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The first published analysis of the price elasticity of cocaine demand is by DiNardo (1993) using state cross-sections aggregated from the 1977–87 Monitoring the Future (MTF) surveys. DiNardo (1993) finds that past month cocaine participation by high school seniors is not related to the price of cocaine, and that neither cocaine demand nor price are related to Drug Enforcement Administration (DEA) cocaine seizures.

Caulkins (1996) indirectly estimates cocaine participation price elasticities from estimates of the price elasticity of the percent arrestees testing positive for cocaine. Invoking conservative assumptions about the fractions of drug users testing positive and of arrests attributable to drug use, drug spending, and other causes, his estimated elasticities range between −1.48 and −2.08.

Two 1999 studies by Saffer and Chaloupka (1999a, 1999b) estimate past year participation equations for cocaine and marijuana in pooled data from 1988, 1990, and 1991 NHSDA respondents age 12 and older. Averaging across five specifications, Saffer and Chaloupka (1999a) estimate a cocaine price elasticity of −0.40 and a positive relationship between marijuana use and living in a state where marijuana is decriminalized. Estimated cross-price and cross-decriminalization effects indicate complementarity between cocaine and marijuana. Saffer and Chaloupka (1999b) divide the same sample into seven demographic groups and estimate past year participation elasticities for each. The estimated cocaine price elasticity is insignificant for blacks and Asians, −1.83 for Native Americans, and between −0.5 and −0.8 for white males, Hispanics, women, and youth. Marijuana decriminalization is positively related to marijuana participation for all groups except Native Americans. However, cross-price and cross-decriminalization relationships are each insignificant for six groups, with each indicating complementarity for the remaining group. A major limitation of these studies is that they do not control for fixed state or regional effects.

Chaloupka et al. (1999) estimate equations for past year cocaine and marijuana participation by high school seniors in the 1982 and 1989 MTF surveys. The estimated cocaine price elasticity is −0.88 in the combined sample but only −0.24 in the 1989 sample. The impact of cocaine possession fines is also negative in 1989. Similarly, decriminalization increases marijuana use and increases in marijuana possession fines decrease marijuana use. Fines for sales do not impact the use of either drug.

Finally, Grossman and Chaloupka (1998) use a rational addiction framework, in which current consumption depends on both past and future consumption, to estimate price elasticities of past year cocaine participation among 18- to 27-year-olds in the 1976–85 MTF panels and associated follow-ups. In a model with person-specific fixed effects, estimated short- and long-run price elasticities are −0.42 and −0.54, respectively. The analogous model without these fixed effects yields short- and long-run elasticities of −0.72 and −1.40. Marijuana decriminalization increases cocaine use, again implying that cocaine and marijuana are complements.

Because of deficient marijuana price data, only two previous studies have estimated the elasticity of marijuana demand with respect to its price.[2] Nisbet and Vakil (1972) surveyed UCLA students regarding quantities of marijuana purchased at current prices and after various hypothetical price changes. Their estimated price elasticities ranged from −0.40 to −1.51. The only study to use actual price data is Pacula et al. (2001) in 1985–96 MTF data on high school seniors. Controlling for state effects, their estimated past year participation elasticity is −0.33 when time effects are omitted but only −0.06 when time is entered quadratically.

our analysis to drug participation for various reasons related to data quality. First, the only frequency measures available in the NHSDA are the number of days in the past month, which is a continuous variable but does not match the time frame of our participation variable, and frequency of use in the past year, which is a categorical variable with intervals ranging from "1–2 days in the past year" to "3–6 days per week." Second, frequency measures are more likely to be affected by recall error than the binary participation decision. Third, because few respondents report using illegal drugs, particularly cocaine, the sample sizes for analyses of frequency conditional on participation are small.

2. Our review of marijuana decriminalization effects covers only those studies that analyze cocaine demand. Pacula et al. (2001) summarize the extensive literature examining the effect of decriminalization on marijuana demand, which has yielded mixed results. We cannot empirically identify decriminalization effects because we control for state-fixed effects and no state has changed its decriminalization policy in the past decade.

The only study besides DiNardo (1993) to directly examine the effect of drug enforcement on drug consumption is Farrelly et al. (2001). Controlling for state fixed effects in 1990–96 NHSDA panels, they find that increases in the probability of arrest for marijuana possession decrease the probability that 12–20-year-olds will use marijuana. Meanwhile, using annual data on 29 large U.S. cities from 1981–95 and controlling for fixed city and year effects, DeSimone (2001) estimates a positive relationship between the price of cocaine and the number of cocaine purchases per capita by undercover drug enforcement agents. Further results indicating a negative relationship between crime rates and cocaine prices suggest that cocaine consumption is price-elastic.

In sum, cocaine consumption appears to respond to cocaine price changes, but the range of elasticity estimates is wide, and all except those from the Grossman and Chaloupka (1998) models that include individual fixed effects come from models that fail to control for geographic fixed effects.[3] Few studies have examined the relationship between drug demand and either marijuana prices or intensity of drug possession law enforcement.

III. DATA

This study analyzes pooled 1990–97 cross-sections from the NHSDA, administered annually to a nationally representative sample of the U.S. household population aged 12 and higher.[4] We restrict our sample to respondents aged 39 and younger, because the overwhelming majority of illegal drug use occurs in this age group,[5] and conduct separate analyses for those aged 12–17 and 18–39 because of the different treatments of these two age groups by the legal system. In that spirit we henceforth refer to the former group as juveniles and the latter as adults.

Dependent Variables

We specify two dependent variables, one corresponding to cocaine use and the other to marijuana use. Both are binary indicators of past year participation in drug consumption.[6]

Cocaine Price

A measure of the average cocaine price per pure gram faced by the respondent is appended to the NHSDA data.[7] Cocaine price data are collected by undercover drug agents, mostly from the DEA, and recorded by the DEA in their System to Retrieve Information from Drug Evidence (STRIDE). These prices are expected to be relatively accurate because an unreasonable price offer by an agent would invoke suspicion by the seller and thus endanger the agent. Transaction sizes must be standardized because, as Caulkins and Padman (1993) and Rhodes et al. (1994) show, sizable quantity discounts exist for cocaine.

We impute cocaine prices from STRIDE data using the ordinary least squares regression

$$\log Price = b_0 + b_1(\log Predicted\ Purity + \log Weight) + b_2 Area + b_3 Year + b_4 Area \times Year + w,$$

where purity represents the weight of actual cocaine in the purchase divided by the total weight, *Predicted Purity* is the predicted value from a regression of purity on the other

3. As long as respondents do not move, geographic fixed effects drop out of an individual fixed effects model because they are time-invariant.

4. The NHSDA excludes individuals with no permanent residence and who reside in group quarters, categories that include the homeless and residents of homeless shelters, prisons, and college dormitories. Although drug use among NHSDA respondents is thus likely to be disproportionately low relative to the U.S. population, the excluded group represents only about 1% of U.S. residents.

5. For instance, in 1997, the fraction of respondents reporting past year cocaine (marijuana) use is 2.2% (15.8%) among 12–17-year-olds, 3.6% (17.1%) among 18–39-year-olds, and 0.7% (3.2%) among 40+-year-olds. The specific choice of age 39 as the cutoff corresponds to the 5-year age groupings reported by SAMHSA (1997).

6. NHSDA interviews are conducted throughout the year, so that the time interval implied by "past year" varies from almost entirely the previous calendar year for January interviews to almost entirely the current calendar year for December interviews. Because the price variables represent the current calendar year and the consumption time frame extends to the previous calendar year, the time intervals for the price and consumption variables do not strictly match. This is done for simplicity, imitates the practice of many previous studies, and should not affect the results much because current and past year prices are highly correlated.

7. All nominally valued variables are converted to 1990 dollar terms using the Consumer Price Index for all urban consumers.

explanatory variables, *Weight* is the total gram weight of the purchase, *Area* is a vector of indicators corresponding to 28 geographic areas in the United States that we assume have distinct cocaine prices, *Year* is a vector of year indicators, and *Area* × *Year* represents a complete set of interactions between the *Area* and *Year* vectors. For a particular area and year our predicted cocaine price is $\exp(b_0 + b_2 + b_3 + b_4)$, which represents the median price of one gram of 100% pure cocaine in the area and year with corresponding indicators set equal to one in the *Area* and *Year* vectors. We follow Caulkins (1994) in excluding outliers before estimating equation (1).[8]

Our methodology assumes that prices vary by census region or division and metropolitan statistical area (MSA) size. Appendix Table A-1 lists the census regions and divisions and the states encompassed by each. MSAs containing more than 1 million people are grouped together within each census division. This creates eight distinct areas, because the East South Central division contains no MSAs of this size. Similarly, MSAs containing between 500,000 and 1 million people are grouped together within each division, creating nine additional areas. Because there are fewer STRIDE observations from less populated areas, we divide the South census region into the South Atlantic and South Central divisions, which partitions the United States into three census regions and two census divisions. Within each of these five regions, grouping together MSAs containing between 250,000 and 500,000 people and MSAs/rural areas containing fewer than 250,000 people creates 10 additional price areas. Finally, the District of Columbia is considered a separate area because abundant STRIDE data are collected there. In each year, therefore, there are 28 different predicted prices corresponding to 28 geographic areas.[9] Each NHSDA respondent is assigned the cocaine price for their area of residence among these 28.

This methodology differs in two important ways from that used by previous researchers. The first is that previous methodologies impute city-level prices by including city rather than area indicators in equation (1) and then calculate a state-level price as the population-weighted average of prices from each city represented in STRIDE from each state. This ignores evidence from Rhodes et al. (1994) and Caulkins (1995) that prices increase as the distance from points of entry into the United States increases, because of lateral transaction costs, and as market size decreases, because of economies of scale associated with distribution. For example, therefore, the price of cocaine in Greenville, North Carolina, is likely to be closer to the price in Charlottesville, Virginia, which is in a different state but the same census division and population category, than that in Charlotte, North Carolina, which is in the same state but has a much larger population.

The second departure of our method is that previous methods have not included an interaction vector analogous to our *Area* × *Year* interaction vector. This is because data are insufficient to identify a complete set of

8. STRIDE contains prices for 35,674 cocaine purchases made during 1990–97. We begin by eliminating the 780 observations with purity less than or equal to .001% or greater than 100%, because these values represent either data errors or purchases containing trivial amounts of the actual drug. Then we estimate an initial price standardized by total weight as the predicted price from a regression of log *Price* on log *Weight* and *Year*. For each year, we throw out observations with standardized prices that are less than one-eighth of or greater than eight times the mean price for that year. We then recompute the mean for each year and throw out observations with standardized prices that are less than one-fourth of or greater than four times the mean price for that year. Finally we calculate an alternative price per pure gram from observed values of price, weight, and purity and eliminate observations with a price per pure gram of greater than $3,000. Overall these latter three steps eliminate only an additional 546, or 1.6%, of the remaining STRIDE observations. Estimated price effects are slightly less precise but otherwise quite similar to those reported here when these latter 546 STRIDE observations are not eliminated.

9. Not surprisingly, the number of STRIDE observations in each price region varies greatly across both population size and census division. Washington, DC, has the largest sample size, averaging 746 observations annually during the analysis period. Average annual sample sizes range from 102 to 435 for the remaining 8 areas formed from populations greater than 1 million, from 30 to 215 for MSAs with between 500,000 and 1 million people, from 23 to 94 for MSAs with between 250,000 and 500,000 people, and from 17 to 130 for areas with less than 250,000 people. The lower ends correspond to the West North Central division for the two larger population sizes and to the Northeast for the two smaller population sizes, whereas the higher ends correspond to the South Atlantic for all population categories. The sample size is less than 10 in only three cases, all in the Northeast for the smaller population categories, with a minimum of four. In the aggregate, the temporal variation is reasonably small, with the average number of STRIDE observations per area ranging from 106 in 1993 to 196 in 1991.

interaction terms when prices are estimated for individual cities rather than broader areas. An advantage of our methodology is that it allows for geographic variation in intertemporal price changes, making detection of price effects in drug demand equations possible even when state fixed effects are included.

For more direct comparison with estimates from previous studies, we also obtain results with a state-level cocaine price measure. The regression used to construct this variable is equation (1) with *Area* representing a vector of state indicators, rather than of the 28 price regions described, and b_4 set to zero. Models estimated with the state-level cocaine price measure exclude state fixed effects, which are no longer identified.

Marijuana Price

A measure of the price of marijuana faced by the respondent is also appended to the NHSDA data. Because DEA agents concentrate on cocaine and heroin trafficking, STRIDE contains insufficient marijuana price information with which to construct regional price estimates. We instead follow Pacula et al. (2001) by constructing a marijuana price estimate from prices listed in the *Illegal Drug Price/Purity Report*, a publication of the DEA Intelligence Division. This report contains quarterly estimates of the minimum and maximum marijuana price in 19 cities for both pound and ounce purchases of two different types of marijuana, commercial-grade and sinsemilla-grade.

Our price variable is the average price per gram of a pound purchase of commercial grade marijuana. We choose the pound-level commercial series because it is by far the most complete of the four series.[10] For each city, a quarterly price is obtained by taking the midpoint of the reported price range for each quarter, and an annual price is obtained by averaging all quarterly prices that are reported for that year.[11]

Our NHSDA data indicate the state of residence of each respondent but nothing more specific regarding location of residence. Of the 19 cities for which marijuana prices are constructed, 14 are the only city reporting a price in that state. Each of these states is assigned the price of its reporting city. Annual prices for the two cities in Texas and the three cities in California are averaged in each year to form annual prices for those states. Each remaining state of residence in the NHSDA, which sampled all states except Vermont and Wyoming during 1990–97, is assigned the price from the closest of these 16 states that is in the same census division as the state. Thus the United States is divided into 16 regions, each of which is assigned a different marijuana price series. Appendix Table A1 lists these 16 regions according to the cities that contribute prices and the states that are assigned the price from each region.

Cocaine and Marijuana Enforcement

Variables representing the probability of arrest for cocaine and marijuana possession violations in the state of residence of the respondent are also appended to the NHSDA data.[12] For each drug, we proxy for the annual possession arrest probability with a variable equaling the number of possession arrests in the state divided by the number of drug users in the state that year. Drug possession arrest information comes from the Uniform Crime Reporting system of the Federal Bureau of

10. Prices of commercial grade marijuana are likely more relevant than those of the more potent sinsemilla variety given evidence from National Narcotics Intelligence Consumers Committee (1998) that commercial marijuana dominates the U.S. market. In contrast, because purchases of a pound likely represent wholesale purchases by low-level dealers whereas purchases of an ounce likely represent retail purchases by a typical user, in principle ounce-level prices are more appropriate for the analysis than pound-level prices. Results from Pacula et al. (2001) indicate that multiplying the estimated marijuana price regression coefficients by 0.5 yields a rough estimate of the response of drug demand to retail price changes. This is because $\partial d/\partial r = (\partial d/\partial w)(\partial w/\partial r)$—where d is drug use, r is the retail price, and w is the wholesale price—the estimated marijuana demand regression coefficient is $\partial d/\partial w$, and their estimated $\partial w/\partial r$ from a regression of the wholesale price on the retail price is 0.5.

11. This typically represents all four quarters because pound-level commercial-grade prices are rarely missing. Also, we cannot control for potency because only one national potency average is reported for each year.

12. A concern regarding the price and enforcement variables is that they measure local prices and enforcement levels faced by respondents with error. If these measurement errors are random, estimated price and enforcement effects are biased toward zero. The extent to which measurement error plagues the estimates, however, is mitigated by fact that NHSDA responses, price observations, and arrests all disproportionately occur in large population centers.

Investigation (FBI).[13] Because there are no published estimates of the number of drug users by state, we construct the denominator by multiplying the unweighted percentage of state respondents who use the drug by the 1 July Census estimate of the state population.[14] The logic behind these arrest rate variables is that the probability of apprehending a given criminal falls as the level of police resources devoted to enforcing the corresponding crime falls, and, as Ehrlich (1973) argues, the number of criminals rises.

However, because the denominator of the arrest rate variable is by construction positively correlated with the dependent variable, the effect of the arrest rate for a drug on the demand for that drug is likely to be overestimated in magnitude for two reasons. First, the variable is statistically endogenous. In particular, reverse causality may result in a reduction in the arrest probability from an increase in the number of users. Second, errors in measurement of the number of users are negatively correlated with errors in measurement of the arrest rate. Overestimates of the number of users, as expected in states with lower than average true drug use rates, generate underestimates of arrest rates, with the opposite occurring in states with higher than average true drug use rates. If the actual elasticity of the number of drug users with respect to arrest rates is less than one in absolute value, measurement error reduces observed arrest rate differences across states by a greater proportion than observed drug use rate differences, making smaller changes in arrest rates appear to induce larger changes in drug use.[15]

To reduce the extent of the endogeneity and measurement error problems, each arrest rate variable is constructed using all ages in both the numerator and denominator rather than separately for each age group.[16] Furthermore, we also obtain results using alternative arrest probability variables that instead specify total type I arrests in the state, which are also reported by the FBI, as the denominator.[17] Previous studies such as Benson and Rasmussen (1991) use this variable to measure enforcement of drug crimes relative to nondrug crimes. The advantage of this variable is that it is neither statistically endogenous by construction nor affected by errors in measurement of the number of drug users. The disadvantage is that it is a less direct proxy for the arrest probability, because the denominator is not a measure of drug consumption activity. For example, if the number of cocaine arrests and users increase by identical proportions while the number of type I arrests does not change, this arrest rate measure increases even though the percentage of cocaine users arrested, a much closer proxy for the probability of arrest for cocaine possession, does not.

Other Explanatory Variables

We control for a variety of additional explanatory variables. Other continuous measures include age and age squared, family size, and real family income. The remaining variables are indicators for males, blacks, Hispanics, other races, marriage, divorce, enrollment in school, four levels of educational attainment (high school graduate, some college, college graduate, some graduate school), the interview not being interrupted,[18] whether the interview interruption variable is missing, three MSA population categories (population greater than 1 million, between 500,000 and 1 million, and between 250,000 and 500,000), and rural residence. Age squared and indicators for divorce, college completion, and graduate

13. The cocaine arrest variable numerator represents total arrests for possession of cocaine and opium derivatives, because the FBI does not separately report cocaine and opium-related arrests.

14. This denominator is multiplied by the percentage of the state population covered by local agencies from which the FBI obtains arrest data, which is 100% for later years but around 90% on average for earlier years. We also considered an alternative denominator calculated as the sample-weighted sum of respondents in each state using the drug. Although this measure yields similar results, it is potentially problematic because the NHSDA sample weights are not designed to represent individual states.

15. We thank an anonymous referee for pointing out this measurement error issue, a mathematical derivation of which is available from the authors.

16. Farrelly et al. (2001) constructs this variable for marijuana and estimates similar effects on marijuana use by 12–20-year-olds regardless of whether the variable is calculated only for 12–20-year-olds or encompasses all ages.

17. Type I crimes are the major nondrug crimes: murder, rape, aggravated assault, robbery, burglary, larceny, and motor vehicle theft.

18. The degree of privacy during the interview is coded on a scale from 1 (completely private) to 9 (constant presence of another). Our indicator of no interruptions represents values of 4 or less for this privacy variable.

school attendance are not included in the juvenile regressions.

Sample Characteristics

Table 1 lists separate summary statistics for adults (18–39-year-olds) and juveniles (12–17-year-olds). Marijuana use is considerably more prevalent than cocaine use for both age groups, and use of each is higher among adults than juveniles. The average cocaine price over the period is about $120 per pure gram, which is substantially higher than the average marijuana price of $2.65 per gram.[19] The percentage of users arrested for cocaine possession is about four times the percentage arrested for marijuana possession. However, the arrests per type I arrest measures show that possession arrests occur slightly more frequently for marijuana than for cocaine.

IV. EMPIRICAL METHODOLOGY

Because the dependent variables are binary indicators, the cocaine and marijuana participation equations are estimated with probit regressions. The full model is

$$(2)\quad D_i = b_0 + b_1 P_i^C + b_2 P_i^M + b_3 A_i^C + b_4 A_i^M + b_5 X_i + S_i + Y_i + e_i,$$

where for respondent i, D_i is a binary indicator of past year cocaine or marijuana use; P_i^C and P_i^M are real prices of cocaine and marijuana, respectively; A_i^C and A_i^M are probabilities of arrest for possession of cocaine and marijuana, respectively; X_i is a vector of other relevant socioeconomic variables; S_i is a vector of binary variables representing each state indicating whether or not the respondent lived in the particular state; Y_i is a vector of binary variables for each year from 1990 to 1997 indicating whether or not the respondent was interviewed in the particular year; and e_i is a random error term. All regressions are weighted using the NHSDA sampling weights.

For both cocaine and marijuana use, six variants of equation (2) are estimated separately for both adults and juveniles. The first regression excludes the arrest variables. The second includes only the own-arrest variable, the third includes only the cross-arrest variable, and the fourth includes both arrest variables, which is the complete equation (2) model. Comparing these specifications might reveal the impact of any collinearity between price and arrest variables and between the individual arrest measures. The fifth is identical to the fourth except that it substitutes census division effects for state effects; the sixth entirely excludes fixed geographic effects.

