ill.: University of Illinois Press, 1963.

—"Prohibition: The Impact of Political Utopianism." In *Change and Continuity in Twentieth Century America: The 1920s*, edited by John Braeman. Columbus: Ohio State University Press, 1968, pp. 257-308.

Hacker, Louis M. "The Rise and Fall of Prohibition." *Current History*, September 1932, pp. 662-72.

Harrison, Leonard V., and Laine, Elizabeth. *After Repeal: A Study of Liquor Control Administration*. New York: Harper & Brothers Publishers, 1936.

Johnsen, Julia E. *Selected Articles on the Problem of Liquor Control*. The Handbook Series. New York: H.W. Wilson Company, 1934.

Joint Committee of the States to Study Alcoholic Beverage Laws. *Alcoholic Beverage Control: Administration, Licensing and Enforcement*, revised and updated by Benjamin W. Corrado. Washington, D.C., 1973.

Jones, Bartlett Campbell. "The Debate Over National Prohibition: 1920-1933." Ph.D. dissertation, Emory University, 1961.

Kobler, John. *Ardent Spirits: The Rise and Fall of Prohibition*. New York: G.P. Putnam's Sons, 1973.

Kolko, Gabriel. *The Triumph of Conservatism: A Reinterpretation of American History, 1900-1916*. New York: The Free Press, 1963.

Kyvig, David E. *Repealing National Prohibition*. Chicago: The University of Chicago Press, 1979a.

—"Objection Sustained: Prohibition Repeal and the New Deal." In *Alcohol, Reform and Society: The Liquor Issue in a Social Context*, edited by Jack S. Blocker, Jr. Westport, Conn.: Greenwood Press, 1979b.

Leuchtenburg, William E. *The Perils of Prosperity 1914-32*. Chicago: The University of Chicago Press, 1958.

Levine, Harry Gene. "The Discovery of Addiction: Changing Conceptions of Habitual Drunkenness in America." *Journal of Studies on Alcohol* 39 (1978):143-174.

—"Temperance and Women in 19th Century America." In *Alcohol and Drug Problems in Women*, edited by O. Kalant. Research Advances in Alcohol and Drug Problems, vol. 5. New York: Plenum Press, 1980.

———. "The Committee of Fifty and the Origins of Alcohol Control." *Journal of Drug Issues* 13 (1983):95-116.

———. "The Alcohol Problem in America: From Temperance to Alcoholism." *British Journal of Addiction* 79 (1984):109-19.

Levine, Harry Gene, and Smith, David C. "A Selected Bibliography on Prohibition and Alcohol Control." Working Paper F-71, Social Research Group, School of Public Health. Berkeley: University of California, 1977.

Literary Digest. "The Rockefeller Study of Liquor Control." October 28, 1933, p. 10.

Manchester, William. *The Glory and the Dream: A Narrative History of America 1932-1972*. Boston: Little, Brown & Company, 1974.

Merz, Charles. *The Dry Decade*. Seattle: The University of Washington Press, 1970.

The Nation. "John D. Rockefeller, Jr., Does a Fine Job." November 8, 1933, p. 539.

———. "Liquor's New Problems." November 22, 1933, p. 581.

National Commission on Law Observance and Enforcement, George W. Wickersham, Chairman (Wickersham Commission). *Report on the Enforcement of the Prohibition Laws of the United States*. H.R. 722, 71st Cong., 1st Sess. Washington, D.C.: U.S. Government Printing Office, January 7, 1931.

The New York Times. "John D. Rockefeller Jr. out for Repeal; Says Dry Law Evils Outweigh Benefits" (and other articles). June 7, 1932, pp. 1 ff.

———. "Leaders of 2,500,000 Wets Unite to Fight for Repeal; Spurred by Rockefeller" (and other articles). June 8, 1932, pp. 1 ff.

———. "Dr. John R. Mott Comes out for Dry Law Resubmission" (and other articles). June 9, 1932, pp. 1 ff.

Odegard, Peter H. *Pressure Politics: The Story of the Anti-Saloon League*. New York: Columbia University Press, 1928.

Review of Reviews. "The Lawless American." July 1929, p. 64.

Rockefeller, John D., Jr. Miscellaneous correspondence on prohibition and related issues. Rockefeller Archives, Rockefeller Center, New York, N.Y., 1920s-1930s.

Sheppard, Mrs. John S. "After Five Years, What Has Repeal Achieved?" *The New York Times Magazine*, December 4, 1938, p. 5.

Shipman, George A. "State Administrative Machinery for Liquor Control." *Law and Contemporary Problems* 7, no. 4 (1940):600-20.

fty and the Origins of Alcohol Control." 3 (1983):95-116.

in America: From Temperance to Alcohol-Addiction 79 (1984):109-19.

h, David C. "A Selected Bibliography on Alcohol Control." Working Paper F-71, Social Science of Public Health. Berkeley: University of California, 1971.

er Study of Liquor Control." October 28, 1932.

y and the Dream: A Narrative History of Prohibition. Little, Brown & Company, 1974.

e. Seattle: The University of Washington Press, 1971.

ller, Jr., Does a Fine Job." November 8, 1932.

s." November 22, 1933, p. 581.

Observance and Enforcement, George W. Wickersham Commission). *Report on the Prohibition Laws of the United States*. H.R. 600. Washington, D.C.: U.S. Government Printing Office, 1931.

