	Estimated persons/ 1000 sq ft floor area. Use only when design occupancy is not known	Required ventilation air, cubic feet per minute per human occupant, (when the number is bracketed, refer to the notes).		Comments
		Minimum	Recommended	
Tea and Spice Handling, Packaging	–	20	25-30	
Packaging	–	20	25-30	
Refrigeration Plants, Steam Plants	–	20	25-30	•

*Thermal effects probably determine requirements
**Special solvent and exhaust problems handled separately
***Special contaminant control systems may be required
****Spaces maintained at low temperatures (−10 to 50 F) are not covered by these requirements unless the occupancy is continuous. Ventilation from adjoining spaces is permissible. When the occupancy is intermittent, infiltration will normally exceed the ventilation requirement. (See Chapter 23, Refrigeration Load, ASHRAE Handbook of Fundamentals, 1972).

Tobacco Processing				
Blending and Shredding	–	20	25-30	
Redrying, Reconstituting	–	20	25-30	
Cigar Manufacturers	–	20	25-30	
Cigarette Manufacturers, Pipe Tobacco Packaging	–	20	25-30	
Power Plants				
Control Rooms	–	10	15-20	
Boiler Rooms	–	35	40-45	
Generator Rooms	–	20	25-30	
Sewage Treatment Plants				
Control Rooms	–	10	15-20	
Compressor/Blower Motor Rooms	–	20	25-30	
Glass and Ceramic Manufacture				
Sand Handling and Mixing Areas	–	20	25-30	
Melting Furnace Support Areas	–	20	25-30	•
Platemaking, Pouring Areas	–	20	25-30	•
Bottlemaking, Blowing Machinery Areas	–	20	25-30	•
Fiber Spinning Areas	–	20	25-30	•
Grinding Rooms	–	20	25-30	••
Ceramics (Powder) Pressing and Molding Areas	–	20	25-30	
Potters Workrooms (wet)	–	20	25-30	
Kiln and Sintering Furnace Service Areas	–	20	25-30	•
Frit and Glaze Sprayrooms	–	20	25-30	••

*Thermal effects probably determine requirements
**Special contaminant control systems may be required

6.4. AGRICULTURAL
(Includes installations on farms, farmers' markets, grain elevators, etc.; for processing operations)

Fodder, Seed and Grain Handling, Storage	–	20	25-30	•

	Estimated persons/ 1000 sq ft floor area. Use only when design occupancy is not known	Required ventilation air, cubic feet per minute per human occupant, (when the number is bracketed, refer to the notes).		Comments
		Minimum	Recommended	
Animal Husbandry	–	20	25-30	•
Vegetable and Fruit Handling, Storage	–	20	25-30	••
Dairy Products	–	20	25-30	••
Natural Fiber Handling	–	20	25-30	
Tobacco Handling, Warehousing	–	20	25-30	
Mushroom Growing	–	10	15-20	

•Special contaminant control systems may be required
••Spaces maintained at low temperatures (−10 to 50 F) are not covered by these requirements unless the occupancy is continuous. Ventilation from adjoining infiltration will normally exceed the ventilation requirement. (See Chapter 23, Refrigeration Load, ASHRAE Handbook of Fundamentals, 1972).

6.5. INSTITUTIONAL

Schools				
Classrooms	50	10	10-15	
Multiple Use Rooms	70	10	10-15	
Laboratories	30	10	10-15	•
Craft Shops, Vocational Training Shops	30	10	10-15	•
Music, Rehearsal Rooms	70	10	15-20	
Auditoriums	150	5	5-7½	
Gymnasiums	70	20	25-30	
Libraries	20	7	10-12	
Common Rooms, Lounges	70	10	10-15	
Offices	10	7	10-15	
Lavatories	100	15	20-25	
Locker Rooms	20	(30)	(40)-(50)	••
Lunchrooms, Dining Halls	100	10	15-20	
Corridors	50	15	20-25	
Utility Rooms	3	5	7-10	
Dormitory Bedrooms	20	7	10-15	

•Special contaminant control systems may be required
••cfm/locker

Hospitals, Nursing and Convalescent Homes				
Foyers	50	20	25-30	
Hallways	50	20	25-30	
Single, Dual Bedrooms	15	10	15-20	
Wards	20	10	15-20	
Food Service Centers	20	35	35	
Operating Rooms, Delivery Rooms	–	20	– –	•
Ready Rooms, Recovery Rooms	–	15	– –	•
Amphitheatres	100	10	15-20	
Physical Therapy Areas	20	15	20-25	
Autopsy Rooms	10	30	40-50	
Incinerator Service Areas	–	5	7-10	••

For Shops, Restaurants, Utility Rooms, Kitchens, Bathrooms and other service items see Hotels.

•Special requirements or codes may determine requirements
••Special exhaust systems required