A major departure from the previous literature, other than Farrelly et al. (2001) and Pacula et al. (2001), is the inclusion of state fixed effects. Cocaine prices vary at the regional level, whereas marijuana prices and arrest probabilities vary at the state level. Unobservable state-level factors, such as public willingness to accept alternative behaviors or the political environment, may thus simultaneously affect both illegal drug consumption and the price and arrest variables. The inclusion of state fixed effects controls for variation in unobservable state-level characteristics, eliminating bias-inducing spurious correlation between illegal drug use and the price and arrest variables. Put differently, controlling for state effects reduces the likelihood that differences in cocaine and marijuana use across states resulting from unobserved variation in attitudes and preferences are incorrectly attributed to variation in drug prices and arrest rates.

V. RESULTS

We first report results for cocaine and marijuana use by adults and then proceed to the analogous models for juveniles. Regression sample sizes are 92,784 for adults, 42,464 for juvenile cocaine use, and 43,147 for juvenile marijuana use.[20]

Cocaine Use by Adults

Column (1) of Table 2 shows that when the two arrest variables are excluded from

19. A crack vial contains roughly 0.1 pure gram of cocaine; a line of powder cocaine has about 0.02 pure grams. A marijuana joint typically contains about 0.4 grams of marijuana.

20. In each cell of each table, the first row indicates the change in probability of drug use resulting from a unit change in the explanatory variable, the second row indicates the standard error of the first row estimate (in parentheses), and the third row indicates the elasticity computed from the weighted sample means (in brackets). All regressions include the additional explanatory variables listed in Tables 1 and A2.

TABLE 1
Descriptive Statistics

Variable	Age 18–39 (n = 92,784)	Age 12–17 (n = 43,147)
Cocaine use past year	.044	.015
	(.205)	(.122)
Marijuana use past year	.157	.118
	(.364)	(.322)
Cocaine price per pure gram	118.88	119.54
	(35.12)	(34.98)
Marijuana price per gram	2.653	2.658
	(.789)	(.793)
Cocaine arrests per user	.041	.040
	(.035)	(.035)
Marijuana arrests per user	.011	.011
	(.007)	(.007)
Cocaine arrests per type I arrest	.120	.115
	(.078)	(.077)
Marijuana arrests per type I arrest	.136	.139
	(.073)	(.073)
Interview without interruptions	.822	.712
	(.382)	(.453)
Interview without interruptions—missing	.016	.022
	(.126)	(.148)
Age	29.05	14.49
	(6.28)	(1.68)
Male	.491	.512
	(.500)	(.500)
Black	.122	.143
	(.327)	(.351)
Hispanic	.112	.122
	(.315)	(.328)
Other race	.043	.044
	(.204)	(.205)
Married	.522	.003
	(.500)	(.056)
Divorced	.092	
	(.289)	
Family size	3.50	4.47
	(1.62)	(1.56)
Enrolled in school	.200	.927
	(.400)	(.260)
High school graduate	.351	.013
	(.477)	(.112)
Some college education	.262	.001
	(.440)	(.031)
College graduate	.146	
	(.353)	
Some graduate school education	.075	
	(.263)	
Family income (1,000s)	34.24	30.78
	(23.54)	(24.90)
MSA population > 1 million	.463	.417
	(.499)	(.493)
MSA population between 500,000 & 1 million	.230	.237
	(.421)	(.425)
MSA population between 250,000 & 500,000	.089	.097
	(.284)	(.296)
Rural	.131	.166
	(.338)	(.372)

Notes: Standard deviations are in parentheses. The sample size for past year cocaine use is 42,464. Statistics are weighted using NHSDA sample weights.

TABLE 2
Probit Estimates for Past Year Cocaine Use by 18–39-Year-Olds

Variable	(1)	(2)	(3)	(4)	(5)	(6)
Cocaine price (×1,000)	−.152**	−.132**	−.149**	−.132**	−.113*	−.234***
	(.062)	(.057)	(.061)	(.057)	(.059)	(.061)
	[−.410]	[−.356]	[−.402]	[−.357]	[−.304]	[−.631]
Marijuana price (×100)	.172	−.064	.080	−.074	−.349*	−.335**
	(.232)	(.232)	(.231)	(.231)	(.211)	(.144)
	[.104]	[−.038]	[.048]	[−.044]	[−.210]	[−.202]
Cocaine arrests per user		−.453***		−.430***	−.200***	−.131***
		(.067)		(.071)	(.047)	(.036)
		[−.425]		[−.403]	[−.187]	[−.123]
Marijuana arrests per user			−1.084***	−.252	−.168	−.529***
			(.277)	(.296)	(.232)	(.187)
			[−.266]	[−.062]	[−.041]	[−.130]
Log likelihood	−15,401	−15,326	−15,383	−15,325	−15,447	−15,505
Geographic fixed effects	State	State	State	State	Division	None

Notes: In each cell, the first row indicates the marginal effect on the probability of cocaine use, the second row indicates the standard error (in parentheses), and the third row indicates the implied elasticity (in brackets). The sample size is 92,784. All equations include the variables listed in Appendix Table A2 along with year indicators. Regressions are weighted using NHSDA sample weights.
*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

the equation, adult cocaine demand is significantly negatively related to the cocaine price with an elasticity of −0.41 but is unrelated to the marijuana price.[21] The next three columns reveal that these results are unchanged from adding the arrest variables except for a slight decrease in the cocaine price elasticity when the cocaine arrest rate is included. The cocaine arrest rate has a highly significant elasticity of around −0.4 regardless of whether the marijuana arrest rate is also included. However, the negative effect of the marijuana arrest rate falls by 75% and becomes insignificant when the cocaine arrest rate is also included. This provides weak evidence of complementarity between the two drugs and suggests some collinearity between the arrest rate variables. However, the fact that the arrest rate standard errors inflated only slightly in column (4) indicates that multicollinearity is not too severe. As expected, the elasticity of the cocaine arrest rate is larger than that of the marijuana arrest rate, even without controlling for the former. These patterns are repeated in Tables 3 and 4 and to some extent in Table 5.

21. In all regressions, price coefficients are unchanged when only one of the two price variables is included.

The estimates are from models that control for state fixed effects. When division effects replace state effects in column (5), the marijuana price elasticity increases by a factor of 5 and becomes significant, but the cocaine arrest rate elasticity halves. Omitting geographic fixed effects entirely doubles the cocaine price elasticity and triples the marijuana arrest rate elasticity while further decreasing the cocaine arrest rate elasticity. This pattern is also repeated in Tables 3–5.[22]

Marijuana Use by Adults

Table 3 shows that the cocaine price has a significant negative effect on adult marijuana

22. Throughout Tables 2–5, replacing state effects with division effects has little impact on cocaine price coefficients but large and significant effects on marijuana price coefficients. A possible explanation is that marijuana prices are not measured precisely enough for identification of coefficients in state fixed effects models because they vary primarily across groups of states within census divisions, and in contrast cocaine prices vary both across census divisions and within divisions by metropolitan area size. One might therefore argue that geographic fixed effects are more appropriately specified at the division than the state level. We prefer the more conservative interpretation that state effects models offer for the source of cross-state price variation and argue that differences in arrest rate coefficients between state and division effects models show the importance of controlling for state effects.

TABLE 3
Probit Estimates for Past Year Marijuana Use by 18–39-Year-Olds

Variable	(1)	(2)	(3)	(4)	(5)	(6)
Marijuana price (×100)	.107 (.594) [.018]	−.387 (.536) [−.065]	−.082 (.610) [−.014]	−.412 (.543) [−.070]	−1.699*** (.490) [−.287]	−1.437*** (.335) [−.243]
Cocaine price (×1,000)	−.284** (.136) [−.215]	−.252** (.125) [−.191]	−.264** (.135) [−.199]	−.248** (.125) [−.188]	−.305** (.132) [−.230]	−.314*** (.113) [−.238]
Marijuana arrests per user		−5.871*** (.839) [−.404]		−5.630*** (.853) [−.388]	−4.006*** (.666) [−.276]	−3.969*** (.494) [−.273]
Cocaine arrests per user			−.457*** (.164) [−.120]	−.110 (.125) [−.029]	−.007 (.107) [−.002]	.044 (.085) [.011]
Log likelihood	−36,935	−36,802	−36,907	−36,800	−36,953	−36,987
Geographic fixed effects	State	State	State	State	Division	None

Notes: In each cell, the first row indicates the marginal effect on the probability of marijuana use, the second row indicates the standard error (in parentheses), and the third row indicates the implied elasticity (in brackets). The sample size is 92,784. All equations include the variables listed in Appendix Table A2 along with year indicators. Regressions are weighted using NHSDA sample weights.
*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

TABLE 4
Probit Estimates for Past Year Cocaine Use by 12–17-Year-Olds

Variable	(1)	(2)	(3)	(4)	(5)	(6)
Cocaine price (×1,000)	.005 (.040) [.040]	.012 (.036) [.096]	.006 (.039) [.046]	.013 (.036) [.099]	.020 (.038) [.158]	−.010 (.036) [−.078]
Marijuana price (×100)	−.058 (.136) [−.102]	−.152 (.134) [−.266]	−.074 (.136) [−.129]	−.152 (.133) [−.267]	−.303** (.140) [−.530]	−.268*** (.082) [−.470]
Cocaine arrests per user		−.165*** (.036) [−.437]		−.180*** (.041) [−.475]	−.101*** (.024) [−.268]	−.069*** (.017) [−.183]
Marijuana arrests per user			−.259* (.146) [−.188]	.117 (.160) [.085]	−.061 (.115) [−.045]	−.199* (.106) [−.145]
Log likelihood	−2,915	−2,895	−2,912	−2,894	−2,942	−2,951
Geographic fixed effects	State	State	State	State	Division	None

Notes: In each cell, the first row indicates the marginal effect on the probability of cocaine use, the second row indicates the standard error (in parentheses), and the third row indicates the implied elasticity (in brackets). The sample size is 42,464. All equations include the variables listed in Appendix Table A2 along with year indicators. Regressions are weighted using NHSDA sample weights.
*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

TABLE 5
Probit Estimates for Past Year Marijuana Use by 12–17-Year-Olds

Variable	(1)	(2)	(3)	(4)	(5)	(6)
Marijuana price (×100)	−.007	−.264	−.293	−.389	−1.295**	−.747**
	(.588)	(.582)	(.598)	(.593)	(.519)	(.357)
	[−.002]	[−.060]	[−.066]	[−.088]	[−.292]	[−.169]
Cocaine price (×1,000)	.013	.023	.044	.043	−.011	−.198*
	(.130)	(.126)	(.129)	(.126)	(.121)	(.115)
	[.013]	[.024]	[.045]	[.043]	[−.011]	[−.201]
Marijuana arrests per user		−3.066***		−2.171***	−1.214**	−2.289***
		(.660)		(.659)	(.510)	(.394)
		[−.287]		[−.204]	[−.114]	[−.215]
Cocaine arrests per user			−.573***	−.40[illegible]**	−.352***	−.205**
			(.156)	(.15[illegible])	(.110)	(.094)
			[−.195]	[−.137]	[−.120]	[−.070]
Log likelihood	−13,795	−13,767	−13,767	−13,755	−13,832	−13,875
Geographic fixed effects	State	State	State	State	Division	None

Notes: In each cell, the first row indicates the marginal effect on the probability of marijuana use, the second row indicates the standard error (in parentheses), and the third row indicates the implied elasticity (in brackets). The sample size is 43,147. All equations include the variables listed in Appendix Table A2 along with year indicators. Regressions are weighted using NHSDA sample weights.

*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

demand with a magnitude about half as large as its effect on cocaine demand. However, adult marijuana demand is not related to its own price. The marijuana arrest rate has a strong negative effect regardless of whether the cocaine arrest rate is included, but the cocaine arrest rate effect indicates complementarity only when the marijuana arrest rate is omitted. Removal of state effects again has a large impact. The cocaine price elasticity increases by one-quarter, and the marijuana price elasticity rises by a factor of four and becomes highly significant.

It thus appears that drug demand is higher in states with lower drug prices, but states with lower drug prices have other unobservable characteristics leading to higher drug demand. These unobservable factors are the predominant reason for the negative correlation between marijuana prices and drug use. In contrast, arrest rates for possession of drugs, particularly cocaine, might be higher in states where drug use is greater, whereas additional drug enforcement reduces use within states over time. In other words, state fixed effects control to some extent for the endogenous enforcement response to drug use that is apparent from estimates relying on variation across states at a given time.

Cocaine Use by Juveniles

Table 4 reveals that the main difference between juvenile and adult cocaine demand is that the former is not responsive to cocaine price changes. The effects of the marijuana price and drug arrest rate variables are similar for juveniles and adults. Removal of state effects again inflates the marijuana price elasticity and allows it to attain significance.

Marijuana Use by Juveniles

Table 5 indicates that juvenile marijuana demand is similar to juvenile cocaine demand in that neither price affects demand. The difference from earlier results is that both arrest rates significantly impact juvenile marijuana demand even when both are simultaneously included in the equation, providing more convincing evidence of complementarity between the two drugs. The effect of the marijuana arrest rate is comparable with that found by Farrelly et al. (2001). The impact of removing state and then division effects is consistent with previous results.

Various potential explanations exist for the insignificant effect of the cocaine price on juvenile drug demand despite its significant negative effect on adult drug demand.

TABLE 6
Probit Estimates with State-level Cocaine Price Measure

Variable	Cocaine use (1)	(2)	(3)	Marijuana use (4)	(5)	(6)
18–39-year-olds						
Original cocaine price measure (×1,000)	−.228*** (.061) [−.615]		−.156** (.062) [−.421]	−.309*** (.114) [−.234]		−.315** (.124) [−.239]
State-level cocaine price measure (×1,000)		−.218*** (.055) [−.701]	−.167*** (.058) [−.537]		−.084 (.104) [−.075]	.015 (.112) [.013]
12–17-year-olds						
Original cocaine price measure (×1,000)	−.011 (.037) [−.089]		.013 (.038) [.101]	−.175 (.116) [−.179]		−.135 (.118) [−.138]
State-level cocaine price measure (×1,000)		−.061** (.027) [−.569]	−.065** (.031) [−.605]		−.140 (.111) [−.169]	−.099 (.115) [−.120]

Notes: In each cell, the first row indicates the marginal effect on the probability of cocaine or marijuana use, the second row indicates the standard error (in parentheses), and the third row indicates the implied elasticity (in brackets). The sample size is 92,136 for the top part, 42,179 for cocaine use the bottom half, and 42,794 for marijuana use in the bottom half. These are slightly smaller than in the earlier tables because state-level prices cannot be imputed for several of the least-populous states. All equations include the variables listed in Appendix Table A2 and year indicators but exclude geographic fixed effects. Regressions are weighted using NHSDA sample weights.
*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

First, some juveniles might use their parents' money to purchase cocaine. Second, if as Koch (2000) argues sellers find it profitable to give the drug away to young initiators in an attempt to hook them and charge them higher prices when they become addicts, or if kids share cocaine with their friends, some juvenile users might not pay the market price for cocaine. Third, sample statistics in Table 1 showing that prevalence for adults relative to juveniles is three times as high for cocaine but only one-third larger for marijuana suggests that a substantial proportion of cocaine initiation occurs after age 17. As Kandel and Yamaguchi (1993) report, tobacco is generally initiated before marijuana, which is initiated before cocaine. This could explain why evidence, as reviewed in Chaloupka and Wechsler (1997), generally shows that the cigarette own-price elasticity is higher for teens than adults, and we find the reverse for cocaine.

State-Level Cocaine Price Measure

To evaluate more directly the extent to which our cocaine price construction methodology accounts for the difference between our results and those of previous studies, Table 6 presents results of models in which the state-level cocaine price measure described earlier is used in place of our preferred cocaine price measure. For each age group and drug type, we estimate three regressions specified identically to column (6) of Tables 2–5: one with our price measure, one with the state-level price measure, and one that includes both price measures.[23] These models exclude geographic fixed effects because state effects are not identified when the state-level price measure is included. For cocaine, the state-level price has a similar effect on adult use and a stronger negative effect on juvenile use than does our price measure. But in both cases, when the state-level variable is included, the effects of our price variable are nearly identical to those with state effects included, as indicated in column (4) of Tables 2 and 4. This suggests that state fixed effects are effectively embedded into the state-level price

23. The coefficients of the models including only our original price variable are slightly different than those shown in column (6) of Tables 2–5 because the Table 6 regression samples exclude respondents from a few low-population states for which insufficient data exist to construct state-level prices and are thus slightly smaller.

series, so that at least some of the estimated state-level price coefficients represent fixed state effects rather than the true impacts of price changes. Indeed, the R^2 from a regression of the cocaine price on a set of state indicators is 0.30 for our price measure but 0.61 for the state-level measure. Meanwhile, for marijuana, our price measure has a stronger effect on adult use and a very similar effect on juvenile use.

Alternative Arrest Measure

Table 7 shows results for the alternative arrest rate variable that uses total type I arrests as the denominator. The cocaine arrest rate has a consistent significant negative effect on both cocaine and marijuana demand for both adults and juveniles regardless of whether the marijuana arrest rate is also controlled for, with own-arrest elasticities slightly larger than cross-arrest elasticities. In contrast, the impact of the marijuana arrest rate is consistently insignificant. These results reinforce the conclusion that cocaine possession enforcement reduces demand for both drugs and that marijuana possession enforcement does not affect cocaine demand. They also suggest that the earlier results showing a negative effect of marijuana arrests on marijuana demand could to some extent be a product of endogeneity of the arrest rate variable.

The limitation of the alternative arrest rate variable is that it is not as close a proxy for the probability of arrest for drug possession as the version specifying the number of users in the denominator. Thus the theoretical link between the alternative arrest variable and drug demand is weaker. For instance, if drug arrests and type I arrests both increase by the same percentage because of an increase in resources devoted to law enforcement while the number of drug users holds constant, the corresponding increase in drug arrest probability is reflected by our original arrest rate variable but not by the type I arrest rate measure. This is a potential alternative explanation for the weaker effects of the own-arrest rate for marijuana. Furthermore, because possession of cocaine is considered more serious than that of marijuana, it is possible that enforcement agencies divert resources from type I arrests to cocaine but not marijuana arrests, making standardization by type I arrests more relevant for cocaine than

TABLE 7
Probit Estimates of Alternative Drug Arrest Measure

	Cocaine use			Marijuana use		
Variable	(1)	(2)	(3)	(4)	(5)	(6)
18–39-year-olds						
Cocaine arrests per type I arrest	−.126**		−.154**		−.490***	−.445***
	(.061)		(.072)		(.169)	(.170)
	[−.344]		[−.420]		[−.374]	[−.340]
Marijuana arrests per type I arrest		−.003	.039	−.181		−.061
		(.033)	(.039)	(.115)		(.114)
		[−.010]	[.123]	[−.157]		[−.053]
12–17 year-olds						
Cocaine arrests per type I arrest	−.069**		−.074**		−.331**	−.358**
	(.032)		(.036)		(.130)	(.152)
	[−.524]		[−.560]		[−.323]	[−.349]
Marijuana arrests per type I arrest		−.016	.005	−.057		.034
		(.018)	(.021)	(.079)		(.093)
		[−.148]	[.050]	[−.067]		[.040]

Notes: In each cell, the first row indicates the marginal effect on the probability of cocaine or marijuana use, the second row indicates the standard error (in parentheses), and the third row indicates the implied elasticity (in brackets). The sample size is 92,784 for the top, 42,464 for cocaine use in the lower half, and 43,147 for marijuana use in the lower half. All equations include the variables listed in Appendix Table A2 along with state and year indicators. Regressions are weighted using NHSDA sample weights.

*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

marijuana. In any event, the combined results for the two arrest variable specifications represent strong evidence that cocaine arrests are negatively related to both cocaine and marijuana demand.

Other Determinants

Appendix Table A2 contains estimates for the full set of explanatory variables in the complete equation (2) models reported in column (4) of Tables 2–5. For adults, drug demand falls as education, family size, and family income rise and is lower for females, nonwhites, married respondents, students, and rural residents. As age increases, cocaine demand increases at a declining rate until age 27 and then decreases, whereas marijuana demand falls. Among juveniles, drug demand increases with age, decreases with family income, and is lower for nonwhite non-Hispanic respondents and students. Interview interruptions and MSA size have no consistent effect on drug demand.

VI. CONCLUSION

This article estimates the effect of cocaine and marijuana prices and probabilities of arrest for cocaine and marijuana possession on past year participation in cocaine and marijuana use, controlling for various socioeconomic variables as well as state fixed effects. The results indicate that adult cocaine and marijuana demand are each sensitive to the price of cocaine but not to that of marijuana, and juvenile drug demand is not price sensitive at all. Meanwhile, for both cocaine and marijuana, increases in the probability of arrest for possession of the drug reduce the probability that the drug is used. Estimated cross-arrest effects, particularly those of cocaine arrests on marijuana demand, suggest complementarity between the two drugs.

Three important conclusions emerge. First, caution is advised when interpreting results of similar analyses that do not control for geographic fixed effects. Although cocaine demand is indeed responsive to cocaine price changes, as such analyses have shown, the magnitude of price responsiveness is overestimated when state fixed effects are excluded. Moreover, marijuana prices appear to affect both cocaine and marijuana demand for both adults and juveniles when state fixed effects are omitted. This suggests that state variation in drug use rates is partially explained by unobserved state characteristics that are also correlated with drug prices. Second, enforcement of drug possession violations reduces drug demand separately from any effects of enforcement on drug prices. Third, both price and arrest probability results provide evidence that cocaine and marijuana are complements.