Rockefeller Jr. out for Repeal; Says Dry Efforts" (and other articles). June 7, 1932, pp. 1 ff.

ets Unite to Fight for Repeal; Spurred by Articles). June 8, 1932, pp. 1 ff.

es out for Dry Law Resubmission" (and other articles). June 12, 1932, pp. 1 ff.

ics: *The Story of the Anti-Saloon League*. University Press, 1928.

s American." July 1929, p. 64.

llaneous correspondence on prohibition in the Rockefeller Archives, Rockefeller Center, New York.

ive Years, What Has Repeal Achieved?" *Time*, December 4, 1938, p. 5.

ministrative Machinery for Liquor Control Problems 7, no. 4 (1940):600-20.

Sinclair, Andrew. *Era of Excess: A Social History of the Prohibition Movement*. New York: Harper & Row, Publishers, 1964.

Smith, Fred B., ed. *Law Versus Lawlessness*. Addresses delivered at the Citizenship Conference, Washington, D.C., Oct. 13, 14, 15, 1923. New York: Fleming H. Revell Co., 1924.

Timberlake, James H. *Prohibition and the Progressive Movement: 1900-1920*. New York: Atheneum, 1970.

Waldo, Dwight. *The Administrative State*. New York: The Ronald Press Company, 1948.

———. "Organization Theory: An Elephantine Problem." In *Readings in Public Administration*, edited by Maurice E. O'Donnel. Boston: Houghton Mifflin Company, 1966.

Warburton, Clark. *The Economic Results of Prohibition*. Ph.D. dissertation. New York: Columbia University Press, 1932.

Warren, Harris Gaylord. *Herbert Hoover and the Great Depression*. New York: W.W. Norton & Company, 1967.

Wiebe, Robert H. *The Search for Order: 1877-1920*. New York: Hill and Wang, 1967.

Williams, Whiting. "Yes, My Workers Are Drier, But—" *The Survey Graphic* 68 (June 1, 1932):213.

Wolfskill, George. *The Revolt of the Conservative: A History of the American Liberty League, 1934-1940*. Boston: Houghton Mifflin Company, 1962.

Children of Alcoholics
Facts and Recommendations
compiled by
Cindy Scales and Joyce Schwettman

FACTS

The following facts about Children of Alcoholics (COA's) have been established:

- One out of four children in every classroom has at least one parent with a drinking problem.* (1, 2)
- One out of every eight americans is the offspring of a parent who has a problem with alcohol. Nearly seven million of these people are children under 18.* (3)
- Native students drop out of high school at a higher rate due to family alcoholism. (4)
- 95 out of 100 children of alcoholics never receive professional help. (5, 6)
- 36% of the children of alcoholic families are heavy abusers of alcohol. In families who had serious problems other than alcoholism, only 16% of the children were inclined to abuse alcohol. (7)
- 50% of today's alcoholics are the children of alcoholics. (8)
- In 1982 the Gallop poll found that 40% of young people between 18 and 24 had a family member with an alcohol problem. (9)
- In up to 90% of child abuse cases, alcohol is a significant factor. (10)
- In the 1970's an estimated 28 million Americans had at least one alcoholic parent. (11)
- One of three families currently reports alcohol abuse by a family member. (12)
- The majority of people served by Employee Assistance Programs are Adult Children of Alcoholics. (13)
- Children of alcoholics are at the highest risk of developing alcoholism themselves or marrying someone who becomes alcoholic. (14)

*Note the difference between these two figures are due to families where alcoholics have more than one child.

- Children of alcoholics are frequently victims of incest, child neglect, and other forms of violence and exploitation. (15)
- Medical research has shown that children born to alcoholics are at the highest risk of developing attention deficit disorders, stress-related medical problems, fetal alcohol syndrome, and other alcoholic-related birth defects. (16)
- Children of alcoholics are prone to experience a range of psychological difficulties, including learning disabilities, anxiety, attempted and completed suicide, eating disorders and compulsive achieving. (17)
- The problems of most COA's remain invisible because their coping behavior tends to be approval seeking and socially acceptable. However, a disproportionate number of those entering the juvenile justice system, courts, and prisons, mental health facilities, and referred to school authorities are COA's. (18)

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1. Robert J. Ackerman, Children of Alcoholics. Holmes Beach, FL: Learning Publications, Inc., 1978.
 2. Joanne M. Pilat, A.C.S.W., John W. Jones, Ph.D., "Alcohol, Health and Research World," Winter 1984/1985.
 3. Report of the Conference on Research Needs and Opportunities for Children of Alcoholics. Children of Alcoholics Foundation, Inc., New York, April 1984.
 4. Herbert Gravitz and Julie D. Bowen, Guide to Recovery. Learning Publications, Inc.
 5. Alcohol & Health. U.S. Dept. of Health and Human Services. Washington: Government Printing Office, 1981.
 6. National Institute on Alcohol Abuse and Alcoholism. Fourth Special Report to the U.S. Congress on Alcohol and Health from the Secretary of Health and Human Services. January 1981. De Luca, J.R., ed. DHHS Pub. No. (ADM)81-1080. Washington. DC: Supt. of Docs., U.S. Govt. Print. Off., 1981
 7. Dorothy Miller, Children of Alcoholics: A Twenty-year Longitudinal Study. (San Francisco: Institute for Scientific Analysis, 1977)
 8. Ibid.
 9. Gallop Poll Information Service, 1982
 10. The National Association on Children of Alcoholics Network, Charter Statement Winter 1984, Vol 1, Number 1.
 - 11-18. Ibid.

RECOMMENDATIONS

- A safe place in every community for children to go when home life is unbearable.
- Support groups in schools so transportation is not a problem.
- Special programs on alcohol/drug and life skills education.
- All adults caring for COA's, be their physicians, nurses, preschool workers, teachers, or neighbors, must be taught to identify and help these children of alcoholics.

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