	Estimated persons/ 1000 sq ft floor area. Use only when design occupancy is not known	Required ventilation air, cubic feet per minute per human occupant, (when the number is bracketed, refer to the notes).		Comments
		Minimum	Recommended	
Research Institutes				
Laboratories (Light-duty, nonchemical)	50	15	20-25	*
Laboratories (Chemical)	50	15	20-25	*
Laboratories (Heavy-duty)	50	15	20-25	*
Laboratories (Radioisotope, Chemically and Biologically Toxic)	50	15	20-25	*
Machine Shops	50	15	20-25	
Darkrooms, Spectroscopy Rooms	50	10	15-20	
Animal Rooms	20	40	45-50	**
*Special contaminant control systems may be required **Special requirements or codes may determine requirements				
Military and Naval Installations				
Barracks	20	7	10-15	
Toilets/Washrooms	100	15	20-25	
Shower Rooms	100	10	15-20	
Drill Halls	70	15	20-25	
Ready Rooms, MP Stations	40	7	10-15	
Indoor Target Ranges	70	20	25-30	*
*Floor area behind firing line only				
Museums				
Exhibit Halls	70	7	10-15	
Workrooms	10	10	15-20	
Warehouses	5	5	7-10	
Prisons (See also Gymnasiums, Libraries, Applicable Industrial Areas)				
Cell Blocks	20	7	10-15	
Eating Halls	70	15	20-25	
Guard Stations	40	7	10-15	
Veterinary Hospitals				
Kennels, Stalls	20	25	30-35	*
Operating Rooms	20	25	30-35	*
Reception Rooms	30	10	15-20	
*Special requirements or codes may determine requirements				

6.6. ORGANIZATIONAL

Churches, Temples (See theaters, schools and offices)	–	–	–	
Legislative Halls				
Legislative Chambers	70	20	25-30	
Committee Rooms and Conference Rooms	70	20	25-30	

	Estimated persons/ 1000 sq ft floor area. Use only when design occupancy is not known	Required ventilation air, cubic feet per minute per human occupant, (when the number is bracketed, refer to the notes).		Comments
		Minimum	Recommended	
Foyers, Corridors	50	20	25-30	
Offices	10	10	15-20	
Press Lounges	20	20	25-30	
Press/Radio/TV Booths	20	20	25-30	
Public Rest Rooms	20	15	20-25	
Private Rest Rooms	–	20	30-50	
(For Food Service, Utilities, etc. see Hotels)				
Police and Fire Stations (See Prisons and Military Installations)	–	–	–	
Survival Shelters	–	5	–	•

•Special requirements or codes may determine requirements

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Public smoking under fire

by Bill White 2-21-83
Times Juneau Bureau

Juneau — Anti-smokers lined up this week behind a bill that would ban cigarette, pipe, and cigar smoking in public places.

"Your bill can be a terrific benefit for the people of Alaska," said Dr. John Middaugh, state immunologist and official with the Alaska Public Health Association. "It endorses the concepts of health prevention and deterrence."

Middaugh's comments came from Anchorage during a statewide teleconference on the proposal, on which the House Judiciary Committee held two days of hearings.

Susan Scott, a state employee in Anchorage, asked, "Is it unreasonable to request not to have ashes in my cauliflower at the grocery store or to stand in a long line at Long's Drug store with a man puffing on a 6-inch-long cigar or to eat a tasty meal without smoking my neighbor's pipe?"

Current law says smoking in public places "may constitute" a nuisance. The public places listed include trains, buses, ferries, elevators, libraries, museums, lecture or concert halls, gymnasiums, public schools, state meeting rooms, restrooms and hospital hallways. Portions of those areas may be designated as smoking sections.

The proposed bill would declare smoking in those areas a nuisance and a public health hazard.

It also would expand the list of areas in which smoking would be banned. On the list would be taxicabs (unless the driver agrees), all indoor dens of recreation, mu-

See Smoke, page A-4

Smoke

(Continued from page A-1)

nicipal meeting halls, businesses (including retail stores), restaurants, banks, offices, factories and warehouses.

The bill would allow civil lawsuits "to recover for personal injuries sustained as a result of the violation." But committee members discussed deleting that language to prevent harassment suits.

Rep. Hugh Malone, D-Kenai, said one of the biggest problems with the current law is no one enforces it. "I'm inclined to focus on enforcement or forget the bill."

State regulations set fines at $10 to $25 for smokers violating the law and $50 to $100 for those who fail to display no-smoking signs. State Department of Environmental Conservation officials testified they could not recall anyone ever being fined.

Rep. Don Clocksin, D-Anchorage, agreed that enforcement seems to be the problem. Narrowing the law to make sure it is enforced is more important than broadening it if there is no enforcement, he said.

Leo Kaye, executive director of the Alaska Lung Association, said studies have shown second-hand smoke is a health hazard. He added that recent court decisions recognize the rights of employees to work in smoke-free environments.

And employers pay higher health insurance and sick pay costs because of workers who smoke, Kaye said.

Rep. Charlie Bussell, R-Anchorage, pegged the cost per 50 smokers at his electric company at $300,000 a year.

Representatives of the tobacco and retailing industries spoke against the proposal.

Rick Lauber, representing the tobacco industry, argued against testimony that second-hand smoke is harmful. "A vast majority of [illegible] show there's no health hazard from second-hand smoke."

State employees who have complained are covered under the current law, he added.

But one state worker, Roberta Banko, said non-smokers are "treated as troublemakers" if they complain about the "noxious fumes."

"There are a lot of things I can choose to do, but I cannot choose not to work," she said.

Banko, who works for the Department of Labor, said Friday she planned to file a complaint against Rep. Ramona Barnes, R-Anchorage, who smoked in apparent violation of the law, through one of the hearings on the bill.

Barnes said she was in the smoking section of the room. But there are no signs posted in the hearing room.

3-2-83
CB

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March 2, 1983

The Honorable Charlie Bussell
Chairman House Judiciary Committee
State Capitol
Pouch V
Juneau, Alaska 99811

Dear Mr. Chairman:

During the testimony last week on HB 84 there was mention of the White-Froeb Study. The conclusion of that report appeared to conflict with the vast array of eminent scientists who agree that no conclusive scientific evidence exists to support the claim that smoking affects the health of nonsmokers.