Data limitations restrict our analysis to pooled cross-sections rather than longitudinal observations. This might be one reason that our estimated price and policy effects fail to account for the substantial increase in past year marijuana and cocaine prevalence among high school students during our period of analysis, as indicated by MTF survey responses summarized in Johnston et al. (2001). Past year participation rates among high school seniors increased from nadirs of 3.1% for cocaine and 21.9% for marijuana in 1992 to 5.5% for cocaine and 38.5% for marijuana in 1997. Yet possession arrests rates, which are significantly negatively related to use of both drugs by juveniles, almost doubled in per user terms during 1992–97, from .033 to .060 for cocaine and from .009 to .018 for marijuana. Changes in arrest rates during 1992–97 predict percentage point decreases of 0.4 in cocaine participation and 3.0 in marijuana participation among juveniles during this time, rather than increases in each.[24] Clearly, factors other than prices and arrests are important in determining changes in drug use across cohorts.[25] Potential demand shifters that have been suggested include a decrease in the perceived risk of using drugs, as indicated in Johnston et al. (2001), the introduction of the Internet as a forum for

24. Prices per gram declined slightly during this time, from $119 to $101 for cocaine and from $2.91 to $2.65 for marijuana. But prices are not significantly related to juvenile use; regardless, the predicted participation changes are unchanged when price effects are taken into account. In addition, because some high school seniors are age 18 or older, it is technically inappropriate to use our juvenile regressions to predict behavior for an entire cohort of high school seniors. However, similar prevalence rate increases are apparent for 8th- and 10th-graders surveyed by MTF.

25. Indeed, year coefficients from the juvenile regressions indicate that, controlling for all other explanatory variables, cocaine participation increased by 1.2 percentage points from 1992 to 1997 and marijuana participation increased by 13.3 percentage points during the same period.

drug information dissemination, an increasing glamorization of drugs in popular culture, and decreased parental supervision because of the increased divorce rate and proportion of single-parent families.

An additional data limitation is the imprecision of our local drug prices and possession arrest probability measures. Still, our improvements over past methods strengthen the reliability of our results and their associated policy implications. In particular, the added temporal variation provided by our methodology for estimating regional cocaine prices allows for the detection of price effects even when controlling for fixed state effects, which is difficult when state-level prices are used. Our methodological changes reveal that increases in marijuana and cocaine prices do not decrease drug demand as much as many previous studies have estimated. But these changes also establish that even though unobserved time-invariant differences across states account for some of the negative cross-sectional correlation between drug use and its cost, in terms of monetary prices and expected punishment, drug enforcement policy that increases prices and arrest rates still has the potential to reduce drug use.

APPENDIX TABLE A1
Census Divisions and Marijuana Price Regions

Census region	Census division	States
Northeast	New England	Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island
	Middle Atlantic	New Jersey, New York, Pennsylvania
North Central	East North Central	Illinois, Indiana, Michigan, Ohio, Wisconsin
	West North Central	Iowa, Kansas, Minnesota, Missouri, Nebraska, North Dakota, South Dakota
South	South Atlantic	Delaware, DC, Florida, Georgia, Maryland, North Carolina, South Carolina, Virginia, West Virginia
	East South Central	Alabama, Kentucky, Mississippi, Tennessee
	West South Central	Arkansas, Louisiana, Oklahoma, Texas
West	Mountain	Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah
	Pacific	Alaska, California, Hawaii, Oregon, Washington

City or state in which marijuana price is reported	States assigned marijuana price from this city/state
Atlanta	Georgia, North Carolina, South Carolina
Boston	Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island
California (Los Angeles, San Diego, San Francisco)	California, Hawaii
Chicago	Illinois, Indiana, Wisconsin
Denver	Colorado, Idaho, Montana, Nevada, Utah
Detroit	Michigan, Ohio
Miami	Florida
New Orleans	Alabama, Arkansas, Kentucky, Louisiana, Mississippi, Tennessee
New York City	New York
Newark	New Jersey
Philadelphia	Pennsylvania
Phoenix	Arizona, New Mexico
Seattle	Alaska, Oregon, Washington
St. Louis	Iowa, Kansas, Minnesota, Missouri, Nebraska, North Dakota, South Dakota
Texas (Dallas, Houston)	Oklahoma, Texas
Washington, DC	Delaware, DC, Maryland, Virginia, West Virginia

APPENDIX TABLE A2
Probit Estimates for Past Year Cocaine and Marijuana Use: Full Models from Column (4), Tables 2–5

Variable	Cocaine ages 18–39	Marijuana ages 18–39	Cocaine ages 12–17	Marijuana ages 12–17
Cocaine price (×1,000)	−.132**	−.248**	.013	.043
	(.057)	(.125)	(.036)	(.126)
Marijuana price (×100)	−.074	−.412	−.152	−.389
	(.231)	(.543)	(.153)	(.593)
Cocaine arrests per user	−.430***	−.110	−.180***	−.403***
	(.071)	(.125)	(.041)	(.150)
Marijuana arrests per user	−.252	−5.630***	.117	−2.171***
	(.296)	(.853)	(.160)	(.659)
Interview w/o interruptions	−.000	.002	−.000	.025***
	(.002)	(.005)	(.001)	(.005)
Interview w/o interruptions—missing	−.008	.000	−.005**	.022
	(.005)	(.014)	(.001)	(.017)
Age	.006***	−.008**	.004***	.043***
	(.001)	(.004)	(.000)	(.001)
Age squared (×100)	−.010***	.006		
	(.002)	(.006)		
Male	.023***	.067***	−.001	.012***
	(.002)	(.004)	(.001)	(.004)
Black	−.004*	−.027***	−.005***	−.011*
	(.002)	(.005)	(.001)	(.005)
Hispanic	−.013***	−.081***	.003	−.006
	(.002)	(.005)	(.002)	(.006)
Other race	−.014***	−.078***	−.003*	−.039***
	(.004)	(.007)	(.002)	(.009)
Married	−.031***	−.091***	−.002	.029
	(.003)	(.005)	(.006)	(.041)
Divorced	.004*	.006		
	(.003)	(.007)		
Family size	−.001**	−.010***	−.001***	−.008***
	(.001)	(.001)	(.000)	(.001)
Enrolled in school	−.015***	−.023***	−.017***	−.038***
	(.002)	(.005)	(.004)	(.009)
High school graduate	−.014***	−.037***	−.004	−.028**
	(.002)	(.005)	(.002)	(.011)
Some college education	−.017***	−.043***	.121***	.087
	(.002)	(.005)	(.087)	(.114)
College graduate	−.026***	−.075***		
	(.002)	(.006)		
Some graduate education	−.028***	−.069***		
	(.002)	(.008)		
Family income (×10,000)	−.002***	−.004***	−.001***	−.004***
	(.000)	(.001)	(.000)	(.001)
MSA pop. > 1 million	−.001	.014	−.002	.016*
	(.004)	(.009)	(.002)	(.010)
MSA pop. between 500,000 & 1 million	.001	.016*	.001	.027***
	(.004)	(.010)	(.002)	(.010)
MSA pop. between 250,000 & 500,000	.005	.010	−.001	.017
	(.005)	(.011)	(.002)	(.013)
Rural	−.010***	−.030***	−.002	−.024***
	(.003)	(.008)	(.002)	(.007)
Sample size	92,784	92,784	42,464	43,147
Log likelihood	−15,325	−36,800	−2,894	−13,755

Notes: In each cell, the first row indicates the marginal effect on the probability of cocaine or marijuana use and the second row indicates the standard error (in parentheses). All equations include state and year indicators. Regressions are weighted using NHSDA sample weights.

*, **, and *** indicate significance at the 90%, 95%, and 99% levels, respectively.

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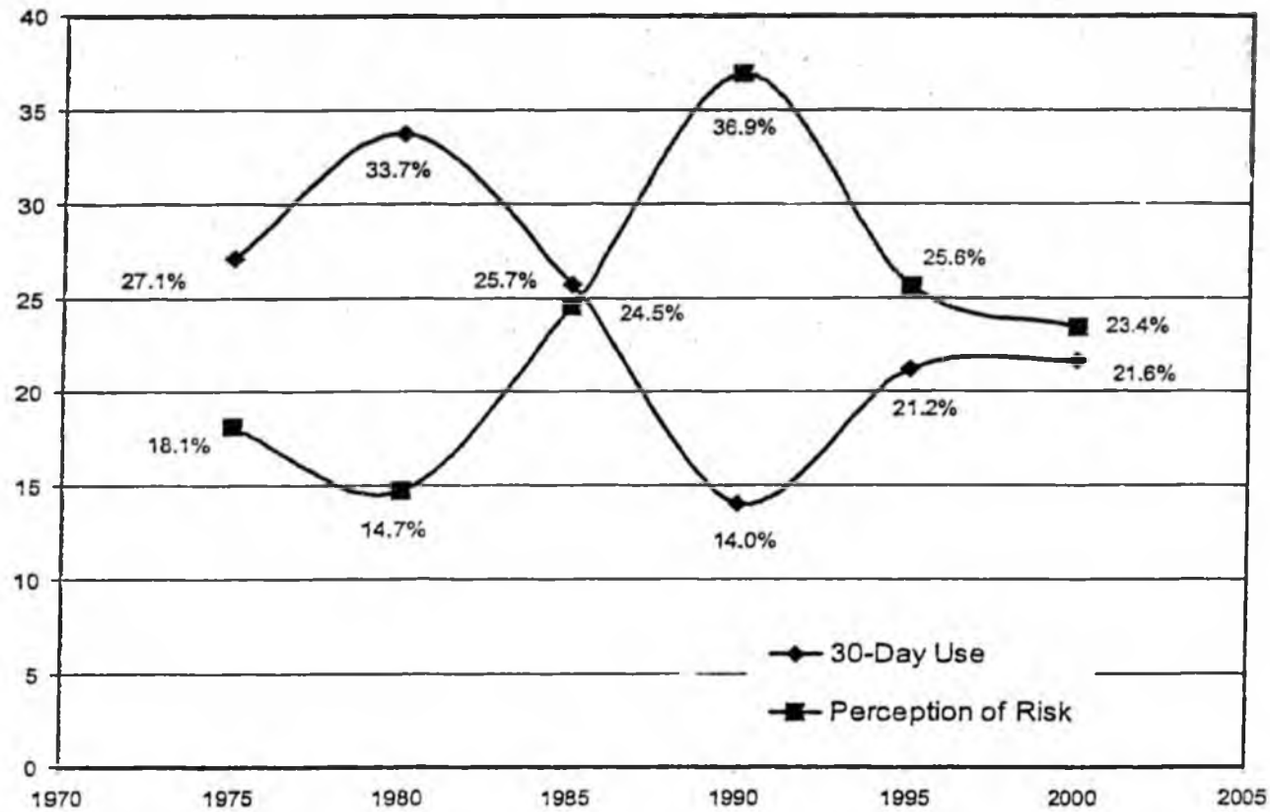
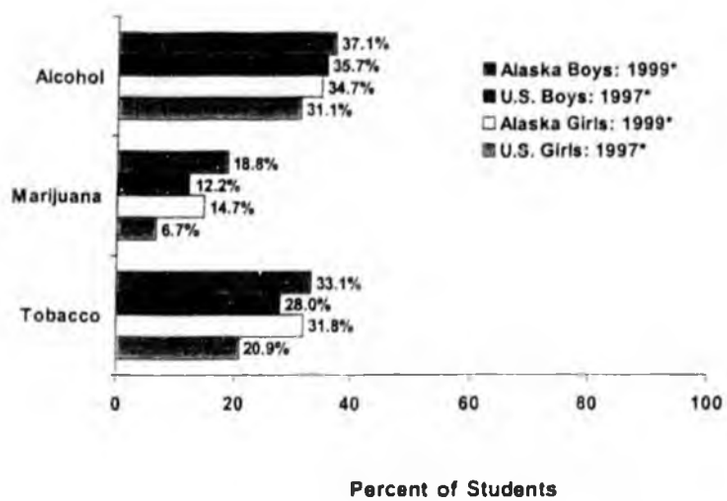
Marijuana Use and Perceived Risk

Data from Monitoring the Future Survey, Dec. 2001
www.monitoringthefuture.org



Use of Alcohol, Marijuana, or Tobacco Before the Age of 13 Years

Almost 40% of Alaska high school boys report having had a first drink of alcohol before age 13 years. Also by age 13 years, 18.8% of boys and 14.7% of girls report having tried marijuana for the first time, accounting for about a quarter of those who have ever used marijuana. Percentages of age at first use are higher for Alaska boys and girls than U.S. boys and girls in use of alcohol, tobacco, and marijuana.



*Use of alcohol, marijuana, or tobacco before the age of 13 years.

Year 2000 Objectives:

- ***Increase by at least 1 year the average age of first use of cigarettes, alcohol, and marijuana by adolescents aged 12-17.***

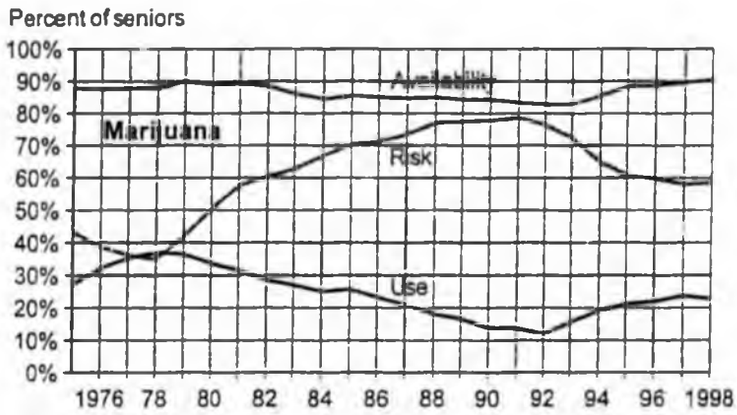
Change in students' use of marijuana and alcohol is tied to their perception of possible harm from use

The annual Monitoring the Future Study, in addition to collecting information about students' use of illicit drugs, alcohol, and tobacco, also collects data on students' perceptions regarding the availability of these substances and the risk of harm from using them.

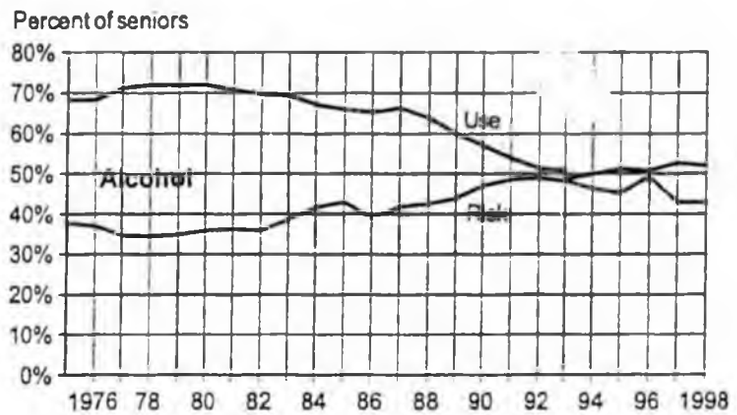
Between 1975 and 1998, the proportion of high school seniors reporting use of marijuana in the 30 days prior to the survey fluctuated, peaking in 1978 and then declining consistently through 1992. Since then, reported use has increased, but the 1998 rate was still far below the peak level of 1978. When the perceived risk of "great harm" from either regular or occasional use of marijuana increased, use declined; when perceived risk declined, use increased. The perception that obtaining marijuana was "fairly easy" or "very easy" remained relatively constant between 1975 and 1998.

Students' reported use of alcohol also shifted from 1975 to 1998. After 1978, alcohol use declined through 1993. Alcohol use fluctuated within a limited range thereafter, but the 1998 rate was far lower than the 1978 rate. As with marijuana, when the perceived risk of "great harm" from either weekend "binge" drinking or daily drinking increased, use declined; when perceived risk declined, use increased.

Over the past 20 years, while availability remained constant, changes in marijuana and alcohol use reflected changes in perceived harm



Availability: Percent saying fairly easy or very easy to get.
Risk: Percent saying great risk of harm in regular use.
Use: Percent using once or more in the past 30 days.



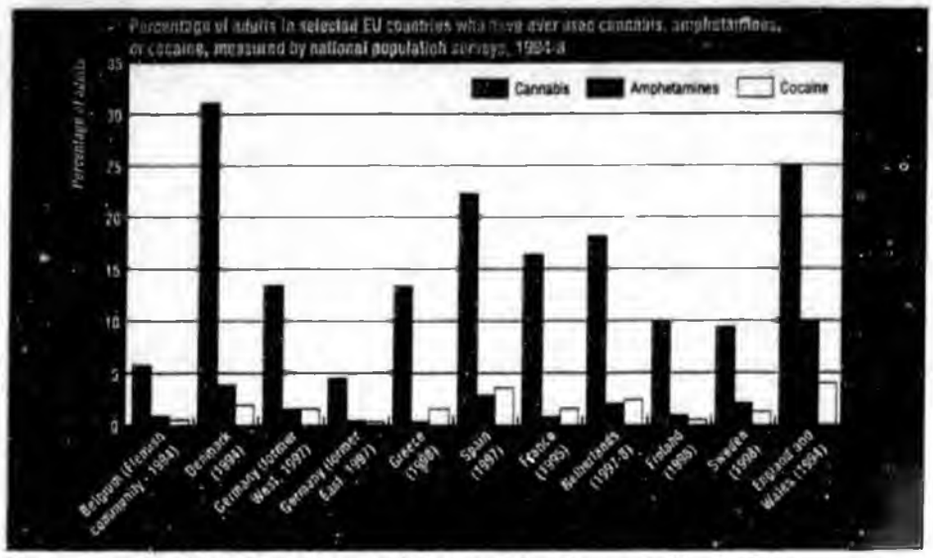
Risk: Percent saying great risk of harm in having five or more drinks once or twice each weekend.
Use: Percent using once or more in the past 30 days.

Note: The survey question on alcohol use was revised in 1993 to indicate that a "drink" meant "more than a few sips." In 1993, half the sample responded to the original question. In 1994 through 1998, all respondents were asked the revised question.

Source: Authors' adaptation of Johnston, O'Malley, and Bachman's *Drug use by American young people begins to turn downward.*

EU centre calls for policies to help female drug users

Rory Watson *Brussels*



The Danes top the table for cannabis use, with the former East Germany trailing the field

Patterns of drug misuse in the European Union are changing, with a chronically ageing population among heroin addicts and a wider use of cocaine, cannabis, and combinations of amphetamines, ecstasy, and medicines.

The picture is contained in the annual report of the European Union's European Monitoring Centre for Drugs and Drug Addiction in Lisbon. It estimates that the number of drug addicts has remained stable at around 1.5 million, but within that figure lie different trends.

The numbers of people starting treatment for heroin are decreasing and users tend to be older with serious social and psychiatric problems. In contrast, new admissions for cocaine or cannabis use are rising, especially among the young.

Among schoolchildren, experience of cannabis ranges from 5-7% in Portugal and Sweden to 30-40% in the Republic of Ireland, the Netherlands, and the United Kingdom.

The centre makes a plea for more tailor-made responses to take account of female drug users who fear they may lose their children if they enrol for treatment. It also highlights the need for policies addressed at women who finance their habit through the sex industry. Although 12 EU countries have specific programmes in this area, Belgium, Finland, and Sweden do not.

The report draws attention to the drug prevention schemes in Austria, Germany, and Sweden directed at very young women and schoolgirls to prevent them from picking up the habit from older boyfriends.

Given that at least 45 million people in the European Union have tried cannabis at least once and that around 15 million have done so in the past year, it is not surprising that policymakers are targeting the phenomenon.

But the European monitoring centre's director, Georges Estievenart, criticised a zero tolerance approach. "No one really sees this as a crime to be repressed with an iron fist," he said. □

Marijuana has potential for misuse

Abi Berger *BMJ*

Marijuana has the potential for misuse, according to a study from the United States. New evidence that monkeys self administer the active component of marijuana has been shown by Dr Steven Goldberg and his team at the National Institutes of Health in Baltimore (*Nature Neurosci...* 2000;3:1073-4).

One of the criteria used to help decide if a drug has the potential for misuse is whether animals will work to obtain it. This is known as self administration. Virtually all psychoactive drugs misused by humans, including nicotine, have been shown to be self administered by animals, but up to now a positive self administration test has been elusive whenever THC (delta-9-tetrahydrocannabinol), the active part of marijuana, has been tested. This has led to some people concluding that marijuana is less likely to lead to drug misuse than other illegal substances.

Dr Goldberg, a pharmacologist at the National Institute of Drug Abuse, has shown now that monkeys can be trained to self administer THC. In this study the team used a low-but clinically relevant-dose of THC administered intravenously in a clear solution. This solution rapidly distributed THC to the brain. Previous attempts to show self administration, using much higher doses of THC held in a suspension, failed. One reason for this may be that, although higher doses were used, the suspension resulted in less brain penetration.

In this study the monkeys had previously been trained to self administer cocaine by pressing a lever 10 times. When saline was substituted for cocaine, self administration stopped. When THC replaced the saline, the monkeys quickly started to press the lever again. The monkeys gave themselves about 30 injections during an hour long session, which equates roughly with the dose received by a person smoking a marijuana joint.

The team went on to confirm that giving the monkeys a second drug that directly blocks cannabinoid receptors in the brain could prevent self administration. This suggests that THC antagonists may be useful in combating marijuana addiction in humans. Dr Goldberg's team will next be trying their approach in "naive" monkeys (animals that have not previously been exposed to other psychoactive drugs) to see if this alters the animals' behaviour.

Dr Goldberg's team concludes from its observations that THC "has as much potential for abuse as other drugs of abuse, such as cocaine and heroin." □

Reactions to the cannabis study

Martin Jarvis, professor of health psychology at University College London said that to suggest that the potential for misusing marijuana is as great as with drugs such as cocaine and heroin is probably overstating the case. He said that misuse is "a judgment best made by looking at patterns of actual human use." He continued: "We shouldn't assume that unreasonable behaviour in society follows from the observation of brain reward behaviour in animals alone".

Ian Stolerman, professor of behavioural pharmacology at the Institute of Psychiatry in London, agreed: "This is an important study because for the first time it provides a method for studying directly the intake of THC by a laboratory animal and thus models a key behavioural feature of addictive states generally. It will lead to studies of how and where THC works in the brain to generate drug abuse. It does show that THC shares properties with other drugs of abuse, but whether it is really as potentially abusive as cocaine and heroin is not so clear."

ceased or reduced smoking before surgery compared with less than 10% of those in the control group.[11] The intervention group was much less likely to experience postoperative complications, especially wound healing and cardiovascular complications, and to need secondary surgery. A Cochrane review found that intensive behavioural interventions with patients admitted to hospital were associated with higher quit rates when linked to follow up contact for at least a month.[12]

Given this evidence, it is arguable that resources expended on smoking rooms might be better used to fund a concerted effort to implement a smoking ban and to expand smoking cessation activities. Hopefully other hospitals facing a similar situation will act differently in the future.

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Competing interests: MM worked at the Royal Victoria Hospital in the early 1980s. TLN is a former US assistant surgeon general, in which role he was involved in negotiations on the framework convention on tobacco control. All authors have received funding from a variety of governmental and intergovernmental agencies for research on tobacco control.

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Comparing cannabis with tobacco

Smoking cannabis, like smoking tobacco, can be a major public health hazard

Britain now has 13 million tobacco smokers. This number has been steadily decreasing due to public awareness of the harm caused by tobacco smoking. At the same time the number of cannabis smokers is increasing. Between 1999 and 2001, the number of 14-15 year olds who had tried cannabis rose from 19% to 29% in boys and 18% to 25% in girls, and a Home Office document estimates that 3.2 million people in Britain smoke cannabis.[1 2] However, the harmful effects of smoking cannabis are widely known and have recently been highlighted.[3 4] Although the active ingredients of the cannabis plant differ from those of the tobacco plant, each produces about 4000 chemicals when smoked and these are largely identical. Although cannabis cigarettes are smoked less frequently than nicotine cigarettes, their mode of inhalation is very different. Compared with smoking tobacco, smoking cannabis entails a two thirds larger puff volume, a one third larger inhaled volume, a fourfold longer time holding the breath, and a fivefold increase in concentrations of carboxyhaemoglobin.[5] The products of combustion from cannabis are thus retained to a much higher degree. How is this likely to translate into adverse effects on health?

We already know that regular use of cannabis is associated with an increased incidence of mental illnesses, most notably schizophrenia and depression,[6] but it is also worth examining its potential to cause other illnesses, especially those of the heart and respiratory system.

At present, there is an understandable dearth of epidemiological evidence of cardiopulmonary harm from cannabis, because its use is a relatively new phenomenon and its potency is changing. The amount of the main active constituent, tetrahydrocannabinol (THC), in cannabis has increased from about 0.5% 20 years ago to nearer 5% at present in Britain, whereas "Nederweed" (the variety smoked in the Netherlands) has an average of 10-11% tetrahydrocannabinol. At the same time little study has been undertaken of any concomitant change in the content of tar. Case-control studies are difficult to perform since cannabis cigarettes do not come in standard sizes, which makes dose-response relations difficult to establish. Furthermore, most users of cannabis also smoke tobacco, which makes it difficult to dissect out individual risks. As with tobacco, there will be a latent period between the onset of smoking and the development of lung damage, cardiovascular disease, or malignant change.

Tobacco smoking is responsible for 120 000 excess deaths each year in Britain, 46 000 from cancers, 34 000 from chronic respiratory disorders, and 40 000 from diseases of the heart and circulation. However, there are indications that smoked cannabis may cause similar effects to smoking tobacco, with many of them appearing at a younger age. Smoking cannabis causes chronic bronchitis, emphysema, and other lung disorders, which were recently summarised in a review released by the British Lung Foundation.[7] A striking feature of cannabis smoking is that it is associated with

BMJ 2003;326:942–3

bullous lung disease in young people.[5] Inflammatory lung changes, chronic cough, and chest infections are similar to those in cigarette smokers, but may also be commoner in younger people.[5,6] Premalignant changes have been shown in the pulmonary epithelium, and there are reports of lung, tongue, and other cancers in cannabis smokers.

Tetrahydrocannabinol has cardiovascular effects, and sudden deaths have been attributed to smoking cannabis.[7,8] Myocardial infarction is 4.2 times more likely to occur within an hour of smoking cannabis.[11] However, despite these alarming facts, there is no evidence at present on whether smoking cannabis contributes to the progression of coronary artery disease, as smoking cigarettes does. More studies of the cardiovascular and pulmonary effects of cannabis are essential.

It may be argued that the extrapolation from small numbers of individual studies to potential large scale effects amounts to scaremongering. For example, one could calculate that if cigarettes cause an annual excess of 120 000 deaths among 13 million smokers, the corresponding figure for deaths among 3.2 million cannabis smokers would be 30 000, assuming equality of effect. Even if the number of deaths attributable to cannabis turned out to be a fraction of that figure, smoking cannabis would still be a major public health hazard. However, when the likely mental health burden is added to the potential for morbidity and premature death from cardiopulmonary disease, these signals cannot be ignored. A recent comment said that prevention and cessation are the two principal strategies in the battle against tobacco.[12] At present, there is no battle against cannabis and no clear public health message.

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People missing as a result of armed conflict

Standards and guidelines are needed for all, including health professionals

Mass graves from past or present conflicts, massacres in the Balkans, disappearances—South American style—and the missing in action are politically sensitive. One reason is that they usually entail violations of international humanitarian law (the wartime rules that protect people who are not in combat or no longer in combat) or human rights law. International criminal tribunals to try individuals believed to be responsible for the violations attract equal attention. Why these events and the reactions to them by the international community are of direct concern to health professionals is not immediately obvious, although it has been widely recognised that they have an important part to play in upholding such laws.[1,2] However, the specific roles, responsibilities, and expertise of the profession either in ascertaining the fate of the missing or in helping affected families have not been as widely recognised.

The story of people unaccounted for as a result of armed conflict or internal violence is told differently according to the narrator's discipline. Each discipline has its own work and objectives. Lawyers uphold international law and attempt to prosecute the perpetrators of violations; forensic specialists identify remains, contribute to the reconstruction of events surrounding the death, and establish the cause of death. Psychologists address the kind of mental torture associated with uncertainty of the whereabouts of a family member. Military bodies emphasise the importance of measures such as the wearing of identification tags and registering deaths of their personnel. Red Cross workers respond to families' requests to trace a missing person and to visit and register prisoners of war. This is an incomplete list, and each discipline has worked largely in its own sphere. Furthermore in a given situation there are different actors each employing, manipulating, or even hindering the work of the different disciplines. These actors may be the governments, military bodies, international organisations including the United Nations, and non-governmental organisations. Clearly it is time for standards and guidelines on best practice for all professionals.

The International Committee of the Red Cross has been forced into undertaking an initiative, "The Missing," which has taken the form of a series of expert workshops and studies and a review of its own practice over time and by continent. The outcome has revealed ambiguity about the legal and ethical basis of any action involving forensic specialists, the lack of best practice guidelines to guide these specialists, the difficulty of accommodating local customs and culture in an investigation, and recognition of an inconsistency of the International Committee of the Red Cross's own practice with regard to missing people. At centre stage, however,

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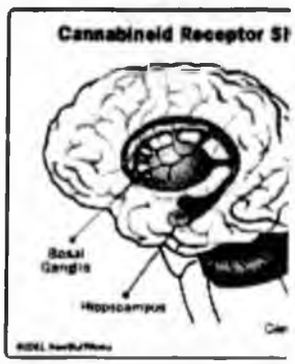
Drug Related News and Announcements

Marijuana may affect blood flow in brain

Study suggests users of drug have narrowed arteries



Reuters: February 7, 2005

WASHINGTON - Marijuana users have faster blood flow in their brains, even after a month of not smoking, U.S. researchers reported Monday.

The findings suggest they have narrowed arteries, similar to patients with high blood pressure and dementia, and could help explain reports that heavy marijuana users have trouble on memory tests, said the researchers at the National Institute on Drug Abuse in Baltimore.

Ronald Herning and Jean Lud Cadet tested 54 marijuana users, who smoked anywhere between two and 350 joints a w and 18 non-smokers.

They used Doppler sonograms to measure blood flow in volunteers' brains at the beginning of the study and a month lat everyone agreed to abstain from marijuana for the four weeks.

The smokers had faster blood flow, both at the start and after a month of abstinence, Herning and Cadet reported in the Neurology.

The smokers also had a higher pulsatility index score, or PI, which measures the amount of resistance to blood flow. Th researchers believe the higher PI is caused by narrower blood vessels.

"The marijuana users had PI values that were somewhat higher than those of people with chronic high blood pressure a diabetes," Herning said in a statement.

"However, their values were lower than those of people with dementia. This suggests that marijuana use leads to abnorr in the small blood vessels in the brain."

They found that blood flow improved in people who smoked up to 70 marijuana cigarettes a week -- people they defined moderate users -- after a month of avoiding cannabis.

Heavy users, who smoked up to 350 joints a week, saw no change in blood flow even after a month, the researchers sai

Researchers at Montreal's McGill University have reported that chronic consumers of cannabis lose molecules called CE receptors in the brain's arteries.

This reduces blood flow to the brain, causing attention deficits, memory loss, and impaired learning ability.

state convention. The Governor may declare to honor an anniversary of the association in conjunction with the convention.

- Another exception may be made when a national organization is holding a national meeting in Tennessee (e.g. NAACP National Conference). The Governor may declare "NAACP Week in Tennessee" in honor of the state and national organization in conjunction with its convention.

- Proclamations are **not** issued in honor of people, living or dead unless required by Tennessee Code Annotated (TCA).

- Requests will be accepted no more than 6 months in advance and will not be issued more than 3 months in advance of the event.

- A 14 business day notice is required to provide proclamations. If notice is given the Governor's office reserves the right to decline.

- Because of cost involved in writing and printing proclamations, a maximum of 5 originals of issued proclamations will be sent per year per constituent.

- Any draft language provided may be edited or rewritten at the discretion of the Governor's office.

- All proclamation requests must be in writing and can be submitted online via webform or via mail or fax and sent to:

Cara Wilson
State Capitol
Nashville, TN 37243
615-532-4562 phone
615-532-9712 fax

Governor's Office
Tennessee State Capitol
Nashville, TN 37243-0001
615.741.2001

National Institutes of Health
National Institute on Drug Abuse

FOR IMMEDIATE RELEASE
October 15, 2000

CONTACT:
Beverly Jackson
Michelle Muth
(301) 443-6245

NIDA Researchers Find That Animals Exposed to Marijuana's Active Component Will Self-Administer the Drug

Scientists at the National Institute on Drug Abuse have demonstrated that laboratory animals will self- administer marijuana's psychoactive component, delta-9-tetrahydrocannabinol, in doses equivalent to those used by humans who smoke the drug. Self-administration of drugs by animals, long considered a model of human drug-seeking behavior, is characteristic of virtually all addictive and abused drugs.

"This study is simple and its findings are clear," says NIDA Director Dr. Alan I. Leshner. "Animals will work to get THC. This emphasizes further the similarity between marijuana and other abusable, addicting substances. Both animals and humans will work to acquire access to marijuana in the same way that both animals and humans change their behavior to get other drugs of abuse, like cocaine and heroin."

Dr. Steven Goldberg and colleagues at NIDA's Intramural Research Program in Baltimore, Maryland, report in the current issue of "Nature Neuroscience" that squirrel monkeys will self-administer intravenous injections of THC.

"This is the first study in which it has been possible to show that monkeys or other research animals will self- administer THC. There are many factors which may explain this behavior, including the fact that in our study we used doses of THC that are directly comparable to doses in marijuana smoke inhaled by humans," Dr. Goldberg says.

Before the study began, the scientists first established self-administration behavior in squirrel monkeys that received repeated intravenous injections of cocaine after pressing a lever 10 times for each injection. At the start of the study, the researchers replaced cocaine with saline solution and the animals' self-administration stopped. When saline was replaced with THC in a solution that would rapidly pass from blood to the brain, the animals resumed self-administration, rapidly pressing the lever to obtain on average 30

injections of THC during each of a series of 1- hour sessions. Treatment with a compound that prevented THC from binding to cannabinoid receptors on brain cells almost completely eliminated self-administration of THC, but had no effect in another group of monkeys self-administering cocaine under identical conditions, according to Dr. Goldberg.

"The drug-seeking behavior in these animals was comparable in intensity to that maintained by cocaine under identical conditions, and was obtained from a range of doses comparable to those self-administered by humans smoking a single marijuana cigarette," Dr. Goldberg says. "This finding suggests that marijuana has as much potential for abuse as other drugs of abuse, such as cocaine and heroin."

NOTE TO REPORTERS: The full text of the brief communication about this study is available in "Nature Neuroscience 2000", volume 3, pgs 1073-74 or at www.neurosci.nature.com.

The National Institute on Drug Abuse is a component of the National Institutes of Health, U.S. Department of Health and Human Services. NIDA supports more than 85 percent of the world's research on the health aspects of drug abuse and addiction. The Institute carries out a large variety of programs to ensure the rapid dissemination of research information and its implementation in policy and practice. Fact sheets on the health effects of drugs of abuse and other topics can be ordered free of charge in English and Spanish from NIDA Infofax at 1-888-NIH-NIDA (644-6432) or 1- 888-TTY-NIDA (889-6432) for the deaf. These fact sheets and further information on NIDA research and other activities can be found on the NIDA home page at www.drugabuse.gov.

1988	132	80	348	327	210	164	112	136
1989	122	96	326	280	175	175	116	99
1990	130	94	309	240	197	135	103	148
1991	154	96	302	265	180	171	160	101
1992	185	159	347	258	222	173	104	82
1993	244	222	364	355	229	210	124	136
1994	276	261	450	394	242	234	123	121
1995	336	274	510	401	226	256	137	141
1996	350	294	523	523	235	268	138	202
1997	329	313	547	478	266	227	145	139
1998	334	313	519	467	236	250	154	175
1999[1]	291	255	446	399	151	175	124	159

* Low precision; no estimate reported.

[1] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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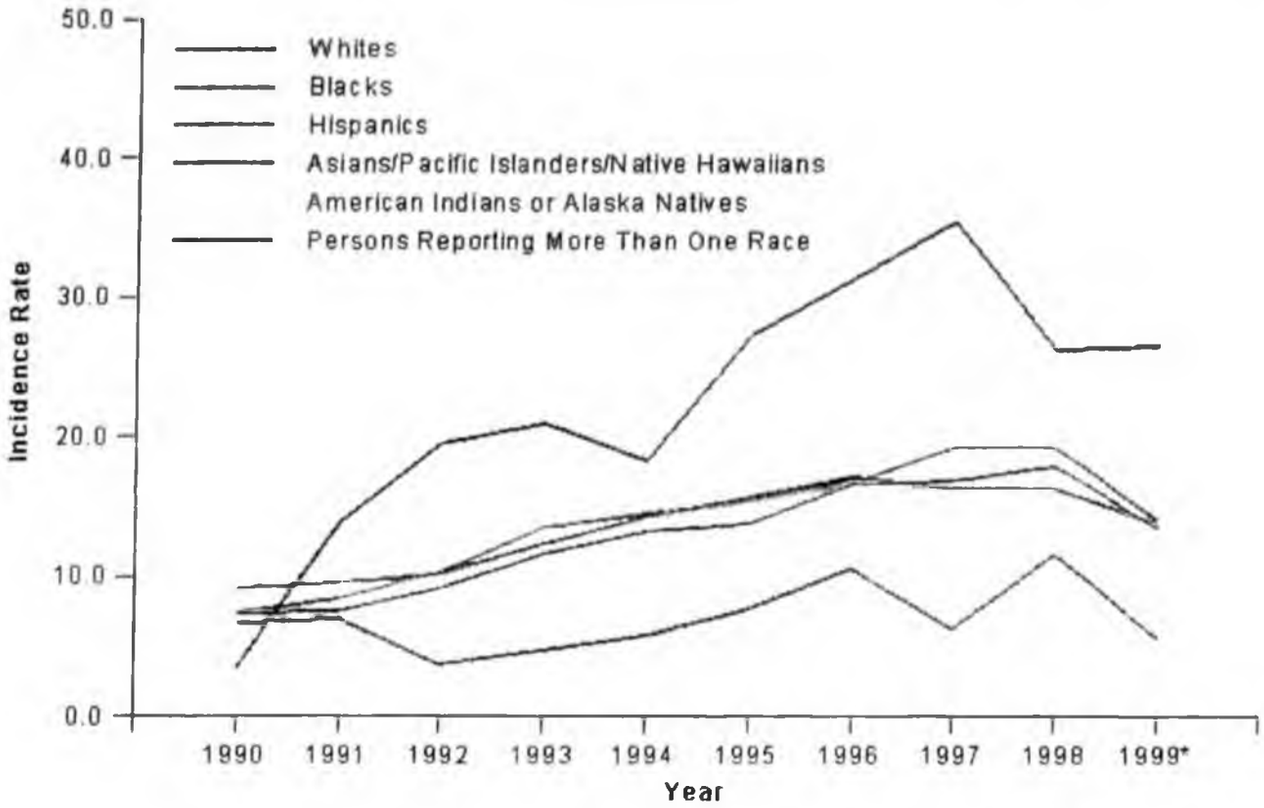
Table 3.3 Estimated Numbers (in Thousands) of Persons Who First Used Marijuana During the Years 1965 to 1999, Their Mean Age at First Use, and the Annual Incidence Rates of First Use (Per 1,000 Person-Years of Exposure), by Gender

Year	Number of Initiates (1,000s)		Mean Age at First Use		Incidence Rates[1]	
	Males	Females	Males	Females	Males	Females
1965	315	239	18.1	23.4	4.9	3.3
1966	642	333	18.8	19.9	9.8	4.5
1967	952	433	19.1	20.4	14.4	5.7
1968	1,212	527	19.0	20.1	18.1	6.8
1969	1,264	859	18.6	19.5	18.7	10.9
1970	1,479	1,112	18.6	19.0	21.7	13.9
1971	1,570	1,218	18.4	19.0	22.9	15.1
1972	1,560	1,258	19.2	18.3	22.7	15.5
1973	1,587	1,267	18.6	18.6	23.1	15.5



Note: The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years for persons aged 12 or older.

* Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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Table 3.1 Estimated Numbers (in Thousands) of Persons Who First Used Marijuana During the Years 1965 to 1999, Their Mean Age at First Use, and the Annual Incidence Rates of First Use (Per 1,000 Person-Years of Exposure), for All Ages

Year	Number of Initiates (1,000s)	Mean Age at First Use	Incidence Rates[1]

1974	1,493	1,360	17.7	18.1	21.7	16.6
1975	1,405	1,469	17.7	18.9	20.4	17.8
1976	1,625	1,559	18.2	18.8	23.6	18.9
1977	1,647	1,517	18.0	18.5	23.9	18.4
1978	1,556	1,411	17.6	18.7	22.5	17.0
1979	1,507	1,352	17.5	18.7	21.7	16.2
1980	1,187	1,335	19.0	19.4	17.0	15.9
1981	896	971	17.2	18.6	12.6	11.4
1982	1,014	1,007	17.9	19.7	14.1	11.7
1983	1,049	815	18.9	17.4	14.4	9.4
1984	1,020	992	18.3	18.2	13.8	11.3
1985	1,021	844	18.2	17.9	13.6	9.5
1986	925	828	17.8	17.4	12.1	9.2
1987	773	815	17.3	17.9	10.0	9.0
1988	834	716	17.1	17.9	10.8	7.9
1989	787	660	17.5	17.8	10.3	7.3
1990	774	633	17.5	19.4	10.2	7.1
1991	837	648	18.1	17.8	11.2	7.3
1992	909	690	16.5	16.8	12.3	7.8
1993	1,009	945	16.8	17.6	13.8	10.8
1994	1,152	1,035	16.7	16.8	16.0	11.9
1995	1,254	1,103	16.4	16.7	17.7	12.9
1996	1,284	1,306	16.4	17.7	18.5	15.5
1997	1,318	1,176	17.0	16.9	19.3	14.1
1998	1,268	1,220	17.6	17.2	18.9	14.9
1999[2]	1,034	993	16.4	17.6	15.5	12.1

[1] The numerator of each rate is the number of persons who first used marijuana in the year, while the denominator is the person-time exposure measured in thousands of years.

[2] Estimated using 2000 data only.

Source: SAMHSA, Office of Applied Studies, National Household Survey on Drug Abuse, 1999 and 2000.