I am enclosing a reprint of the Congressional Record, "White-Froeb Study Discredited By Scientists", for your information.

Sincerely,

Rick Lauber

Richard B. Lauber

RBL:ml

United States of America

Congressional Record

PROCEEDINGS AND DEBATES OF THE 97th CONGRESS, SECOND SESSION

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House of Representatives

WHITE-FROEB STUDY DISCREDITED BY SCIENTISTS

HON. L. H. FOUNTAIN
OF NORTH CAROLINA
IN THE HOUSE OF REPRESENTATIVES

Thursday, December 16, 1982

Mr. FOUNTAIN. Mr. Speaker, after 30 years of service to the people of the Second District of North Carolina, I am about to retire from the U.S. House of Representatives. Before leaving I would like to submit, for the RECORD, an item dealing with an issue with which I and many others have long been interested; namely, the alleged effect of smoking on the health of the nonsmoker.

Mr. Speaker, let me briefly place the issue into its proper context. In 1978, the Subcommittee on Tobacco of the House Committee on Agriculture heard testimony from a vast array of eminent scientists and physicians on the issue of the effect of tobacco smoke on nonsmokers. Those individuals who testified generally agreed that no conclusive scientific evidence exists to support the claim that smoking affects the health of nonsmokers. In 1980, however, an article appeared in the New England Journal of Medicine by Drs. White and Feoeb entitled "Small Airways Dysfunction in Nonsmokers Chroncially Exposed to Tobacco Smoke," in which the authors concluded that smoking in the workplace adversely affects the lung function of nonsmokers. This conclusion appeared to conflict with the testimony presented to the Subcommittee on Tobacco.

Since its publication, the White-Froeb study has been used to support both regulatory and legislative activities in the United States. For example, the study was referred to in testimony before the Civil Aeronautics Board during its recent consideration of rules regarding smoking aboard commercial aircraft. The National Research Council report entitled "Indoor Pollutants" which was issued in 1981 under an EPA contract also relies on the study. Finally, the White-Froeb study has received widespread attention in both State and local legislative and policymaking bodies.

The White-Froeb study continues to play an important role in legislative considerations, despite the fact that the study itself has been heavily criticized by scientists and health practitioners. Most recently, at the 1982 joint meeting of the American Lung Association-American Thoracic Society, Dr. Michael D. Lebowitz, professor of internal medicine, college of medicine, University of Arizona and special consultant to the Subcommittee on Tobacco, presented reasons why, in his own words, "the results of this study cannot be used to demonstrate an effect of passive smoking on forced expiratory flows in adults exposed in the workplace." Dr. Lebowitz, a noted specialist in epidemiology and respiratory diseases, said that the basic problem with the White-Froeb study is that it is "improperly designed" and that "there are problems with the whole data set and with the conclusion." Dr. Lebowitz also expressed concern that the significance of the White-Froeb data appeared to depend upon their unexplained omission of data from 3,000 subjects originally included in the study.

Mr. Speaker, Dr. Lebowitz wrote a letter, dated July 10, 1981, to our colleague, Congressman CHARLES ROSE, Chairman of the Tobacco and Peanuts Subcommittee of the House Agriculture Committee, as a result of a personal interview which Chairman ROSE and Dr. Lebowitz had with Dr. White. With the personal consent of Chairman ROSE, I am inserting herewith Dr. Lebowitz's letter. It more fully explains the author's views regarding the White-Froeb study.

I also want to mention another evaluation of the White-Froeb study, one which was made by Dr. J. G. Gostomzyk, director of the department of health of the city of Augsburg, West Germany. After an extensive, detailed review of the White-Froeb study, Dr. Gostomzyk has concluded that the White-Froeb data were incompletely presented and did not satisfy the prerequisites for scientific credibility. In addition, Dr. Gostomzyk remarked that "Dr. White's methodology is not scientific but that of a lay person with convictions," and concluded that "we assume that Dr. White's study is an attempt at scientific validation of his credo and that he possibly is unaware of the inadequacy of this methodology." It is obvious that Dr. Gostomzyk is referring to Dr. White's outspoken antismoking activities in California, including Dr. White's endorsement of public smoking referendums which were, incidentally, twice rejected by the California voters.

Given these and other criticisms of the White-Froeb study, it would appear that the New England Journal of Medicine has, perhaps unwittingly, performed a disservice to its readership. It is extremely unfortunate that a study so fraught with methodological problems, as indicated through numerous criticisms by scientists in the United States and elsewhere, should have been published in such a reputable journal of medicine. The White-Froeb study should, therefore, not be relied upon by the Congress, Federal agencies, or other legislative or policymaking bodies when considering restrictions on smoking in public places.

THE UNIVERSITY OF ARIZONA,
COLLEGE OF MEDICINE,
Tucson, Ariz., July 10, 1981

Congressman CHARLES ROSE,
Chairman, Subcommittee on Tobacco and Peanuts, House of Representatives, Rayburn Building, Washington, D.C.

DEAR CONGRESSMAN ROSE: The following is a summary of my notes on our visit to Dr. James White at UC San Diego, as per our discussion. Unfortunately, despite the statement in the editorial of the New England Journal of Medicine (27 March 1980), Dr. White and his co-author did not "faultlessly demonstrate a reduction in measures of small airways of healthy non-smokers exposed to cigarette smoke in the work place". It is apparent from our visit and the article that there were various faults in the present study, which shall be discussed.