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1965	553	20.4	4.0
1966	975	19.2	7.0
1967	1,385	19.5	9.7
1968	1,738	19.4	12.0
1969	2,123	19.0	14.5
1970	2,592	18.7	17.5
1971	2,789	18.7	18.7
1972	2,819	18.8	18.8
1973	2,854	18.6	19.0
1974	2,853	18.9	18.9
1975	2,874	18.3	19.0
1976	3,184	18.5	21.0
1977	3,163	18.3	20.9
1978	2,967	18.1	19.5
1979	2,859	18.1	18.7
1980	2,522	19.2	16.4
1981	1,867	17.9	12.0
1982	2,021	18.8	12.8
1983	1,865	18.2	11.7
1984	2,012	18.3	12.4
1985	1,865	18.1	11.4
1986	1,753	17.6	10.6
1987	1,588	17.6	9.5
1988	1,550	17.4	9.2
1989	1,447	17.7	8.7
1990	1,407	18.3	8.5
1991	1,485	18.0	9.1
1992	1,599	16.7	9.8
1993	1,954	17.2	12.2
1994	2,187	16.7	13.8
1995	2,357	16.5	15.1
1996	2,590	17.1	16.8
1997	2,494	17.0	16.5
1998	2,488	17.4	16.7
1999[2]	2,028	17.0	13.6

SB

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TESTIMONY AGAINST (FILE 1)

Alaska Civil Liberties Union

An Affiliate of the American Civil Liberties Union
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Phone: (907) 258-0044 Fax: (907) 258-0288 Email: akclu@akclu.org

Documents Submitted by William R. Satterbert, Jr., Esq., in Support of Response of David S. Noy to State's Petition for Hearing Dated 1/22/04, State v. Noy, Supreme Court No. S-11297

Submitted to Senate Health, Education and Social Services Committee
Friday, April 1, 2005

SB 74 – *An Act making findings relating to marijuana use and possession…*

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A

6
Cannabis Pharmacology

Understanding marijuana's impact on biological systems requires a description of its active components. This chapter begins by identifying the mind-altering molecules in marijuana, the substances that contain these chemicals, and their respective potencies. It continues with a discussion of the way these substances enter the body, metabolize, and reach their sites of action. The remainder of the chapter focuses on the receptors that respond to these cannabinoids and the natural substances in the body that work at these same sites.

Marijuana contains more than 60 compounds unique to the plant called cannabinoids. They interact with each other in interesting ways, altering their impact. Cannabinoids appear in a variety of strengths in marijuana, hashish, hash oil, and synthetic medications like nabilone, dronabinol, and levonantradol. People eat or smoke these products, leading to slower or faster absorption of chemicals. The cannabinoids alter the permeability of nerve membranes. They also react with their own special receptors—CB1 in the brain and nervous system and CB2 in the immune system. Researchers have identified substances native to the body that also work on these receptors, including anandamide and arachidonolyl-glycerol. Details of each of these topics appear in the following sections.

Active Ingredients—the Cannabinoids

A first step in understanding marijuana's impact involves identifying its active components. Cannabis contains more than 400 different chemical

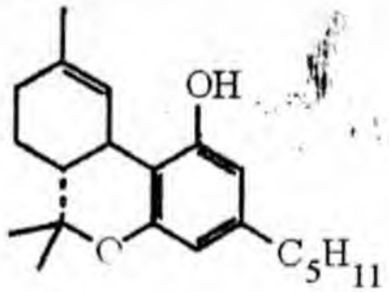

Figure 6.1. Delta-9-THC. This cannabinoid is responsible for most of marijuana's psychoactive effects.

compounds. At least 66 are unique to the plant and receive the name "cannabinoids." The best known cannabinoid is probably delta-9 tetrahydrocannabinol (THC). Considerable research has also examined the related molecule delta-8 THC. Two conventions exist for naming chemicals: formal and monoterpenoid. Thus, delta-9 THC (the formal name) is also called delta-1 THC (the monoterpenoid name). Similarly, delta-8 THC is also known as delta-6 THC. This text uses only the formal names. THC alone refers to the delta-9 variety. Delta-9 THC and delta-8 THC appear to produce the majority of the psychoactive effects of marijuana. As figures 6.1 and 6.2 reveal, the molecules differ only in the location of the double bond in the first carbon ring.

Delta-9 THC is more abundant in the plant, leading researchers to hypothesize that it is the main source of the drug's impact. The liver breaks delta-9 THC down into 11-OH-delta-9 THC (11-hydroxy-delta-

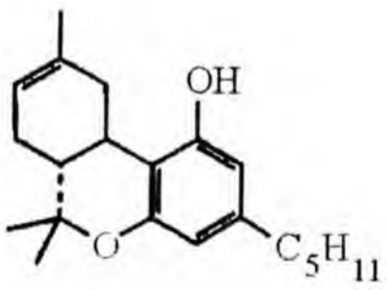

Figure 6.2. Delta-8-THC. This cannabinoid produces some of marijuana's psychoactive effects, but it is less abundant than delta-9-THC.

9-THC). This metabolite also causes psychoactive effects, including changes in subjective sensations. As figure 6.3 reveals, it only differs from THC by a few atoms, but it may be three times as potent because it reaches the brain more readily (Razdan, 1986).

Two other common cannabinoids are cannabinol and cannabidiol, depicted in figures 6.4 and 6.5, respectively. Delta-9-THC, cannabinol, and cannabidiol are the most prevalent psychoactive chemicals in the plant and provide the majority of marijuana's effects. For example, THC and cannabidiol account for 95% of marijuana's active ingredients (Doorenbos, Fetterman, Quimby, & Turner, 1971). Dozens of other cannabinoids exist, but most are variants of delta-9 THC, delta-8 THC, cannabinol, and cannabidiol. Research has uncovered six additional families of molecules unique to marijuana. All begin with the familiar "cannab" prefix. These include cannabichromene, cannabicyclol, cannabielsoin, cannabigerol, cannabinidiol, and cannabitriol. Many differ little from each other. All are lipophilic, meaning that they dissolve in fat, fatty tissue, or fatty fluids. They are not soluble in water. Thus, despite the claims of many aging hippies, teas made from boiled marijuana probably do not create extensive cannabinoid effects.

A great deal of research focuses on THC. Some investigators have turned their attention to the other cannabinoids, particularly cannabinol and cannabidiol. Studies address the activity of each of these chemicals alone and in combination with THC. Cannabinol has generated considerable interest, in part, because THC breaks down into this compound

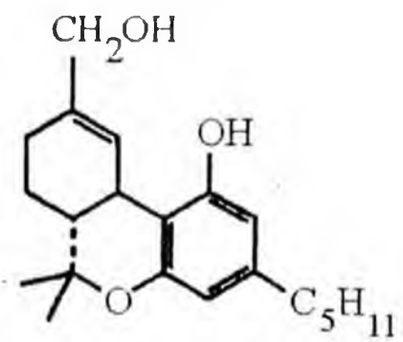

Figure 6.3. 11-hydroxy-delta-9-THC. The liver breaks delta-9-THC into this compound, which reaches the brain faster and may be 3 times as psychoactive.

research has also addressed cannabinol's interaction with THC. Cannabinol diminishes the intensity of THC's subjective effects but prolongs their duration. Cannabinol may also decrease some of the stimulation that THC can produce (Brazis & Mathre, 1997).

Considerable research also focuses on cannabidiol. Cannabidiol becomes THC as the marijuana plant matures, and this THC later breaks down into cannabinol. Up to 40% of the cannabis resin from some marijuana strains is cannabidiol (Grilly, 1998). Yet the amount varies in different plants. Some African varieties contain little of the chemical (Turner & Hadley, 1974). Early studies suggested that cannabidiol administered alone had no impact, much like early studies of cannabinol (Hollister, 1974). Later research, however, revealed its effects only appeared at moderate doses. Too little or too much of the drug created no response. The appropriate dosage, however, can decrease anxiety in healthy people. It also reduces psychotic symptoms such as hearing voices or thinking incoherently. In addition, cannabidiol induces sleepiness and may protect epileptics against seizures (Zuardi & Guimaraes, 1997).

Research has also addressed the impact of cannabidiol on THC's effects. The two cannabinoids in combination may create different experiences from either drug alone. Synergistic interactions like this can be very difficult to study. Drug lore suggests that cannabidiol minimizes THC's psychoactive effects and delays their onset. Yet formal studies reveal that the interaction is not quite so simple. Cannabidiol may exaggerate some of THC's effects while attenuating others. It may increase THC-induced euphoria, but limit the production of anxiety and disordered thinking. Cannabidiol slows THC metabolism in the liver. Thus, a dose of THC combined with cannabidiol will create more psychoactive metabolites than the same dose of THC administered alone (Bornheim, Kim, Perotti, & Benet, 1995). By slowing THC's metabolism, cannabidiol can exaggerate some of its effects, including euphoria and the subjective sense of feeling high.

Although cannabidiol slows THC metabolism, it also may limit the drug's negative side effects. THC alone can produce anxiety, panic, and psychotic symptoms, particularly at high doses. Cannabidiol not only decreases anxiety on its own, but it also buffers against THC-induced panic and discomfort. Cannabidiol minimizes psychotic symptoms like bizarre thoughts or odd perceptions. Thus, it may attenuate these negative aspects of THC intoxication. Strains of marijuana that lack cannabidiol may produce more panic or psychotic effects. These findings may prove par-

ticularly useful given recent research on dronabinol (Marinol), the synthetic version of THC used to treat nausea and weight loss. Negative side effects of this drug might decrease if physicians combined it with cannabidiol (Zuardi & Guimaraes, 1997).

Cannabis Preparations

Users can ingest cannabinoids in a number of forms, including marijuana, hashish, hash oil, and synthetic medications. Nearly all parts of the marijuana plant contain psychoactive ingredients, but most of the cannabinoids appear in the resinous glands and flowering tops. Thus, the price for glands or tops is markedly higher than for other parts of the plant. Different cannabis preparations have different names. "Marijuana," a Spanish word purportedly coined in Mexico, originally meant cheap tobacco. The term may stem from the Portuguese expression "mariguango," which means intoxicant (Maisto et al., 1995). Later the word referred to the dried leaves and flowers of cannabis. Residents of India distinguish among three forms: bhang, ganja, and charas. Bhang is the dried leaves of the plant, comparable to marijuana. People smoke these leaves or combine them with milk and spices to form a drink that is also called bhang. Ganja refers to the sap-carrying tops of female plants in India, but in Jamaica the term applies to the leaves as well. Charas is hashish, the dried resin separated from the flowers and pressed together (McKim, 1997).

Many legends surround hashish. A well-known report concerns an exotic technique for collecting resin. Harvesters allegedly pranced naked through sunny fields of cannabis, gathering shiny sap and scraping it gently from their bodies to form cakes. These tales sound like contemporary urban legends or a marketing strategy for modern dealers. Nevertheless, comparable stories appeared as early as the 1850s. Most reporters from that era found workers who wore leather aprons to catch resin as they ran through fields. These workers then told of naked harvesting in other locations (Johnston, 1855; Von Bibra, 1855).

Stories like these may have been an attempt to fool outsiders, similar to the way children from farms tell children from the city that chasing a cow will turn her milk to cottage cheese. Perhaps poorer gatherers of hashish resin could not afford leather aprons. Whatever the arrangement in the past, modern hashish production does not employ naked trips through fields. Instead, manufacturers shake the resinous glands from the

plants and press it into hash. Others simply form blocks by pressing hash oil into powdered cannabis.

In addition to ganja, bhang, and charas or hashish, hash oil also appears in the illicit drug market. Producers create this viscous liquid by boiling hashish or cannabis in a solvent, straining it through a filter, and then letting the solvent evaporate, leaving the oil. The process can be extremely dangerous given the flammability of the solvents, which usually include alcohol or ether (Gold, 1989). The risk may prove worthwhile because hash oil commands higher prices. The oil has the potential to generate huge profits because it is relatively compact and easy to smuggle. It is often more potent than hashish or cannabis, too. Yet hash oil is not particularly popular. Smoking the oil by itself can require special glass pipes as part of a messy and cumbersome process. The solvents used to form the oil may be unhealthy to smoke. Most users find hashish or cannabis easier to ingest and potent enough to create the effects they desire (Clarke, 1998).

Synthetic cannabinoids also exist; they are usually ingested orally. Dronabinol (Marinol), a synthetic version of THC suspended in sesame oil, can treat poor appetite, nausea, and vomiting (e.g., Lefkowtiz et al., 1995). Current studies also address dronabinol's efficacy as a treatment for spasticity associated with multiple sclerosis and pain after surgery (Hanigan, Destree, & Truong, 1986). Some initial work also suggests that this drug might help disturbed behavior in Alzheimer's patients (Volicer et al., 1997). The abuse potential for this substance appears to be minimal. It is the only cannabinoid approved for medical use in the United States. Nabilone (Cesamet), an analogue of THC available in the United Kingdom, also limits nausea, vomiting, and spasticity (e.g., Steel et al., 1980). Levonantradol, another synthetic THC analogue unavailable in the United States, shows some promise in treating acute surgical pain, nausea, and vomiting (Jain et al., 1981; Tyson et al., 1985). Apparently, no pharmaceutical company has pursued its development as a medication (IOM, 1999). The medicinal uses of these synthetic cannabinoids appear in more detail in chapter 8.

Potency

Cannabis preparations vary dramatically in their effects. The most common indicator of potency is the percentage of delta-9 tetrahydrocannab-

inol (THC). Although THC is not the only source of psychoactive effects in the plant, it is the most abundant chemical that clearly alters subjective experience. Hashish typically contains 20% THC, with some estimates as high as 50%. Hash oil can contain up to 70% THC. Yet each of these products can vary dramatically in potency. Some samples of hash oil and hashish contain no THC at all. These products with no THC obviously create few subjective effects, except for those that arise from expectancy. Hashish and hash oil with the highest potencies often cause the most dramatic experiences of intoxication.

Marijuana also shows considerable variation in potency depending upon the plant strain, growing conditions, and storage. Some varieties of plants contain more THC than others. *Cannabis sativa* used for industrial hemp often contains less than 1% THC. Smoking marijuana this low in potency does not change subjective experience. Marijuana with less than 1% THC has the same effects as a placebo (Zimmer & Morgan, 1997). Thus, hemp products are not psychoactive. No one will grow intoxicated from smoking the various shampoos, soaps, or clothes currently manufactured from these plants. Psychoactive strains of marijuana typically contain 2 to 5% THC, but concentrations as high as 22% have been documented (Iversen, 2000). The moisture and temperature of the growing season can alter potency. Storage in hot environments can degrade the cannabinoids and lower THC content (Clarke, 1998). Exposure to light also accelerates the breakdown of THC. A year of storage in a bright place can produce nearly three times the decrease in THC as a year of storage in a dark place (Brazis & Mathre, 1997).

Many media reports suggest that cannabis has increased in potency quite dramatically in recent years. These reports have generated considerable debate. Yet the magnitude of the increase is difficult to document. In addition, the tacit assumption that increased potency translates into greater danger from the drug may not be true. Reports of a stronger drug actually began over 30 years ago. By the middle of the 1980s, some authors suggested that marijuana's potency had increased by a factor of 100 (MacDonald, 1984). These claims clearly suffered from exaggeration or misinformation. Other arguments about increased potency arose from the University of Mississippi's Potency Monitoring Project. This program reports the average THC content of cannabis taken in drug arrests. Estimates were extremely low in the 1970s, sometimes below 1%. As discussed above, cannabis with this little THC has no impact on subjective experience. The idea that a drug with no effects would increase in pop-

ularity over the years makes little sense. Thus, these estimates from the 1970s were probably poor reflections of the amount of THC in marijuana available at the time.

Investigators hypothesize that the data from the Potency Monitoring Project underestimate the true amount of THC in marijuana from the 1970s. First, the estimates were based on very few samples of seized cannabis. In some years there were no more than 50 samples to analyze (Potency Monitoring Project [PMP], 1974–1996). In addition, police may have stored the marijuana in hot lockers that allowed the THC to degrade rapidly (Mikuriya & Aldrich, 1988). Despite the small samples and poor storage, the average THC content in 1976 was 2% (ElSohly, Holley, & Turner, 1985).

An alternative source of potency information, an independent laboratory in California, analyzed many more samples than the Potency Monitoring Project and found a large range in THC concentration. In 1973, this laboratory tested over 100 samples and found that marijuana had an average THC content of 1.6 % (Ratcliffe, 1974). Later analyses ranged up to almost 8% THC (Perry, 1977). Thus, the idea that all cannabis of the 1970s had less than 1% THC seems unlikely. Ratcliffe's (1974) estimate of 1.6% may be conservative but credible; the 1976 estimate of 2% may be closer to the truth.

Potency data from the 1980s through the middle of the 1990s suggest that THC content continued to vary dramatically from strain to strain and sample to sample. With improved storage techniques and much larger samples, the Potency Monitoring Project found THC concentrations varied from 2% to almost 4%. Average concentrations approached 4% THC in 1984, 1988, 1990, and 1991 (PMP, 1974–1996). Trends in the rest of the 1990s showed comparable THC content, with a peak around 4.5% THC in 1997. Other cannabinoids like cannabinol and cannabidiol have not increased in concentration over the years (ElSohly et al., 2000). Thus, claims of 1,000% (Cohen, 1979) or 10,000% (MacDonald, 1984) increases in marijuana potency are clearly inaccurate. A threefold increase from approximately 1.5% in the early 1970s to 4.5% in the late 1990s may be closer to the truth. A simple doubling from an average of 2% to an average of 4% also seems plausible.

Although many media reports warn that increased potency translates into greater danger, data suggest otherwise. The implications of a two or threefold increase in THC concentration remain unclear. Marijuana with greater amounts of THC may not prove more hazardous than weaker

cannabis. First, acute administration of the drug is essentially nontoxic. No one has ever died from THC poisoning. Smoking enough cannabis to ingest a lethal amount of THC may be physically, if not financially, impossible.

Estimates of a fatal dose of any drug arise from some rather gruesome animal research. Different groups of animals receive large amounts of a drug until a particular dosage kills 50% of them. Researchers refer to the dose that is lethal for 50% of the animals as the LD 50. Investigators then extrapolate from these data to estimate a lethal dose for humans. The LD 50 for THC is approximately 125 mg for every kilogram of body weight (Nahas, 1986). Thus, a 160-pound (approximately 73-kilogram) person would need 9,125 mg of THC to have a 50% chance of dying. A typical marijuana cigarette weighs one gram and contains roughly 20 mg of THC, suggesting that roughly 450 joints would prove fatal. Furthermore, at least 50% of the THC is destroyed in the burning process or lost to sidestream smoke. Given this loss, 900 joints would be a more appropriate estimate of a fatal amount (Doweiko, 1999). The 900 joints would weigh roughly 2 pounds. Although experienced users tell many exaggerated tales about smoking large amounts of cannabis, this dosage exceeds 100 times the quantity typically consumed by the heaviest users.

Given the limited fear of lethal overdose, marijuana with larger percentages of THC may actually have some benefits. Stronger cannabis may lead to smoking smaller amounts in order to achieve desired effects. Smoking smaller quantities could provide some protection against the health problems normally associated with inhaling smoke. Smokers may take smaller, shorter puffs when using more potent marijuana (Heishman, Stitzer, & Yingling, 1989). Smoking less may decrease the amount of tars and noxious gases inhaled, limiting the risk for mouth, throat, and lung damage (Matthias, Tashkin, Marques-Magallanes, Wilkins & Simmons, 1997). Obviously, avoiding smoke completely would eliminate these problems. Thus, eating cannabis products may have fewer negative consequences than smoking them. Comparisons between these two ways of administering the drug appear next.

Cannabinoid Administration

A thorough understanding of marijuana's effects requires some knowledge of how it enters a biological system. Drugs can penetrate the body

in many ways. Humans inject drugs intramuscularly (into muscles) or intravenously (into veins). They also inject subcutaneously (under the skin), a process known as "skin popping." People snort drugs intranasally. Some substances can be absorbed sublingually by placing them under the tongue. A few drugs can dissolve through the skin in transdermal administration, like the ubiquitous nicotine patch. None of these methods is particularly common for marijuana. Cannabis has two popular routes of administration: inhalation (smoking) and oral ingestion (eating). In addition, researchers have examined intravenous injections of THC and rectal administration via the marijuana suppository. Drug companies have also proposed a deep lung aerosol, a nasal spray, a nasal gel, and a sublingual preparation of synthetic THC (IOM, 1999).

All these alternative techniques for administering the drug remain relatively rare, but inhalation is quite common. In addition to cannabis, humans inhale nicotine, opium, crack and freebase cocaine, methamphetamine, glue, gasoline, and anesthetics. Inhalation serves as one of the fastest modes of administration for any drug, and THC is no exception. Smoke held in the lungs contacts the bloodstream directly through a rich network of capillaries. This blood travels almost immediately to the brain, the site of the majority of cannabinoid receptors. Thus, the first hints of intoxication can appear within 10 seconds of exhaling the smoke (Levinthal, 1999). People may reach their peak blood concentration of THC while still smoking. The rapid absorption during smoking parallels the dramatic increases associated with intravenous doses of THC. THC dissolves readily in fatty tissues of all sorts, but eventually travels through the blood to the liver and kidneys. It is subsequently metabolized and excreted.

Several factors influence the amount of THC absorbed during smoking. Larger puffs held deeply in the lungs for a long time create the most dramatic effects. At least 30% of the THC in cannabis disappears in the combustion of smoking. More cannabinoids escape while the marijuana cigarette burns between puffs (Davis, McDaniel, Cadwell, & Moody, 1984). Some studies suggest that experienced smokers can take in more THC than inexperienced ones, that is, they may inhale more efficiently. Experienced smokers seem to know their lung capacity and understand the amount of smoke that they can hold without coughing. In contrast, inexperienced smokers may take larger puffs that they subsequently cough out, or smaller puffs that do not provide much of the psychoactive chemicals.