The problems with the research design are as follows:

The participants were not only volunteers, but generally had to pay for the physical fitness course; this is the reason most were white-collar. Employees in specific factories invited White to run the physical fitness course in their factories as well, which would also bias the population sample. Blue-collar workers were not distributed randomly. [It has to be assumed that volunteers in the physical fitness courses fall into unrepresentative categories: the highly motivated, with an interest in health and usually healthier, those who are worried about health and generally less healthy; the first group would include fewer smokers and the second group would include more smokers.]

The questionnaire utilized was not a vali-

dated one per se; test-retest comparisons were made only on the smoking questions and very small groups of subjects. The smoking information was not validated. There were no test-retest or validations on symptoms asked in the questionnaire. The questionnaire itself was derived by the investigator, and included some questions from standard questionnaires; this did not appear to include standard respiratory questions, and in fact various typical respiratory questions (such as phlegm) were not asked. The questionnaire did not include questions on attitude, but did include questions on activity levels and jobs (duration, type). The questionnaire did ask how many smokers were in their work area, room size, and nature of the air conditioning. It also included questions about residences in the last 20 years (zip codes), so that exposures away from work were assessed by residential location. A question was asked about smokers in the home. [Thus, the smoking information is not validated, but is probably relatively accurate. The information about exposure to passive smoking is only approximate, as is the information on other occupational exposures. Exposures to air pollutants or to unknown toxic gases in the working place is only approximate, and their effects underestimated.]

Dr. White presented a paper to the American College of Sports Medicine, the abstract for which in 1977 indicated there were 7,122 subjects enrolled between 1969 and 1977. However, in the New England Journal of Medicine article, he states that the base population analyzed is only 5,210 smokers and non-smokers enrolled between 1969 and 1979. Although he excluded all the ex-smokers, some whose zip codes were missing, his answers as to why the rest of the subjects were excluded were entirely unclear and tend to indicate potential bias in selection of subjects for consideration for analyses. It might be added that the 2,100 subjects analyzed in the NEJM article and those analyzed and presented in the Sports Medicine abstract appear to be the same as they yield exactly the same table of results (as determined from comparison of the table in the Sports Medicine manuscript and the NEJM table).

In addition to the sources of bias mentioned above, it is apparent that the non-smokers in clean work environments and those in smoking work environments have not only chosen not to smoke, but it is likely that those non-smokers working in smoking environments may be different for a variety of reasons from non-smokers working in clean environments. Furthermore, it is apparent that the non-smokers in non-smoking environments are quite different in that their lung function is "super normal" in comparison even with the Seventh Day Adventists (the source of the Morris prediction equations).

Dr. White did state that from the questionnaire and from the baseline tests that there were no significant differences in the three non-smoking/non-inhaling groups in terms of the amount of previous exercise or oxygen consumption, but he was unsure of the difference in percent of body fat. Smokers did have less body fat, were less in terms of having lower oxygen consumption, and had less activity. He says further that there were no differences between the groups in terms of childhood respiratory history (lower respiratory tract illnesses) from his submitted questionnaire information, but he did not ask about family history. He did not ask sufficiently about respiratory questionnaires to appropriately exclude groups on the bases of productive cough ("cough bronchitis"). He states that there were no differences in prevalence rates of questionnaire responses by zip codes; if so, this contradicts other evidence vis-a-vis the effects of air pollution in these areas. He was not able to assess other exposures such as those from hobbies, exposures to gas stoves, or transportation. In terms of passive smoking in the home, he excluded such passive smokers from the non-smoking and passive smoking groups, but not from any smoking groups. He was not able to provide any information about the distribution of characteristics in those eliminated from the original 7,000 or the 2,208 that qualified because of other questionnaire results.

With regards to the pulmonary function testing done by Dr. White, it must be first noted that the instrument used is not considered a satisfactory instrument in that it is non-linear (highly biased) at both high volumes and low volumes. [This has the effect of maximizing differences in that anyone with minor aberrations of total vital capacity or of flows at the end of the flow volume curve would have very different, that is, low, flows.] The comparisons that Dr. White did and reported on in his response letter in the NEJM (14 August 1980) would not in any way modify this opinion. Furthermore, Dr. White has the only pulmonary function technician and reader. Even though he was trained at the VA hospital and his techniques were evaluated by test—retest and by comparison to other readers, any biases inherent in Dr. White's thinking (see below) would affect the way he read the tests. Furthermore, he took the FEV$_1$ and flows off the same spirogram using an approximation technique published by Morris, et al., which is not an adequate or accurate representation of those measures. All of his tests were baseline tests done after two and a half hours in the classroom in the evening on those without acute respiratory illnesses (usually on a Monday or Tuesday evening); thus, there is probably little diurnal variation or pretest biases other than those experienced by the workers during their work day and in their activities prior to the classroom. Although it is difficult to judge the effects of these factors, they may have influenced the test results, especially in those with any significant exposures during the day.

The major problem with the pulmonary function test results as reported is that they are not age- and height-adjusted, since lung volumes and flow rates are associated with both of these factors. In other words, Dr. White used raw values of flows and volumes to do comparisons. He did this on the assumption that the mean age and height were similar for the different groups. This is a mistake, since the distributions for those ages and heights could have differed. Furthermore, his quoted figures for percent predicted are strictly for the average person, age 49, with an average height, and does not represent the group for which they are provided. In terms of these statistical analysis, he just chose the SNK package among many. There is no correlation coefficient per se. "Normality" was not an objective of this study, so he cannot state anything about the normality of the subjects studied, including those he considered to have significantly different results from the non-exposed non-smokers. He does not understand the difference between clinical meaningfulness and statistical significance. It is quite obvious that the majority of those in the passive smoking and in the non-inhaling group are quite normal and that very few would be consdered abnormal by any criteria.