In laboratory studies, heavy users absorbed approximately 27% of the THC available in a joint; light users absorbed only about 14% (Ohlsson, et al., 1982; Ohlsson, Agurell, Lindgren, Gillespie, & Hollister, 1985). The increased efficiency of inhalation in experienced users may account for a curious phenomenon called reverse tolerance or sensitization. Many regular users of cannabis report rapid effects at extremely low doses of the drug. Researchers once posited that people grew more and more sensitive to the drug with repeated exposure, allowing them to experience the subjective effects with less and less of the substance.

Limited absorption may contribute to the minimal effects that novice smokers report the first few times they try cannabis. Many eventually learn to inhale and report more impact from the drug. Some never learn to inhale and subsequently run for public office. The amount of THC an individual assimilates while smoking can vary dramatically. Yet users rarely complain about an inability to absorb enough THC. The effects of smoking are rapid, and people can modify their doses quite readily. A detectable increase in dosage is usually a mere puff or two away. Instead, complaints related to smoking concern irritation of the mouth, throat, and lungs. These complaints occasionally lead a smoker to eat marijuana or hashish instead.

Oral administration has the longest history of all the techniques for using drugs, beginning with alcohol consumption around 8000 B.C. (Roueche, 1963). Most substances taken by mouth must travel the entire gastrointestinal tract, which contains several natural barriers to absorption. These barriers are important in helping minimize the toxic effects of many substances. The interior of the stomach, a highly acidic environment, can break down a variety of noxious chemicals. Unfortunately, this environment also neutralizes potentially helpful medications. For example, stomach acid destroys insulin, making oral administration of this drug useless.

Drugs that survive the stomach pass to the small intestine. Membranes between the intestinal wall and surrounding blood capillaries include two layers of fat molecules. Thus, only fat-soluble substances pass into these capillaries, which then send the drug to the liver for further metabolism. Those drugs that survive the liver metabolism reach general circulation. Yet entering the bloodstream is no guarantee of reaching the brain. The blood-brain barrier, a tightly knit bed of capillaries, keeps all but a few of the most lipid-soluble substances from reaching the brain. This barrier

separates the brain from the circulatory system, minimizing the impact of any potential toxins.

Given all the natural barriers inherent in oral administration, it remains dramatically slower than inhalation. Eating marijuana may create smaller effects than smoking an equal amount because so much of the drug breaks down in digestion. Eating cannabis or hashish leads to delayed, erratic absorption, depending upon the state of the gut at the time. A gram of cannabis might lead to extreme intoxication when smoked but hardly alter subjective experience if eaten after a full meal.

The concentration of THC also tends to peak much later after oral administration than after smoking. Concentrations peak between 1 and 6 hours after eating THC in chocolate cookies and around 2 hours in sesame oil pills like dronabinol (Ohlsson et al., 1980; Ohlsson et al., 1985; Wall, Sadler, Brine, Harold, & Perez-Reyes, 1983). In one study, marijuana-laced brownies led to some effects in 30 minutes, but peak responses did not occur for 2½ to 3½ hours (Cone, Johnson, Paul, Mell, & Mitchell, 1988). Cannabis resin eaten in a meat sandwich also took at least 2 hours to create peak effects (Law, Mason, Moffat, Gleadle, & King, 1984). Thus, the digestive process decreases the bioavailability of the drug. Bypassing this digestive process via smoking enhances effects. The marijuana suppository also bypasses degradation in the liver and leads to greater availability of THC (Mattes, Shaw, Edling-Owens, Engelman, & ElSohly, 1993).

Despite the potential drawbacks of decreased availability and slower initiation of effects, orally administered marijuana has developed quite a history. Cannabis products heated in oil or butter and combined with sweets have served as confections for centuries (Abel, 1980). Modern recipes for brownies, soup, meatloaf, guacamole, banana bread, and cookies can include cannabis. Hashish recipes for cookies, brownies, and soup are also quite common (Powell, 1971). Tinctures made from marijuana soaked in alcohol also provide a vehicle for oral administration. In addition, dronabinol, the synthetic version of THC dissolved in sesame oil, is marketed in capsules for easy swallowing. Eating the drug avoids the obvious throat and mouth irritation and risk for lung problems that accompany smoking.

THC Metabolism

The amount of time required to metabolize THC has shown considerable variation from person to person and study to study. The period required to eliminate THC from the body should not be confused with the duration of the drug's psychoactive effects. People stop feeling high long before THC has left their bodies. Intoxication rarely last more than a few hours, with orally administered doses lasting longer than smoked cannabis. After an intravenous injection of THC, blood levels peak almost immediately and then decrease by 90% in the first hour. This rapid drop does not mean that the drug has exited the body; it simply leaves the blood to dissolve into fat tissue. THC in the blood partitions into fat tissue, then leaks slowly from fat to be degraded and excreted. Although media accounts of marijuana's effects often treat THC's fat solubility as a novelty, sedatives like the barbiturates and benzodiazapines are stored in fat, too. After the first hour, blood levels of THC do not drop as rapidly. As THC from the blood is eventually excreted in urine and feces, THC stored in fat returns to circulation, but in doses too small to create psychoactive effects.

Researchers express the time required to metabolize a drug as its half-life—the period required to break the dose down to 50% of its original amount. Suppose the half-life of a hypothetical drug was one day. People who absorbed 100 mg of this drug would reduce it to 50 mg in one day. The next day they would again cut the available dose in half, to 25 mg. The next day would decrease the amount to 12.5 mg, and so on. Zeno's paradox would suggest that this consistent splitting in two would actually never lead to a blood level of zero. The amount would decrease by 50% repeatedly, growing smaller and smaller, but it would never disappear. Practically, drugs reach an undetectable concentration after 4.5 or 5 half-lives (Diaz, 1997).

Estimates of the half-life of THC based on urinary excretion show incredible variation. Research estimates of THC's half-life range from as little as 19 hours (Hunt & Jones, 1980) to as much as 4 days (Johansson, Arguell, Hollister, & Halldin, 1988). Early work suggested that users might grow more efficient at metabolizing THC as they gain experience with the drug (Lemberger, Axelrod, & Kopin, 1971). This study found a half-life of 28 hours for chronic smokers, but naive users took 57 hours to metabolize half of the dose. These results had considerable intuitive

appeal because they helped explain marijuana tolerance. The authors of this research implied that people grew tolerant to the drug because they essentially eliminated it more readily.

In contrast to evidence of increased rates of metabolism in experienced users, a later study found that people who had received THC each day for two weeks did not metabolize faster than moderate users who had not ingested daily doses (Hunt & Jones, 1980). In addition, the study with the longest estimated half-life used chronic, regular smokers (Johansson et al., 1988). These results seem confusing in light of the previously reported shorter half-life for heavier users. Thus, THC's rate of metabolism may not increase with repeated use. Most studies show a half-life between 1 and 1.5 days (Ohlsson et al., 1982; Ohlsson et al., 1985; Wall et al., 1983). A recent study using an extremely sensitive measuring technique and a two-week follow-up period found THC half-life ranges up to 2.5 days (Huestis & Cone, 1998). This study used the best methods available and suggests that a dose of THC leaves the body completely after 12 or 13 days.

The extreme variation in the estimates for the half-life of THC may stem from studying small samples of people over relatively short durations, using measurement techniques that vary in sensitivity. This large range of estimates likely reflects individual differences among people. Some simply metabolize more quickly than others. Techniques that do not rely on urine samples suggest THC stays in the body even longer. Analysis of fat cells rather than urine samples has revealed that the drug can remain in the body up to a month in some people (Johansson, Noren, Sjovall, & Halldin, 1989). Popular authors imply that this long elimination period is the norm (DuPont, 1984), but many people metabolize THC faster. Despite all the variability in elimination periods, marijuana does appear to have a longer half-life than some other drugs. For example, nicotine's half-life is about 2 hours; caffeine's is 3 to 6 hours (Henningfield, Cohen, & Pickworth, 1993). However, some sedatives that are more fat soluble show half-lives around 2 days or more (Diaz, 1997). Thus, marijuana takes more time to metabolize than some drugs, but less than others.

Popular authors often misinterpret THC's long half-life by frequently implying that intoxication or some sort of residual effect of the drug remains for weeks at a time. Yet intoxication dissipates in a couple of hours. The amount of THC released gradually from fat cells does not create any subjective, cognitive, or emotional effects but may register on

drug tests. Thus, a person may test positive for cannabis even a week or two after smoking, when all signs of intoxication have clearly terminated (Zimmer & Morgan, 1997). A number of underground legends suggest that goldenseal, cranberry juice, or various other concoctions might shorten this period of testing positive; no systematic research addresses this question. Drinking enormous quantities of fluids may dilute THC metabolites in the urine and alter the outcome of a test, but these fluids do not actually alter metabolic rate (Coombs & West, 1991).

Cannabinoid Receptors

Once cannabinoids enter the body, they must find a site to create their effects. The quest to understand the biological function of cannabinoids has generated a large body of research. Initial studies tracked radioactive THC through the body. This work revealed that THC attached to all the surfaces of the neuron, suggesting that it might alter the permeability of cell membranes to create its impact (Makriyannis & Rapaka, 1990). Researchers were familiar with the idea of drugs altering membrane permeability because some of alcohol's effects may stem from a comparable process (Doweiko, 1999).

Later work revealed that at least some of marijuana's impact could not arise solely from changes in the permeability of cell membranes. Newer studies found that cannabinoids could inhibit the synthesis of an extremely important compound, cyclic adenosine monophosphate (cyclic AMP or cAMP), which helps initiate nerve impulses (Howlett, Johnson, Melvin, & Milne, 1988). Other drugs that work in this way have special receptors that alter the cAMP. These receptors inhibit adenylyl cyclase (AC), the enzyme used to make cAMP. Examinations of every receptor known to inhibit AC revealed that none of them responded to the cannabinoids.

With all these other receptors ruled out, researchers concluded that cannabis must work via its own site. Investigators soon identified the cannabinoid receptor and mapped its distribution in the brain (Bidaut-Russell, Devane, & Howlett, 1990; Devane, Dysarz, Johnson, Melvin, & Howlett, 1988 Herkenham et al., 1990). The cannabinoid receptors in the nervous system, which are known as the CB1 type, are quite numerous. By way of comparison, CB1 receptors are 10 times more abundant than mu opiod receptors, the sites of action for morphine. After

researchers identified the CB1 receptor in the brain, other work revealed a second site of action in the immune system. This receptor was dubbed, surprisingly, CB2.

Contrary to what we all learned in high school courses on biology, receptors are not locks that either open or close in response to some key. The cannabinoid receptors (and all others) are proteins—strings of amino acids that span the membrane of the cell. Some of the amino acids are embedded in the cell membrane; some are implanted inside the cell; others extend outside. Cannabinoids bind with the portion of the receptor outside the cell and trigger activity inside. Different cannabinoids bind in different ways, leading to varied amounts of activity within the nerve cell.

One action triggered by the cannabinoid receptor is the inhibition of AC and subsequent inhibition of cAMP, as mentioned above. Thus, any process that requires cAMP will slow down if the cannabinoid receptor has been activated. The receptor also opens the potassium channels of the neuron, which decreases its rate of firing. Unlike the potassium channels, calcium channels close when the cannabinoid receptor activates. Closed calcium channels decrease the release of neurotransmitters. Thus, by inhibiting cAMP, slowing the nerve's firing rate, and decreasing neurotransmitter release, cannabinoids alter the communication between nerve cells. These actions may account for many of the effects of THC as well as other cannabinoids.

The cannabinoid receptors and their associated brain systems do not work in a vacuum. Any alterations of one neurotransmitter can change the functioning of others. THC clearly creates changes in the dopamine system, as cocaine, amphetamine, nicotine, and alcohol do (Koob & Le Moal, 1997). The cannabinoids can enhance dopamine's activation of movement, suggesting that they might help treat Parkinson's disease (Sanudo-Pena & Walker, 1998). Cannabinoids can inhibit or enhance gamma-aminobutyric acid (GABA), the neurotransmitter that may contribute to alcohol's sedative effects (Pacheco, Ward, & Childers, 1993; Shen, Piser, Seybold, & Thayer, 1996). THC also interferes with acetylcholine, a neurotransmitter involved in memory. The effect on acetylcholine may underlie the memory problems associated with cannabis intoxication. Thus, the cannabinoid receptor and related neurotransmitter systems clearly play an important role in the functioning of the brain.

Human cannabinoid receptors are extremely similar to those found in rodents, suggesting that evidence from animal studies may apply to people (Gerard, Mollereau, Vassart, & Parmentier, 1991). Leeches, mollusks,

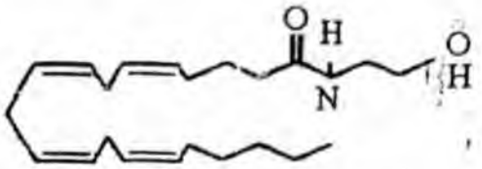

Figure 6.6. Anandamide. This cannabinoid occurs naturally in the body. It is the first endogenous cannabinoid discovered.

chickens, turtles, trout, and fruit flies have cannabinoid receptors, too (Howlett, Evans, & Houston, 1992; Stefano, Salzet, & Salzet, 1997). Even a primitive protozoan, the Hydra, has a cannabinoid receptor that appears to alter its feeding (De Petrocellis, Melck, Bisognor, Milone, & DiMarzo, 1999). The presence of the receptor in such a wide variety of species suggests that it must have an important and universal function. The ubiquitous presence of the CB1 and CB2 receptors inspired a search for substances within the body that might react at these sites.

The Body's Own Cannabinoids

It seems unlikely that so many animals would develop receptors simply to respond to some green weed. The identification of the cannabinoid receptors inspired the search for the body's own substances that might activate them. Studies of the functions of these endogenous cannabinoids could reveal a lot about how marijuana works, as well as how the brain works. Several endogenous chemicals appear to interact with the cannabinoid receptor. The two studied most are arachidonylethanolamine (anandamide) and 2-arachidonolyl-glycerol (2-AG), pictured respectively in figures 6.6 and 6.7.

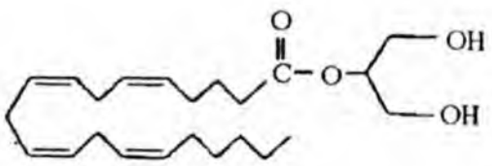

Figure 6.7. 2-arachidonolyl-glycerol (2-AG). This substance is the most abundant endogenous cannabinoid.

The first cannabinoid identified in the body was dubbed anandamide, from the Sanskrit word *ananda*, which means "bliss" or "ecstasy" (Devane et al., 1992). Its actual chemical name is arachidonylethanolamine. As the name suggests, its components include arachidonic acid and ethanolamine. Arachidonic acid serves as the building block of dozens of other chemicals, including some of those involved in the way aspirin works. Ethanolamine is related to alcohol. The anadamide molecule does not particularly resemble THC, but both interact with the cannabinoid receptors. Other natural compounds work on drug receptors but do not share the drug's molecular shape. For example, endorphins, the endogenous opiates, do not look much like opium or morphine. Yet they work at the same sites on the neuron. Clearly, molecules of different shapes may still connect to the same receptor. The critical aspects of the shape of the molecule are not known precisely.

Enzymes metabolize anandamide quite quickly, so its duration of action is shorter than THC's. Anandamide creates less intense effects than THC, perhaps because of this rapid breakdown. It also has only 25 to 50% of THC's affinity for receptors. Still, anandamide creates some of the same reactions. For example, this endogenous cannabinoid induces overeating (Williams & Kirkham, 1999) and lowers activity, body temperature, and pain sensitivity (Fride & Mechoulam, 1993). Researchers find anandamide in many of the brain areas rich in CB1 receptors, including the hippocampus, a structure involved in memory. Thus, this receptor may play a role in the deficits in short-term memory associated with marijuana intoxication. CB1 receptors also appear in the cerebellum, a motor center of the brain. The ability of cannabinoids to relieve spasticity and tremor may involve the receptors in this area (Baker et al., 2000).

Anandamide also appears in the thalamus, a structure involved with pain and emotion. Oddly, the thalamus has relatively few cannabinoid receptors. Perhaps anandamide works on other receptors in this structure. Anandamide works in systems outside the brain as well. It appears in spleen tissue, which has many CB2 receptors, and acts in the immune system (IOM, 1999). Anandamide even inhibits the growth of breast cancer cells (De Petrocellis et al., 1998). Research on anandamide's exact functions in the brain and immune system continues.

Several compounds related to anandamide also bind to cannabinoid receptors, including 2-arachidonolyl-glycerol (2-AG). The compound 2-AG is a prominent constituent of brain tissue, about 170 times more

abundant than anandamide (Stella, Schweitzer, & Piomelli, 1997). It clearly interacts with both the CB1 and CB2 receptors (Sugiura, et al., 1999; Sugiura et al. 2000). It also alters heart rate and blood pressure in mice (Jarai et al., 2000). Its exact role in other cannabinoid effects is not yet clear. Research on the endogenous cannabinoid system progresses rapidly. Studies have already isolated new substances that bind to cannabinoid receptors, but they have not been identified yet. Their precise biological impact remains unknown.

Conclusions

Marijuana contains more than 400 chemical components; at least 66 of them are cannabinoids unique to the plant. The most prevalent ones include delta-9-THC, cannabinol, and cannabidiol. THC causes cannabis's intoxicating effects. Cannabinol has about one-tenth the psychoactive effects of THC. At high doses, it can increase sleep and decrease body temperature. Cannabinol may decrease THC's psychoactive effects, particularly the stimulating aspects of the subjective experience, but it may also extend the duration of intoxication. Cannabidiol may decrease anxiety and psychotic symptoms as well as minimize seizures. In combination with THC, cannabidiol may increase THC concentrations, slow its metabolism, and limit any anxious or paranoid feelings associated with intoxication. Researchers have identified dozens of other cannabinoids, many with shapes similar to these three. All are soluble in fat, but their other chemical properties are not understood completely.

Many different preparations of cannabis exist, including wide varieties of hashish, hash oil, and marijuana. Their potencies can vary dramatically, with hashish containing up to 50% THC and hash oil running as high as 70% THC. Cannabis itself is often between 2 and 4% THC, with some claims of potency reaching markedly higher. Concerns about dramatic increases in potency over the last 30 years may stem from poorly analyzed or misrepresentative samples of cannabis from the 1970s. Assertions about increases in potency of 10 to 100 times seem extremely unlikely. THC concentrations have probably increased by a factor of 2 or 3. These increases may not justify alarm. THC is not toxic at high doses like alcohol, nicotine, or many other common drugs. High-potency marijuana may actually minimize risk for lung problems because less is required to achieve desired effects.

Users often smoke cannabis products and occasionally eat them. Researchers are currently experimenting with other ways to administer THC, including a nasal spray and rectal suppository. Smoke inhalation provides a rapid absorption of THC into the blood and brain, creating striking changes in subjective experience that often last a couple of hours. Ingesting marijuana or hashish requires digestion in the gastrointestinal tract and liver, delaying and reducing effects but lengthening their duration. Although intoxication rarely lasts more than a few hours, the complete elimination of THC clearly takes at least a few days. The chemical can remain in fat cells for up to one month. The fat cells eventually release the THC back into the blood stream, but in quantities too small to have any subjective effect. The liver breaks down this released THC and its metabolites are excreted.

In an effort to understand which neurotransmitter systems create THC's effects, investigators eventually identified two receptors that respond specifically to the cannabinoids. One receptor (CB1) exists in the brain and appears in high concentrations in areas involved with memory and motor control. The other receptor (CB2) is most prevalent in the immune system. The identification of this new neurotransmitter system has generated considerable research and reveals that the brain remains more complex than previously thought. The presence of these receptors inspired a search for the body's own chemicals that may activate them. Research has uncovered two natural cannabinoids; anandamide and 2-AG. These appear to mimic some of THC's effects, though they are less potent and have a shorter duration of action. Future research in this area will likely continue to inform us about the way this drug works as well as how the brain functions.

B

Sénat Senate

CANADA

CANNABIS:

OUR POSITION FOR A CANADIAN PUBLIC POLICY

REPORT OF THE SENATE SPECIAL COMMITTEE ON ILLEGAL DRUGS

SUMMARY REPORT

CHAIR

PIERRE CLAUDE NOLIN

DEPUTY CHAIR

COLIN KENNY

SEPTEMBER 2002

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GLOSSARY OF KEY TERMS

Abuse
Vague term with a variety of meanings depending on the social, medical and legal contexts. Some equate any use of illicit drugs to abuse: for example, the international conventions consider that any use of drugs other than for medical or scientific purposes is abuse. The Diagnosis and Statistical Manual of the American Psychiatric Association defines abuse as a maladaptive pattern of substance use leading to clinically significant impairment or distress as defined by one or more of four criteria (see Chapter 7). In the Report, we prefer the term excessive use (or harmful use).

Acute effects
Refers to effects resulting from the administration of any drug and specifically to its short term effects. These effects are distinguished between central (cerebral functions) and peripheral (nervous system). Effects are dose-related.

Addiction
General term referring to the concepts of tolerance and dependency. According to WHO addiction is the repeated use of a psychoactive substance to the extent that the user is periodically or chronically intoxicated, shows a compulsion to take the preferred substance, has great difficulty in voluntarily ceasing or modifying substance use, and exhibits determination to obtain the substance by almost any means. Some authors prefer the term addiction to dependence, because the former also refers to the evolutive process preceding dependence.

Agonist
A substance that acts on receptor sites to produce certain responses.

Anandamide
Agonist neurotransmitter of the endogenous cannabinoid system. Although not yet fully understood in research, these neurotransmitters seem to act as modulators as THC increases, the liberation of dopamine in nucleus accumbens and in the cerebral cortex.

At-risk use
Use behaviour which makes users at risk of developing dependence to the substance.

Cannabinoids
Endogenous receptors of the active cannabis molecules, particularly Delta 9-THC. Two endogenous receptors have been identified: CB1 densely concentrated in the hippacampus, basal ganglia, cerebellum and cerebral cortex, and CB2, particularly abundant in the immune system. The central effects of cannabis appear to be related only to CB1.

Cannabis
Three varieties of the cannabis plant exist: *cannabis sativa*, *cannabis indica*, and *cannabis ruredalis*. *Cannabis sativa* is the most commonly found, growing in almost any soil condition. The cannabis plant has been known in China for about 6000 years. The flowering tops and leaves are used to produce the smoked cannabis. Common terms used to refer to cannabis are pot, marijuana, dope, ganja, hemp. Hashish is produced from the extracted resin. Classified as a psychotropic drug, cannabis is a modulator of the central nervous system. It contains over 460 known chemicals, of which 60 are cannabinoids. Delta-9-tétrahydrocannabinol, referred to as THC, is

the principal active ingredient of cannabis. Other components such delta-8-tétrahydrocannabinol, cannabinol and cannabidiol are present in smaller quantities and have no significant impacts on behaviour or perception. However, they may modulate the overall effects of the substance.

Commission on narcotic drugs (CND)
The Commission on Narcotic Drugs (CND) was established in 1946 by the Economic and Social Council of the United Nations. It is the central policy-making body within the UN system for dealing with all drug-related matters. The Commission analyses the world drug abuse situation and develops proposals to strengthen international drug control.

Chronic effects
Refers to effects which are delayed or develop after repeated use. In the report we prefer to use the term consequences of repeated use rather than chronic effects.

Decriminalization
Removal of a behaviour or activity from the scope of the criminal justice system. A distinction is usually made between *de jure* decriminalization, which entails an amendment to criminal legislation, and *de facto* decriminalization, which involves an administrative decision not to prosecute acts that nonetheless remain against the law. Decriminalization concerns only criminal legislation, and does not mean that the legal system has no further jurisdiction of any kind in this regard. Other, non-criminal, laws may regulate the behaviour or activity that has been decriminalized (civil or regulatory offences, etc.).

Diversion
The use of measures other than prosecution or a criminal conviction for an act that nonetheless remains against the law. Diversion can take place before a charge is formally laid, for example if the accused person agrees to undergo treatment. It can also occur at the time of sentencing, when community service or treatment may be imposed rather than incarceration.

Depenalization
Modification of the sentences provided in criminal legislation for a particular behaviour In the case of cannabis, it generally refers to the removal of custodial sentences.

Dependence
State where the user continues its use of the substance despite significant health, psychological, relational, familial or social problems. Dependence is a complex phenomenon which may have genetic components. Psychological dependence refers to the psychological symptoms associated with craving and physical dependence to tolerance and the adaptation of the organism to chronic use. The American Psychiatric Association has proposed seven criteria (see Chapter 7).

Dopamine
Neuromediator involved in the mechanisms of pleasure.

Drug
Any chemical agent that alters the biochemical or physiological processes of tissues or organisms. In this sense, the term drug refers better to any substance which is principally used for its psychoactive effects. Also used to refer to illicit rather than licit (such as nicotine, alcohol or medicines) substances.

European Monitoring Centre on Drugs and Drug Addiction (EMCDDA)
The European Monitoring Centre was created in 1993 to provide member states within the EU objective, reliable and comparable information on drugs, drug addictions and their consequences. Statistical information, documents and techniques developed in the EMCDDA are designed to give a broad perspective on drug issues in Europe. The Centre only deals with information. It relies on national focal points in each of the Member States.

Fat soluble
Characteristic of a substance to irrigate the tissues quickly. THC is highly fat-soluble.

Gateway / Gateway Theory
Theory suggesting a sequential pattern in involvement in drug use from nicotine to alcohol, to cannabis and then to "hard" drugs. In regard to cannabis, the theory rests on a statistical association between the use of hard drugs and the fact that these users have generally used cannabis as their first illicit drug. This theory has not been validated by empirical research and is considered outdated.

Half-life
Time needed for the concentration of a particular drug in blood to decline to half its maximum level. The half-life of THC is 4.3 days on average but is faster in regular users than in occasional users. Because it is highly fat soluble, THC is stored in fatty tissues, thus increasing its half-life to as much as 7 to 12 days. Prolonged use of cannabis increases the period of time needed to eliminate it from the system. Even one week after use, THC metabolites may remain in the system. They are gradually metabolised in the urine (one third) and in feces (two thirds). Traces of inactive THC metabolites can be detected as long as 30 days after use.

Hashish
Resinous extract from the flowering tops of the cannabis plant transformed into a paste.

International conventions
Various international conventions have been adopted by the international community since 1912, first under the League of Nations, then under the United Nations, to regulate the possession, use, production, distribution, sale, etc., of various psychotropic substances. Currently, the three main conventions in force are the 1961 Single Convention, the 1971 Convention on Psychotropic Substance and the 1988 Convention against Illicit Traffic. Canada is a signatory to all three conventions. Subject to countries' national constitutions, these conventions establish a system of regulation where only medical and scientific uses are permitted. This system is based on the prohibition of source plants (coca, opium and cannabis) and the regulation of synthetic chemicals produced by pharmaceutical companies.

International Narcotics Control Board (INCB)
The Board is an independent, quasi-judicial organization responsible for monitoring the implementation of the UN conventions on drugs. It was created in 1968 as a follow up to the 1961 Single Convention, but had predecessors as early as the 1930s. The Board makes recommendations to the UN Commission on Narcotics with respect to additions or deletions in the appendices of the conventions.

Intoxication
Disturbance of the physiological and psychological systems through substance use. Pharmacology generally distinguishes four levels of intoxication: light, moderate, serious and fatal.

Joint
Cigarette of marijuana or hashish with or without tobacco. Because joints are never identical, scientific analyses of the effects of THC in their use are more difficult, especially to determine the therapeutic benefits of cannabis and to examine its effects on driving.

League of Nations
International organisation organization of Sstates until in existence until 1938; now the United Nations.

Legalization
Legislating under a regulatory system the culture, production, marketing, sale and use of substances. Although no such provision currently exist in relation to "street-drugs" (as opposed to alcohol or tobacco which are regulated products), a legalization system could take two forms: free of state control (free markets) and with state controls (regulatory regime).

Marijuana
Mexican term originally referring to a cigarette of poor quality. Has now become a synonym for cannabis in popular language usage.

Narcotic
Substance which can induce stupor or artificial sleep. Usually restricted to opiates. Sometimes used incorrectly to refer to all drugs capable of inducing dependence.

Office of National Drug Control Policy (ONDCP) USA
Created in 1984 under the Reagan administration, the Office is under the direct authority of the White House. It coordinates US policy on drugs. Its budget is currently US $18 billion.

Opiates
Substance derived from the opium poppy. The term opiate excludes synthetic opioids such as heroin and methadone.

Prohibition
Historically, the term most often refers to the period of national interdiction of alcohol sales in the United States between 1919 and 1933. By analogy, the term is now used to describe UN and State policies aiming for a drug-free society. Prohibition is based on the interdiction to cultivate, produce, fabricate, sell, possess, use, etc., some substances except for medical and scientific purposes.

Psychoactive substance
Substance which alters mental processes such as thinking or emotions. We prefer to use this term as it is more neutral than the term "drug" and does not refer to the legal status of the substance.

Psychotropic substance (see also psychoactive)
Used synonymously with psychoactive substance, however the term refers to drugs primarily used in the treatment of mental disorders, such as anxiolytics, sedatives, neuroleptics, etc. More specifically,the term refers to the substances covered in the 1971 Convention on Psychotropic Substances.

Regulation
System of control specifying the conditions under which the cultivation, production, marketing, prescription, sales, possession or use of a substance are allowed. Regulatory approaches may rest on interdiction (as for illegal drugs) or controlled access (as for medical drugs or alcohol). Our proposal of an exemption regime under the current legislation is a regulatory regime.

Tetrahydrocannabinol (Δ9-THC)
Main active component of cannabis, Δ9-THC is highly fat-soluble and has a lengthy half-life. Its psychoactive effects are modulated by other active components in cannabis. In its natural state, cannabis contains between 0.5% to 5% THC. Sophisticated cultivation methods and plant selection, especially female plants, lead to higher levels of THC concentration.

Tolerance
Reduced response of an organism and increased capacity to support the effects of a substance after a more or less lengthy period of use. Tolerance levels are extremely variable between substances, and tolerance to cannabis is believed to be lower than for most other drugs,including tobacco and alcohol.

Toxicity
Characteristic of a substance which induces intoxication, i.e., "poisoning". Many substances, including some common foods, have some level of toxicity. Cannabis presents almost no toxicity and cannot lead to an overdose.

United Nations Drug Control Program (UNDCP)
Established in 1991, the Program works to educate the world about the dangers of drug abuse. The Program aims to strengthen international action against drug production, trafficking and drug-related crime through alternative development projects, crop monitoring and anti-money laundering programs. UNDCP also provides accurate statistics through the Global Assessment Programme (GAP) and helps to draft legislation and train judicial officials as part of its Legal Assistance Programme. UNDCP is part of the UN Office for Drug Control and the Prevention of Crime.

World Health Organization (WHO) The World Health Organization, the United Nations' specialized agency for health, was established on April 7, 1948. WHO's objective, as set out in its Constitution, is the attainment by all peoples of the highest possible level of health. Health is defined in WHO's Constitution as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.

INTRODUCTION

The **Senate Special Committee on Illegal Drugs** addressed the question of drugs just as everyone else does, with the same preconceptions, attitudes, fears and anxieties we all share. Of course, we had at our disposal the 1996 study our colleagues conducted on government legislation dealing with illegal drugs, which had enabled them to hear a number of witnesses over several months. We also knew at the outset that research expertise would be available to us, but it is still difficult to overcome attitudes and opinions that we have long taken for granted. Whether one is in favour of enhanced enforcement or, on the contrary, greater liberalization, opinions often resist the facts and in a field such as this the production of facts, even through scientific research, is not necessarily a neutral undertaking. We, like you, have our prejudices and preconceptions. Together we must make the effort to go beyond such predispositions. That is one of the objectives of this report.

The public policy regime we propose expresses the fundamental premise underlying our report: *in a free and democratic society, which recognizes fundamentally but not exclusively the rule of law as the source of normative rules and in which government must promote autonomy as far as possible and therefore make only sparing use of the instruments of constraint, public policy on psychoactive substances must be structured around guiding principles respecting the life, health, security and rights and freedoms of individuals, who, naturally and legitimately, seek their own well-being and development and can recognize the presence, difference and equality of others.*

We are aware, as much now as we were at the start of our work, that there is no pre-established consensus in Canadian society on public policy choices in the area of drugs. In fact, our research has shown us that there are few societies where there is a broadly shared consensus among the general public, let alone between the public and experts. We are well aware, perhaps more so than at the outset, that the question of illegal drugs, viewed from the standpoint of public policy, has a broad international context and that we cannot think or act in isolation. We know our proposals are provocative, that they will meet with resistance. However, we are also convinced that Canadian society has the maturity and openness to welcome an informed debate.

PART I – GENERAL ORIENTATION

CHAPTER 1 – OUR MANDATE

"That a special committee of the Senate be struck to examine:

- *the approach taken by Canada to cannabis, its preparations, derivatives and similar synthetic preparations, in context;*
- *the effectiveness of this approach, the means used to implement it and the monitoring of its application;*
- *the related official policies adopted by other countries;*
- *Canada's international role and obligations under United Nations agreements and conventions on narcotics, in connection with cannabis, the Universal Declaration of Human Rights and other related treaties; and*
- *the social and health impacts of cannabis and the possible consequences of different policies;*

That the special committee consist of five senators, three of whom shall constitute a quorum;
That the Honourable Senators Banks, Kenny, Nolin, Rossiter and (a fifth Senator to be named by the Chief Government Whip) be named to the committee;
That the committee be authorized to send for persons, papers and records, to hear witnesses, to report from time to time, and to print from day to day such papers and evidence as may be ordered by it;
That the briefs and evidence heard during consideration of Bill C-8, An Act respecting the control of certain drugs, their precursors and other substances and to amend certain other Acts and repeal the Narcotic Control Act in consequence thereof, by the Standing Senate Committee on Legal and Constitutional Affairs during the 2nd Session of the 35th Parliament be referred to the committee;
That the documents and evidence compiled on this matter and the work accomplished by the Special Senate Committee on Illegal Drugs during the 2nd Session of the 36th Parliament be referred to the committee;
That the committee be empowered to authorize, if deemed appropriate, the broadcasting on radio and/or television and the coverage via electronic media of all or part of its proceedings and the information it holds;
That the committee present its final report no later than August 31, 2002; and that the committee retain the powers necessary to publicize its findings for distribution of the study contained in its final report for 30 days after the tabling of that report;
That the committee be authorized, notwithstanding customary practice, to table its report to the Clerk of the Senate if the Senate is not sitting, and that a report so tabled be deemed to have been tabled in the Senate."

The Committee's mandate is a continuation of the evolution of drug legislation passed by the Parliament of Canada in 1996, the *Controlled Drugs and Substances Act.* While this legislation was being studied by the Sub-Committee on Bill C-7 of the Standing Committee on Health of the House of Commons in 1994 and 1995, the vast majority of witnesses were highly critical of the bill. The most common criticisms concerned three points: first, the lack of basic principles or an expressed statement as to the purpose of the act; second, the fact that the bill perpetuated the prohibition system of the 1920s, and third, the absence of any emphasis on harm reduction and prevention criteria. Despite the amendments made by the Sub-Committee of the House, the testimony heard by the Senate Committee was equally critical. Witnesses noted that the Act did not categorize drugs on the basis of the dangers they represented, that it did not contain any specific, rational criteria and that it was impossible, particularly in view of the Act's complexity, to determine how it would be implemented in practice. All of these criticisms led that Senate Committee to "*propose energetically*" the creation of a Joint Committee of the House of Commons and the Senate that would review all Canadian drug legislation, policies and programs. However, the 1997 federal election intervened. Senator Nolin, convinced of the need for action and faced with the inaction of the House of Commons, tabled his first motion in 1999 - that a Senate Committee be struck and given a mandate to examine the legislation, policies and programs on illegal drugs in Canada. The motion was adopted by the Senate in April 2000.

However, that Committee was dissolved by general election of October 2000, and was restruck on March 15, 2001, with an amended mandate: the scope of its work was now restricted to cannabis "in its context". We chose to interpret this sentence broadly.

CHAPTER 2 – OUR WORK

At the Committee's public hearings, the Chair presented the research program as follows:

> *"In order to fully satisfy the mandate conferred upon the committee, the committee has adopted an action plan. This plan centres around three challenges. The first challenge is that of knowledge. We will be hearing from a wide variety of experts, both from Canada and afar, from academic settings, the police, legal specialists, medical specialists, the government sector and social workers. (...)*
> *The second challenge, surely the most noble challenge, is that of sharing knowledge. The committee hopes that Canadians from coast to coast will be able to learn and share the information that we will have collected. In order to meet this challenge, we will work to distribute this knowledge and make it accessible to all. We would also like to hear the opinions of Canadians on this topic and in order to do so, we will be holding public hearings in the spring of 2000 throughout Canada.*
> *And finally, the third challenge for this committee will be to examine and identify the guiding principles on which Canada's public policy on drugs should be based."*

In view of our mandate, including an obligation to provide Canadians with objective and rigorous information, we have emphasized rigour and openness throughout the entire process, an approach that was all the more important as opinions on all sides of the illegal drugs issue are strong and often categorical. But rigour is not enough. For the information to reach Canadians, we could not reserve it for our exclusive use, hence the second principle that guided us: openness. From the outset, we insisted that all our work be made available as soon as possible on our Web site and we entered into direct dialogue with our fellow citizens as well as with experts.

The Committee approved a research program divided into five major axes of knowledge, sub-dividing each one into specific issues:

- the socio-historical, geopolitical, anthropological, criminological and economic issues of the use and regulation of cannabis;
- the medical and pharmacological aspects of the consumption, use and regulation of cannabis;
- the legal aspects from a national perspective;
- the legal and political issues in an international perspective; and
- the ethical issues and Canadians' moral and behavioural standards.

In an attempt to answer these questions in the most effective and economical manner possible, the Committee agreed to perform two tasks concurrently: conduct a research program and hear expert witnesses–complementary activities. We asked the Parliamentary Research Branch and other researchers to produce syntheses and analyses of the relevant literature. In all, the Committee received 23 reports and benefited from summaries of work conducted in other countries, including attendance at international conferences. In all, the Committee held more than 40 days of public hearings in Ottawa and 10 other Canadian communities, hearing more than 100 witnesses from all backgrounds, from across Canada and abroad.

The second component of our program of work was to examine public opinion. That meant we had two closely related responsibilities. The first was a duty to inform, indeed, to educate. We hope those who are offended by that term will pardon our presumption, but we are convinced that on public policy topics that are societal issues, it is the duty of political leaders to transmit information that educates, not merely convinces. The level of knowledge about drugs, even about cannabis, perhaps the best known drug, is often limited and clouded by myth. Our second responsibility in taking public opinion into account was to go out and discover it. We did so in three ways. We publicized our work as widely and as openly as possible to enable everyone to learn about it and react to it. Many chose to write us, although they were relatively few compared with the number of people in this country. We commissioned a qualitative public opinion study. The focus groups conducted across the country as part of that study are described in detail in Chapter 10. We also held public hearings in eight

communities across the country, enabling citizens to come and tell us what they thought, what they knew and what they had experienced.

In order to be able to interpret all this knowledge and come to conclusions and recommendations, the third component of our work focussed on guiding principles.

CHAPTER 3 – GUIDING PRINCIPLES

It has now been thirty years since the Royal Commission of Inquiry on the Non-Medical Use of Drugs, the Le Dain Commission, studied issues similar to those we are studying today. Its report on cannabis, whose scientific conclusions on the effects of the drug were generally accepted by all members of the Commission, led to three reports: a majority report by three of the members, and two minority reports. Each expressed a different concept of the role of the State and of criminal law, and the roles of science and ethics in the choices that had to be made. Having examined each of these subjects, we have elected to set down the guiding principles that clarify the concept we have of the roles that the state, criminal law, science and ethics must play in the development of a public policy on cannabis.

Ethical considerations take us through what is, that is the realm of facts, to the realm of what should be, what would be desirable, moving from recognized facts to standards, then more importantly to values and finally to the means of passing on and above all implementing these values. This is why ethics was our first subject. As a guideline, we have adopted the principle that an ethical public policy on illegal drugs, and on cannabis in particular, must **promote reciprocal autonomy built through a constant exchange of dialogue within the community.**

We always find ourselves in paradoxical situations where, to a certain degree, each person has the free will to make decisions and makes free decisions for himself, while at the same time rules are established in order to regulate interaction with others, a complex and more or less formal, but appropriate approach. The goal of governance is freedom, and not control. It is a question of defining the goals of society through policies and programs of action that are then implemented through systems and processes and upheld by those who govern that permits the encouragement and affirmation of those goals for human action. The law, as a vehicle of choice of governance, does not merely express rules or limitations passed for the benefit of and on behalf of citizens, but seeks a reciprocal process of building social relationships through which people, citizens and governments, can constantly adjust their expectations of behaviour. We therefore accept as a guiding principle for governance that **all of the means the State has at its disposal must work towards facilitating human action, particularly the processes allowing for the building of arrangements between government of the citizenry and governance of the self.**

On the whole, the legal basis of the criminal law is weak where the prescribed standard first, does not concern a relationship with others and where the characteristics of the relationship do not establish a victim and a perpetrator able to recognize his/her

actions; second, has to find its justification outside fundamental social relationships; and third, results in the form of enforcement, the harmful effects of which undermine and challenge the very legitimacy of the law. Where criminal law is involved in these issues, the very standard prescribed by the law turns the perpetrator into the victim and tries to protect him from himself, something it can do only by producing a never-ending stream of knowledge that remains constantly out of his reach. In this context **only offences involving significant direct danger to others should be matters of criminal law**.

The Committee's Report - especially the second part - puts great emphasis on research-based knowledge. This focus is an attempt to do justice to the knowledge that has been developed over the past few decades. We considered it important and indeed necessary to give it detailed consideration. Indeed, the Committee recommends that the drive to acquire knowledge on specific issues we deem important be continued. We do not claim, however, to have answered the fundamental question of why people consume psychoactive substances, such as alcohol, drugs or medication. We were indeed surprised, given the quantity of studies conducted each year on drugs, that this area has not been covered. It is almost as if the quest for answers to technical questions has caused science to lose sight of the basic issue!

Scientific knowledge cannot replace either personal reflection or the political decision-making process. It supports those processes, science's greatest contribution to public drug policy. Our guiding principle is that **science, which must continue to explore specific areas of key issues and reflect on overarching questions, supports the public policy development process.**

These principles have guided our interpretation of the available information as well as our choice of recommendations; the reader should always keep them in mind when reading our report.