In his reported results, he quotes as incorrect significance level of $p.<.005$, whereas the level provided by the technique is $p.<.05$. This is very different, given the number of comparisons made, and indicates that some of the results would not be significant if corrections were made for the number of comparisons. Furthermore, the data presented in Table 1 was used to recompute the SNK analysis by Mary C. Townsend, MPH (Department of Epidemiology, University of Pittsburgh). Those results differ from those published by Dr. White and are provided in the attachment. The most important of the differences is the finding that the passive smokers and light smokers differ for the male FEV 75-85 percent. Thus, the effect of passive smoking on non-smokers is still unconfirmed, despite Dr. White's unfailing conviction that it is confirmed.

Other minor points: In terms of the carbon monoxide sampling, although it is stated that it was randomized, it was really on only 40 smoking and 40 non-smoking situations chosen by chance but not by random selection. Dr. Froeb, the co-author with Dr. White, is a private practitioner in La Jolla and helped Dr. White in drafting the NEJM manuscript from the manuscript presented at the American College of Sport Medicine. It might be pointed out that San Diego is not strictly low in air pollution concentrations, nor uniformed throughout the area; this may bias some results. Dr. White performed the pulmonary function tests until "reproducible curves were obtained", but they do not necessarily follow the Intermountain, Snowbird, or ATS recommendations.

In reviewing Dr. White's response to the letter to the Editor in the NEJM (14 August 1980), it is quite clear that Dr. White did not satisfactorily answer all the questions raised, many of which are similar to those raised in this letter. It is questionable, from the discussion, whether Dr. White would pursue any further re-analysis of the data, nor necessarily could it be pursued. It is questionable, given the basic underlying problems in the research design, that re-analysis of the data would be worthwhile. On the other hand, given other results that contradict Dr. White's, including those now in press (such as Comstock et al., Johns Hopkins, presented at the Society for Epidemiological Research in June of 1981), it would be likely that a panel discussion of passive smoking might be valuable. I will be glad to furnish further discussion or help in that matter.

Sincerely,

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development programs are under way through sponsorship of the U.S. Department of Agriculture, National Science Foundation, Southwest Border Regional Commission, Four-Corners Regional Commission, and the state of California.

Germ plasm collections have been made from wild guayule plants in Mexico and Texas. Plantings have been established to test yields, to increase seed supplies, and to conduct plant breeding work. Test plots have been established to determine desirable planting and cultivation practices. Research is being conducted on the possibility of increasing rubber yield by treating guayule plants with plant growth regulators.

The recent development of a seed coating process to promote germination, and the development of selective herbicides, will make direct seeding in field plantations a possibility. Eliminating nursery or greenhouse propagation could produce considerable savings in production costs.

The only guayule yield figures now available are estimates developed during the ERP. During the life of the ERP the [illegible] per hectare, approximately 480 kg of guayule rubber per year. Kelly (*15*) obtained yields of approximately 860 kg per hectare per year from one test plot in California. Foster *et al.* (*16*) have outlined the state of the art of guayule technology and described present and projected world rubber market conditions and areas of the United States where conditions favor guayule cultivation.

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17. The Diamond Shamrock Corporation, Cleveland, Ohio, is sponsoring the hydrocarbon research program at the University of Arizona.

M. Dunn

Indoor Air Pollution, Tobacco Smoke, and Public Health

James L. Repace and Alfred H. Lowrey

Serious health effects from air pollution have led to federal standards for the regulation of outdoor exposure levels. However, Americans spend about 90 percent of their time indoors (*1*). Thus the levels of indoor air pollution are important in determining total exposure to air pollutants (*2–6*). Indeed, in a recent review article (*4*) it was concluded that indoor air pollution in public office buildings is of greater potential harm than the outdoor variety, and that these exposures may constitute a real threat to the health of many urban people. The U.S. Surgeon General asserted in his report on *Smoking and Health* that tobacco smoke can be a significant source of atmospheric pollution in enclosed areas (*7*). Some 53 million U.S. smokers consumed 615 billion cigarettes in 1978 (*8*). Thus it is apparent that indoor air pollution from tobacco smoke is pandemic.

In the presence of cigarette smoke, many normal nonsmokers experience eye and throat irritation, headache, rhinitis, and coughing; allergic persons report wheezing, sneezing, and nausea as well. Particularly acute symptoms may be found in infants, children, persons with cardiovascular or respiratory disease, and wearers of contact lenses (*7, 9*). Determining the extent of the exposure of nonsmokers to cigarette smoke is important because smoking is a cause of chronic obstructive pulmonary disease, cardiovascular disease, and lung cancer, and is associated with cancers in other parts of the body (*7*); because these diseases also occur in nonsmokers; and because the products of tobacco combustion have been detected in nonsmokers (*10*).

Although measurements of indoor carbon monoxide pollution from smoking are abundant (*7*), published reports of the exposure of the population to the particulate phase of ambient tobacco smoke are rare (*7, 11–13*). Furthermore, a comprehensive theory of the generation and removal mechanisms for tobacco particulates in naturally or mechanically ventilated habitable spaces has not been presented.

We therefore undertook a systematic study of the levels of respirable suspended particulates (RSP) in several common indoor environments in an attempt (i) to determine the relation of these levels to the aerosol from tobacco smoking, (ii) to understand the effect of ventilation on tobacco smoke concentra-

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tions, and (iii) to develop a general model for estimating the range of the public's exposure. Our goal was to provide a quantitative basis for assessing the health hazards to nonsmokers posed by repeated exposure to tobacco combustion products.

Model Development

To relate the contribution of smoking to indoor RSP requires a model describing the behavior of the tobacco aerosol in indoor spaces. Bridge and Corn (*6*) found that a reduced form of an equation by Turk (*14*) reliably predicts carbon monoxide (CO) concentrations from tobacco smoke in ventilated spaces and so is of major value in assessing the possible hazards in occupied spaces (*11*). The equation is not valid, however, for a pollutant that is affected by physical decay due to adsorption on room surfaces. Penkala and DeOliviera (*15*) showed that decay of the tobacco aerosol in a well-mixed unventilated chamber is exponential.

We modify the Turk equation in differential form by adding a decay term to the removal rate and equating the rate of change of pollutant mass to the algebraic sum of the generation and removal rates.

Table 1. Recommended values of the mixing factor m. After Corn (*11*).

Configuration of air supply system	m
Perforated ceiling*	1/2
Trunk system with anemostats	1/3
Trunk system with diffusers	1/4
Natural draft and ceiling exhaust fans	1/6
Infiltration and natural draft	1/10

*This is the best standard condition.

The mixing factor is an empirical number that accounts for room-specific effects on pollutant transport. Pollutant removal is more rapid in a well-mixed atmosphere (where m is large) than in a poorly mixed, stable one (where m is small). Factors that affect m include type and placement of ventilation grills, ventilation flow rates, inhomogeneous pollutant distribution, barriers, circulation fans, and room traffic.

decay time, a time constant associated with the removal of a pollutant from a room through adsorption on surfaces and filtration; and m is the mixing factor, an empirically determined number (*16*) that modifies the ventilation time as τ_v/m, where $m \leq 1$ ($m = 1$ implies ideal mixing). Corn (*11*) suggested values of m for various ventilation systems (Table 1). We postulate that m also modifies the ideal decay time as τ_a/m. The pollution generation rate, in micrograms per minute, is given as $G = nC_0/t_b$, where n is the number of cigarettes being smoked at time t; C_0 is the total particulate matter (TPM) from both sidestream and exhaled mainstream smoke; and t_b is the duration of cigarette smoking.

Summary. An experimental and theoretical investigation is made into the range and nature of the exposure of the nonsmoking public to respirable suspended particulates from cigarette smoke. A model incorporating both physical and sociological parameters is shown to be useful in understanding particulate levels from cigarette smoke in indoor environments. Observed levels of particulates correlate with the predictions of the model. It is shown that nonsmokers are exposed to significant air pollution burdens from indoor smoking. An assessment of the public health policy implications of these burdens is presented.

The solution yields the density $A(t)$, in micrograms per cubic meter, of smoke in the room as a function of time:

$$A(t) = A_{eq}(1 - e^{-t/\tau}) \qquad (1)$$

where $A_{eq} = G\tau/V$ is the equilibrium concentration of the pollutant in the room, and where the time constant

$$\tau = \frac{\tau_a\tau_v}{m(\tau_a + \tau_v)} \qquad (2)$$

is the mean ventilation time, or the time for the smoke concentration to decrease to $1/e$ of its value (where e is the base of natural logarithms); V is the room volume in cubic meters; $\tau_v = V/Q$ is the ideal ventilation time, or the time required to replace a volume of air equal to the volume of the room by ventilation and infiltration; Q is the volume rate of ventilation and infiltration; τ_a is the ideal

If $\tau = \tau_v/m$) it becomes the reduced Turk equation of Bridge and Corn (*6*), with $m = 1$; and (ii) in the case of adsorption only (the unventilated room), $\tau = \tau_a/m$. Then, if the generation of smoke ceases at time t_b, prior to equilibrium, A will decay according to

$$A(t) = A_0 e^{-m(t-t_b)/\tau_a} \qquad (3)$$

where A_0 is a constant related to the equilibrium concentration by

$$A_0 = A_{eq}[e^{m t_b/\tau_a} - 1]$$

Equation 3 becomes the decay equation described by Penkala and DeOliviera ($t > t_b$, $m = 1$).

The modified Turk equation (Eq. 1) contains only measurable quantities, and thus in principle can be used to estimate the concentration of TPM or CO from tobacco smoke (or other indoor air pollutants), as a function of time, for any room for which the pollutant generation rate, volume, and mean ventilation time are known.

Controlled Experiments

Equation 2 shows that the mixing factor affects the time constant for decay as well as ventilation. Experiments under conditions of known ventilation were therefore necessary to assess the influence of mixing factors, decay time constants, and generation rates on the growth and decay of tobacco smoke particulates. To increase the usefulness of the experimental values determined for the mean ventilation time for the removal of tobacco smoke, we conducted these experiments in actual occupied spaces rather than in experimental chambers.

A piezobalance (TSI model 3500) (*17–19*) was used in sampling the aerosol. It collects respirable particulates (*20*) between 0.01 and 3.0 micrometers in diameter with near 100 percent efficiency (decreasing to 50 percent at 3.5 μm and to 10 percent at 4 μm). The sampling rate is 1 liter/min (*18*); the sampling time is variable. Factory-calibrated with welding smoke, the detecting crystal in the instrument used has a sensitivity of 5.74 $\mu g/min\text{-}m^3$ per hertz. The instrument underestimates the mass concentration of tobacco aerosol by about 15 percent compared to measurements made with low-volume filter sampling techniques. Readings can be affected by changes in humidity; the maximum expected error due to changes in relative humidity when sampling a hygroscopic aerosol (such as tobacco smoke) is given as $\pm$ 10 $\mu g/m^3$. The overall instrument error is about $\pm$ 10 percent compared with low-volume filter measurements of welding smoke (*19*). The aerosol from sidestream cigarette smoke (that portion emitted by the burning tip), an important component of many indoor aerosols, is lognormal, with 99 percent of the mass < 1 μm in aerodynamic diameter and with an initial mass median diameter (MMD) from 0.2 to 1.5 μm depending on dilution (*20, 21*). The relative particle sizes of fresh sidestream and mainstream smoke (the latter being that portion inhaled by the smoker) are about the same; for ex-

Table 2. Parameters for Eq. 1, as determined with experiments 1 to 3 (unventilated rooms).