CHAPTER 4 – A CHANGING CONTEXT

This chapter puts the Committee's work in context. In recent years, in fact, in the past few months, events of some significance have taken place; some directly linked to illegal drugs, others far removed from them. Obviously, September 11 comes to mind. In social and political terms, the claims of medical users, of recreational users, within the changing context of drug use and, more generally, inter-generational conflict, have to be taken into account. Legislation passed in the aftermath of September 11, some provisions of which could affect police drug investigations, the fight against organized crime and the trial of the Hells Angels in Quebec, must also be taken into account. In legal terms, court decisions have had a direct effect on medical use and a decision will be rendered in the next few months by the Supreme Court on recreational use. In international terms, the fragility of the UNDCP and the development of a continental drug policy for the Americas are relevant to an understanding of certain issues that may even overdetermine national policy. Finally, globalization and the more extreme forms

of economic liberalism have been factors too, primarily in western societies but worldwide as well, in the increase of addictive behaviours, be they the use of drugs or other substitutes for social life.

PART II – CANNABIS: EFFECTS, TYPES OF USE, ATTITUDES

CHAPTER 5 – CANNABIS: FROM PLANT TO JOINT

This chapter first describes the cannabis plant and the various forms in which it becomes a consumer drug. We then take a brief look at the geographical origin of the cannabis plant and the routes along which it circulates in the modern world, noting at the same time current modes of production (soil-based and hydroponic) that have developed in certain regions of Canada. We then describe the pharmacokinetics of the cannabis plant, in particular its main active ingredients, and their metabolism in the body.

Available information on cannabis markets is weak and contradictory. Since 1997, the RCMP's annual reports on drugs suggest that 800 tons of cannabis circulate in Canada each year. Yet, many people told us that cannabis production has increased significantly and that cannabis has become more available than ever in this country. Data on the economic value of the cannabis market are no more reliable. We noted that:

- The size of the national production has significantly increased, and it is estimated that 50% of cannabis available in Canada is now produced in the country;
- The main producer provinces are British Columbia, Ontario and Quebec;
- Estimates of the monetary value of the cannabis market are unreliable. For example, if 400 tons are grown yearly in Canada, at a street value of $225 per ounce, the total value of the Canadian production would be less than $6 billion per year, less than the often quoted value of the BC market alone;
- An unknown proportion of national production is exported to the United States; and
- A portion of production is controlled by organized crime elements.

We heard many alarmist comments on the increased level of active ingredient (THC) in cannabis, however, it is currently impossible to estimate the average content of cannabis available in the market. More sophisticated growing methods have likely contributed to increasing the THC concentration. We observed that:

- In its natural state, cannabis contains between 0.5% and 3% THC. Sophisticated growing methods and genetic progress have made it possible to increase THC content in recent years, but it is impossible to estimate the average content of cannabis available in the market; it is reasonable to consider that content varies between 6% and 31%.

- THC is fat soluble and readily spreads in the innervated tissues of the brain; it reaches a peak in the blood plasma in less than nine minutes and falls to approximately 5% after one hour.
- The body is slow to eliminate THC and inactive THC metabolites can be detected in urine up to 27 days after use in the case of regular users.
- Psychoactive effects generally last two to three hours and may last as many as five to seven hours after use.

CHAPTER 6 – USERS AND USES: FORM, PRACTICE, CONTEXT

Who uses cannabis? How do the patterns of use in Canada compare to those in other countries? In what context is cannabis used? Why? What populations are most vulnerable? What are the social consequences of cannabis, specifically on delinquency and criminal behaviour? Most important, what trajectories do cannabis users follow, specifically with respect to consumption of other drugs?

At the very least, partial answers to these questions are prerequisite to establishing policy on a substance. In Canada, knowledge of patterns and contexts of cannabis use verges on the abysmal. In the early 1980s, the USA, the United Kingdom, and Australia introduced monitoring systems for the general population and the student population. In the last five years, a number of European countries have introduced data collection systems as part of the European Monitoring Centre for Drugs and Drug Addiction (EMCDDA). Canada, by contrast, has carried out only two epidemiological general population surveys specific to drugs (in 1989 and 1994), and only some provinces conduct surveys of the student population, using different methods and instruments that preclude data comparison. Furthermore, few sociological or anthropological studies are conducted on the circumstances or context of illegal drug use, specifically for cannabis. The result is that our pool of knowledge on users and characteristics of use is sorely lacking.

We have no explanation for this situation, at least no satisfactory explanation. In the 1970s, following up on the work done by the Le Dain Commission, Canada could have set up a trend monitoring system. In the 1980s, when Canada's Anti-Drug Strategy was adopted, to which the federal government allocated $210M over five years, a data collection system could well have been created. The fact that it was not could be due to an absence of leadership or vision, a fear of knowing, the division of powers among levels of government, or the absence of a socio-legal research tradition within the departments responsible for justice and health. In fact, all of the above are probable factors. Whatever the case, it is our contention that this situation, unacceptable by definition, requires timely remedial action. We must resign ourselves to working with the scarce Canadian data available, and, more significantly, the virtually non-existent comparable data. We will also look at studies and data from other countries.

The chapter is divided into four sections. The first covers consumption patterns in the population as a whole, then specifically in the 12-18 year age group and compares the patterns in various countries. In the adult population we observed that:

- The epidemiological data available indicates that close to 30% of the population (12 to 64 years old) has used cannabis at least once;
- Approximately 2 million Canadians over age 18 have used cannabis during the previous 12 months, approximately 600,000 have used it during the past month, and approximately 100,000 use it daily. Approximately 10% used cannabis during the previous year; and
- Use is highest between the ages of 16 and 24.

For youth in the 12-17 age group, we observed that:

- Canada would appear to have one of the highest rates of cannabis use among youths;
- Approximately 1 million would appear to have used cannabis in the previous 12 months, 750,000 in the last month and 225,000 would appear make daily use; and
- The average age of introduction to cannabis is 15.

The second section looks at what we know about reasons for and details on use, including origins and cultural differences. The third section deals specifically with cannabis user trajectories, including escalation. We have observed the following:

- Most experimenters stop using cannabis;
- Regular users were generally introduced to cannabis at a younger age. Long-term users most often have a trajectory in which use rises and falls;
- Long-term regular users experience a period of heavy use in their early 20s;
- Most long-term users integrate their use into their family, social and occupational activities; and
- Cannabis itself is not a cause of other drug use. In this sense, we reject the gateway theory.

The fourth and last section covers the relationship between cannabis use and delinquency and crime. Based on research evidence, we concluded that:

- Cannabis itself is not a cause of delinquency and crime; and
- Cannabis is not a cause of violence.

CHAPTER 7 – CANNABIS: EFFECTS AND CONSEQUENCES

When it comes to cannabis, one hears anything and its opposite. While in some areas more research is needed and in others research results are contradictory, there exists nevertheless a strong basis of information contradicting many of the myths that continue to be perpetuated.

This chapter is divided into five sections. The first is a collection of statements on the presumed effects of marijuana that the Committee heard or became aware of through its research. The following three sections examine the acute effects of cannabis, followed in turn by the physiological and neurological consequences, the psychological consequences and the social consequences. Then, because of its significance and the central place it holds in social and political concerns, we turn our attention specifically to the question of any possible dependence arising from prolonged use of cannabis.

With respect to the effects of cannabis, the Committee observed that:

- The immediate effects of cannabis are characterized by feelings of euphoria, relaxation and sociability; they are accompanied by impairment of short-term memory, concentration and some psychomotor skills; and
- Long term effects on cognitive functions have not been established in research.

The Committee has distinguished between use, at-risk use and excessive use. Quantities used, psychosocial characteristics of the users and factors related to use contexts and quality of the substance all come into play to explain the passage from one category to the other. On at-risk use, the Committee observed that:

- Most users are not at-risk users insofar as their use is regulated, irregular and temporary, rarely beyond 30 years of age;
- For users above 16, at-risk use is defined as using between 0.1 to 1 gram per day; and
- Available epidemiological data suggests that approximately 100,000 Canadians might be at-risk users.
- The Committee feels that, because of its potential effects on the endogenous cannabinoid system and cognitive and psychosocial functions, any use in those under age 16 is at-risk use.

With respect to excessive use we observed that:

- More than one gram per day over a long period of time is heavy use, which can have certain negative consequences on the physical, psychological and social well-being of the user. According to the epidemiological data available, there is reason to believe that approximately 80,000 Canadians above age 16 could be excessive users;
- For those between the ages of 16 and 18, heavy use is not necessarily daily use but use in the morning, alone or during school activities;
- Heavy use can have negative consequences for physical health, in particular for the respiratory system (chronic bronchitis, cancer of the upper respiratory tract);
- Heavy use of cannabis can result in negative psychological consequences for users, in particular impaired concentration and learning and, in rare cases and with people already predisposed, psychotic and schizophrenic episodes;
- Heavy use of cannabis can result in consequences for a user's social well-being, in particular their occupational and social situation and their ability to perform tasks; and

- Heavy use of cannabis can result in dependence requiring treatment; however, dependence caused by cannabis is less severe and less frequent than dependence on other psychotropic substances, including alcohol and tobacco.

CHAPTER 8 – DRIVING UNDER THE INFLUENCE OF CANNABIS

If there is one issue, other than the effects of cannabis use on young people or the effects of substance abuse, that is likely to be of concern to society and governments, then it is certainly the effect of the use of cannabis on the ability to drive a vehicle. We are already familiar with the effects of alcohol on driving and the many accidents involving injuries or deaths to young people. In spite of the decreases in use noted in recent years, one fatal accident caused by the use of a substance is one accident too many.

Next to alcohol, cannabis is the most widely used psychoactive substance, particularly among young people in the 16-25 age group. Casual use occurs most often in a festive setting, at weekend parties, often accompanied by alcohol. People in this age group are also the most likely to have a car accident and are also susceptible to having an accident while impaired.

Cannabis affects psychomotor skills for up to five hours after use. The psychoactive effects of cannabis are also dependent on the amount used, the concentration of THC and the morphology, experience and expectations of users. But what are the specific effects of cannabis on the ability to drive motor vehicles? What are the effects of alcohol and cannabis combined? And what tools are available to detect the presence of a concentration of THC that is likely to significantly affect the psychomotor skills involved in vehicle operation?

This chapter is divided into three sections. The first considers the ways of testing for the presence of cannabinoids in the body. The second analyzes studies on the known prevalence of impaired driving, in both accident and non-accident contexts. The third and last summarizes what is known about the effects of cannabis on driving based on both laboratory and field studies. As in the other chapters, the Committee then draw its own conclusions.

The Committee feels it is quite likely that cannabis makes users more cautious, partly because they are aware of their deficiencies and compensate by reducing speed and taking fewer risks. However, because what we are dealing with is no longer the consequences on the users themselves, but the possible consequences of their behaviour on others, the Committee feels that it is important to **opt for the greatest possible caution** with respect to the issue of driving under the influence of cannabis.

Given what we have seen, we conclude the following:

- Between 5% and 12% of drivers may drive under the influence of cannabis; this percentage increases to over 20% for young men under 25 years of age;
- Cannabis alone, particularly in low doses, has little effect on the skills involved in automobile driving. Cannabis leads to a more cautious style of driving. However it has a negative impact on decision time and trajectory. This in itself does not mean that drivers under the influence of cannabis represent a traffic safety risk;
- A significant percentage of impaired drivers test positive for cannabis and alcohol together. The effects of cannabis when combined with alcohol are more significant than is the case for alcohol alone;
- Despite recent progress, there does not yet exist a reliable and non intrusive rapid roadside testing method;
- Blood remains the best medium for detecting the presence of cannabinoids;
- Urine cannot screen for recent use;
- Saliva is promising, but rapid commercial tests are not yet reliable enough;
- The visual recognition method used by police officers has yielded satisfactory results, and
- It is essential to conduct studies in order to develop a rapid testing tool and learn more about the driving habits of cannabis users.

CHAPTER 9 - USE OF MARIJUANA FOR THERAPEUTIC PURPOSES

There has been renewed interest in the issue of the use of marijuana for therapeutic purposes in recent years, particularly in Canada. In the wake of an Ontario Court of Appeal ruling which found the provisions of the *Controlled Drugs and Substances Act* to be unconstitutional pertaining to the therapeutic use of marijuana, the federal Minister of Health made new regulations in July 2001 that give people with specified medical problems access to marijuana under certain conditions.

However, the scientific community, the medical community in particular, is divided on the real therapeutic effectiveness of marijuana. Some are quick to say that opening the door to medical marijuana would be a step toward outright legalization of the substance.

But none of that should matter to physicians or scientists. It is not a question of defending general public policy on marijuana or even all illegal drugs. It is not a question of sending a symbolic message about "drugs". It is not a question of being afraid that young people will use marijuana if it is approved as a medicine. The question, and the only question, for physicians as professionals is whether, to what extent and in what circumstances, marijuana serves a therapeutic purpose. Physicians should have to determine whether people with certain diseases would benefit from marijuana use and weigh the side effects against the benefits. If they do decide the patient should use marijuana, they then have to consider how he or she might get it.

This chapter is devoted to the history of the use of marijuana for therapeutic purposes and the status of contemporary knowledge of marijuana and synthetic cannabinoids. We then give a brief account of compassion clubs and other organizations that supply marijuana for therapeutic use, as well as various public policy regimes. We conclude with our views on medical use of marijuana. In a later chapter, we discuss which public policy regime would be most appropriate given the status of medical use of marijuana

We observed that:

- There are clear, though non-definitive indications of the therapeutic benefits of marijuana in the following conditions: analgesic for chronic pain, antispasm for multiple sclerosis, anticonvulsive for epilepsy, antiemetic for chemotherapy and appetite stimulant for cachexi;
- There are less clear indications regarding the effect of marijuana on glaucoma and other medical conditions;
- Marijuana has not been established as a drug through rigorous, controlled studies.
- The quality and effectiveness of marijuana, primarily smoked marijuana, have not been determined in clinical studies;
- There have been some studies of synthetic compounds, but the knowledge base is still too small to determine effectiveness and safety;
- Generally, the effects of smoked marijuana are more specific and occur faster than the effects of synthetic compounds;
- The absence of certain cannabinoids in synthetic compounds can lead to harmful side effects, such as panic attacks and cannabinoid psychoses;
- Smoked marijuana is potentially harmful to the respiratory system;
- People who smoke marijuana for therapeutic purposes self-regulate their use depending on their physical condition and do not really seek the psychoactive effect;
- People who smoke marijuana for therapeutic purposes prefer to have a choice as to methods of use;
- Measures should be taken to support and encourage the development of alternative practices, such as the establishment of compassion clubs;
- The practices of these organizations are in line with the therapeutic indications arising from clinical studies and meet the strict rules on quality and safety;
- The studies that have already been approved by Health Canada must be conducted as quickly as possible;
- The qualities of the marijuana used in those studies must meet the standards of current practice in compassion clubs, not NIDA standards;
- The studies should focus on applications and the specific doses for various medical conditions; and
- Health Canada should, at the earliest possible opportunity, undertake a clinical study in cooperation with Canadian compassion clubs.

CHAPTER 10 - CANADIANS' OPINIONS AND ATTITUDES

It is always difficult to gauge the public's opinions, attitudes and concerns. The traditional method of surveying a representative sample of the population was too expensive for our resources. Surveys also have limits that we discuss in more detail. However, we did commission a qualitative study using focus groups, the results of which are presented in this chapter. We also report the results of other surveys that we researched and considered. As well, many Canadians wrote to us or sent us e-mails, and others came out to our public hearings to participate. Obviously we cannot draw solid conclusions from this. The people who wrote to us were probably those to whom the issue is very important, regardless of which way they may lean. Some are cited in our Report but we must reiterate that no conclusion should be drawn from these opinions in terms of representativeness. No account of Canadians' opinions on and attitudes toward drugs in general would be complete without an examination of the role of the media in shaping those opinions and attitudes. In recent years, as a result of this Committee's work and other initiatives, various Canadian newspapers and magazines have run stories or have written editorials on the issue. These are the focus of the first part of the chapter. The next part presents the results of surveys and polls, including the survey we commissioned and surveys conducted in different provinces. The last part covers our understanding of what Canadians told us.

We observed the following:

- Public opinion on marijuana is more liberal than it was 10 years ago;
- There is a tendency to think that marijuana use is more widespread and that marijuana is more available than it used to be;
- There is a tendency to think that marijuana is not a dangerous drug;
- The concern about organized crime is significant;
- Support for medical use of marijuana is strong;
- There is a tendency to favour decriminalization or, to a lesser degree, legalization;
- People criticize enforcement of the legislation in regards to simple possession of marijuana; and
- There is a concern for youth and children.

PART III — POLICIES AND PRACTICES IN CANADA

CHAPTER 11 - A NATIONAL DRUG STRATEGY?

Based on the impo tance of the subject, it would probably surprise many Canadians to learn that only from 1987 to 1993 did Canada have a fully funded national drug strategy. It is true that Canada has had legislation dealing with the use of psychoactive substances since the passage of the *Opium Act* in 1908. This Act was followed by several pieces of criminal legislation over the years that increased federal

enforcement powers over psychoactive substances and expanded the list of illicit substances. These pieces of legislation have historically focused on the supply of psychoactive substances, adopting a prohibitionist approach to use. It is widely acknowledged now, however, that a more balanced approach is required if one is to deal effectively with those who abuse psychoactive substances.

This chapter recounts the development and implementation of the 1987 National Drug Strategy, which had as an objective the promotion of a balanced approach to the problem of psychoactive substance abuse. This is followed by a discussion of what became of the national strategy and what goals have been achieved.

We observed the following:

- Canada urgently needs a comprehensive and coordinated national drug strategy for which the federal government provides sound leadership;
- Any future national drug strategy should incorporate all psychoactive substances, including alcohol and tobacco;
- To be successful, a national drug strategy must involve true partnerships with all levels of government and with non-governmental organizations;
- Over the years, the intermittency of funding has diminished the ability to coordinate and implement the strategy; adequate resources and a long-term commitment to funding are needed if the strategy is to be successful;
- Clear objectives for the strategy must be set out, and comprehensive evaluations of these objectives and the results are required;
- At the developmental stage, there is a need to identify clear and shared criteria for "success";
- The core funding for the Canadian Centre on Substance Abuse (CCSA) has been insufficient for it to carry out its mandate; proper funding for the CCSA is essential;
- There is a need for an independent organization – the CCSA – to conduct national surveys at least every second year; there is also a need to achieve some level of consistency, comparability and similar time frames for provincially-based school surveys;
- Coordination at the federal level should be given to a body that is not an integral part of one of the partner departments; and
- Canada's Drug Strategy's should adopt a balanced approach – 90% of federal expenditures are currently allocated to the supply reduction.

CHAPTER 12 - THE NATIONAL LEGISLATIVE CONTEXT

Drugs have been prohibited for fewer than a hundred years; cannabis for slightly more than 75 years. It is tempting to think that the decisions made over the years to use criminal law to fight the production and use of certain drugs are in keeping with social progress and the advancement of scientific knowledge about drugs. But is this really the case? The history of legislation governing illegal drugs in Canada, like the analysis in Chapter 19 of the structure of international conventions, suggests that it is highly

doubtful. To what extent is such reasoning really rational? Is the rationale of the system of controls acceptable in the eyes of civil society, users as well as abstainers? What criteria motivated legislator decisions? Indeed, were there criteria? What motivated parliamentarians from Canada and elsewhere to prohibit certain substances, to control access to certain others, and to permit still others to be sold over the counter?

Knowing where we have been helps in understanding where we are going. That is the goal of this chapter, retracing the evolution of Canadian drug laws from 1908 to the present day. We have identified three legislative periods. The first, and longest, spans 1908 to 1960, the period of hysteria. We were told that drugs were made criminal because they are dangerous. Analysis of debates in Parliament and in media accounts clearly shows how far this is from truth. When cannabis was introduced in the legislation on narcotics in 1923, there was no debate, no justification, in fact many members did not even know what cannabis was.

The second period, much shorter, runs from 1961 to 1975, the search for lost reason. Following the explosion in drug use in the early 1960s and demands for reform from various sectors of society, governments appointed a commission of inquiry in Canada, the Le Dain Commission. Last comes the contemporary period at the beginning of the 1980s. Reform is not on the policy agenda any more and anti-drug policies have forged ahead.

In summary, we observed that:

- Early drug legislation was largely based on a moral panic, racist sentiment and a notorious absence of debate;
- Drug legislation often contained particularly severe provisions, such as reverse onus and cruel and unusual sentences; and
- The work of the Le Dain Commission laid the foundation for a more rational approach to illegal drug policy by attempting to rely on research data. The Le Dain Commission's work had no legislative outcome until 1996 in certain provisions of the *Controlled Drugs and Substances Act*, particularly with regard to cannabis.

CHAPTER 13 - REGULATING THERAPEUTIC USE OF CANNABIS

Cannabis has an extremely long history of therapeutic use, going back several thousands of years. It was often used for the same medical conditions it is used for today. With the development of the pharmaceutical industry in the last century, the medical community has gradually discontinued its use. Various factors may explain this. Developments in the pharmaceutical industry provided the medical community with more stable and better tested medication. The practice of medicine itself has changed and so has our conception of health. Then, at the turn of the 20th century, the plants from which opium, cocaine and cannabis are derived were banned by the international community, except for medical and scientific purposes. In the case of cannabis, no rigorous study had been done until recently.