Experiment	A_{eq} ($\mu g/m^3$)	A_u ($\mu g/m^3$)	τ_a/m (min)	m	r^{2*}	C_0† (mg of TPM)	Cigarette condition
1‡	530	503	10	1	.98	12.3	Smoldered
2‡	5178	551	89	1/9	.42	16.0	Smoldered
3§	1773	681	16.4	< 1	.81	23.0	Smoked

*Coefficient of determination for the decay curve. †The estimated amount of TPM liberated if the entire cigarette had been consumed, according to FTC protocol. The FTC mainstream TPM level for this cigarette is 18 mg (*24*). ‡V = 21.9 m³. §V = 29 m³.

haled mainstream smoke, particle size is estimated to be ~ 0.8 μm (MMD). Since the ambient cigarette smoke aerosol is reproducible and coagulates very slowly, it has been used as a test aerosol (*21*) and in evaluation of heating, air-conditioning, and ventilating systems (*22*). [The bulk of the ambient tobacco aerosol is probably due to cigarettes, since less than 15 percent of smokers smoke cigars or pipes (*23*).]

Unventilated Growth and Decay of Tobacco Smoke

Experiments were carried out to determine the usefulness of Eq. 3, which predicts a rapid decay for good mixing and a slow decay for poor mixing; and also to discover the limits of τ_a/m.

Experiments 1 and 2 were conducted in a wood-panelled den in a private residence. In the geometric center of the room (volume, 21.9 m³), a popular filter cigarette [containing 65 millimeters of tobacco and ranking 94th on the Federal Trade Commission (FTC) scale of tar and nicotine content (17 milligrams of tar and 1 milligram of nicotine) (*24*)] was ignited and allowed to smolder until 89 percent of its tobacco was consumed. During the first experiment, two box fans (51 centimeters in diameter) with antiparallel exhausts were used to ensure ideal mixing; each fan's exhaust, measured with a Velometer, was 55 m³/min. The growth and decay of the RSP were measured with the piezobalance. Experiment 2 was similar to experiment 1, except that the cigarette was extinguished after 75 percent of its tobacco was consumed and the circulating fans were not used, so that mixing was natural. The results of both experiments are plotted in Fig. 1, with the background levels of RSP subtracted. The data points generally represent 1-minute average values. The differences in mixing dramatically affect the slopes of the decay curves.

The theoretical curves shown in Fig. 1 were generated by fitting the data points from the decay curves to Eq. 3 with a regression analysis; A_0 and τ_a/m were determined and used to calculate the growth curves from Eq. 1, case (ii). The ratio of the slopes of the decay curves for ideal and natural mixing yields the mixing factor for the room. Table 2 gives the values obtained for the various parameters. The value of the mixing factor obtained is in good agreement with the expected value given in Table 1 for the case of infiltration and natural draft. The growth curve for the case of natural mixing (experiment 2) shows a poor fit initially because of the effect of the warm smoke rising to the ceiling and remaining

Table 3. Field survey of indoor RSP in the absence of smoking.

Locale	Room volume (m³)	Persons per room	Persons per 100 m³	Indoor RSP level* ($\mu g/m^3$)	Average time per RSP sample (min)	Outdoor RSP level† ($\mu g/m^3$)	Comment
Crepes restaurant (Washington, D.C.)	124	43	35	29	20	44	No smoking section; aroma of frying crepes evident
Sandwich restaurant (Laurel, Md.)	326	40	12	55	21	40	No smoking section; near kitchen; three smokers in smoking section
Sandwich restaurant (Laurel, Md.)	326	55	17	51	21	55	No smoking section; near kitchen; one smoker in smoking section
Fast-food restaurant (Bowie, Md.)	1,400	22	1.6	38	7		Aroma of hamburgers frying
Private residence (Seabrook, Md.)	120	11	9	24	20		Cocktail party; one candle burning 6 m from RSP detector
Private residence (Bowie, Md.)	124	1	0.8	44	15		One hour after sweeping basement floor
Private residence (Greenbelt, Md.)	22	2	9	24	6		Natural mixing‡
	22	2	9	55	1		Two fans moving 110 m³ of air per minute§
Private residence (Glenn Dale, Md.)	29	7	24	57	5		One fan moving 55 m³ of air per minute‖
Conference room (Greenbelt, Md.)	113	10	9	53	10		Two fans moving 50 m³ of air per minute¶
Public library meeting room (Bowie, Md.)	1,415	30	2.1	29	30		During piano recital
Library of Congress (Washington, D.C.)	27,000	130	0.48	30	10		Main reading room
Church (Bowie, Md.)	4,224	300	7	30	42		During Sunday service
Bagel bakery (Yonkers, N.Y.)	510	30	6	25	10	8	Aroma of baking bagels evident
Private residence (Hawthorne, N.Y.)	150	17	11	26	16		During dinner party

*Mean ± standard deviation for the Washington area samples, 40 ± 13 $\mu g/m^3$. †Duration of sampling, 5 minutes. ‡Experiment 2 background. §Experiment 1 background. ‖Experiment 3 background. ¶Experiment 4 background.

out of the detector's range for about 3 minutes. Experiment 3 demonstrated that Eq. 3 is valid under more general conditions, that is, when a cigarette is actually smoked.

We conclude that these experiments show that for the unventilated room, Eq. 3, the reduced form of Eq. 1, is useful in describing the growth and decay of cigarette smoke particulates.

Ventilated Growth and Equilibrium of Tobacco Smoke

An experiment was conducted in a ventilated conference room of a modern office building to test Eq. 1 in the case of removal of uniformly generated tobacco smoke by both decay and ventilation. The experiment involved measuring the growth of cigarette-generated RSP from background levels to near equilibrium. Analysis of the RSP-versus-time curve determines τ, the mean ventilation time, and C_T, the total RSP liberated from the combined sidestream and exhaled mainstream smoke.

The RSP detector was located in the geometric center of the 113-m³ room. Two fans with antiparallel exhausts were used to establish a vigorous circulation of 100 m³/min. The ideal ventilation time τ_a, calculated from the volumetric flow rates of the ventilation system, was 49.2 minutes for a complete change of air. Thirty-two cigarettes were smoked in 40 minutes by a relay of seven smokers, with an average of four persons smoking at any given time. When smoking their own brands, they averaged 9.8 minutes per cigarette; when smoking cigarettes supplied to them, they averaged 5.8 minutes per cigarette. All butts were collected and the amount of tobacco consumed was measured for each cigarette. The estimated mainstream TPM (M) (tar plus nicotine) generated by the 32 cigarettes was determined by weighting the TPM values for each cigarette (*24*) by the fraction of tobacco consumed, and adding the results to obtain M = 418 mg [TPM is emitted from cigarettes at a linear rate after the fourth puff (*25*)].

Figure 2 shows the growth against time of RSP from the cigarette smoke. The data points are corrected for background RSP levels and are 2-minute averages. A regression analysis using Eq. 1 yields A_{eq} = 1947 μg/m³ and τ = 14 minutes, with a coefficient of determination = .964 (from Eq. 2, τ_a = 19.5 minutes). Finally, C_T, or the total amount of RSP liberated in the room during the entire smoking period, 772 mg, is calculated by using Eq. 1; C_T/M = 1.85. This ratio represents a weighted average for six different brands of filter cigarettes that together commanded a 23 percent share of the market in 1976 (*26*).

From the goodness of fit of the theory to the data and from the observation of predicted interactive behavior among mixing, growth, and decay processes for RSP from cigarette smoke, it appears that all the room-specific factors affecting the removal of tobacco smoke (ventilation, decay, and mixing) can be combined into a single time constant τ, which can be determined for any room by regression analysis of the decay or growth-equilibrium curves, or by calculation from the equilibrium concentration if the smoke generation rate and room volume are known. The ratio of the slopes of the decay curves for ideal and natural mixing yields the mixing factor. We conclude that Eq. 1 is a useful tool for predicting the levels of tobacco smoke in both ventilated and unventilated occupied space.

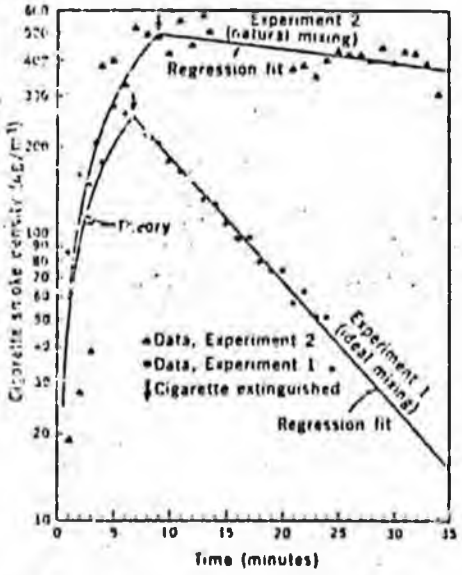

Fig. 1. Theoretical predictions versus experimental results for the growth and decay of RSP from a smoldering, average-tar cigarette in a 22-m³ unventilated room. The dramatic difference in the slopes of the decay curves reflects the difference in room air turbulence (mixing) for the otherwise similar experiments.

Field Survey of RSP

We now address the complex problem of surveying the levels of RSP indoors and determining what portion of this aerosol may be attributed to cigarette smoke by means of Eq. 1. The problem is complicated by differences in smoking rates, numbers of smokers, room volumes, effective ventilation rates, and the TPM values of various brands of cigarettes. The problem may be simplified by assuming that smoking is a random process when it occurs among large groups of people. It follows that cigarette smoke RSP values may be treated as equilibrium values; that all of the smokers may be treated as habitual smokers who smoke identical average-tar cigarettes in the same way at the same average rate, uniformly distributed over a 16-hour day. An average smoking rate r of two cigarettes per hour is calculated from the 1975 figures for the number of U.S. smokers and the U.S. domestic cigarette consumption (*8*). In 1978, the sales-weighted average mainstream TPM value M_a was 17.6 mg for all the cigarettes sold in the United States (*7*). The estimated emission rate C_0 (combined sidestream plus exhaled mainstream TPM) from a habitual smoker is given by $E = 1.85\,rM_a$ = 65 mg/hour, where 1.85, used for the ratio C_0/M_a, is taken from the conference room experiment. Physically observable in any field survey of smoking is n_s, the number of burning cigarettes (the number of "active" smokers); n_s can be related to the number of habitual smokers n_{hs} by considering that the average time for smoking a cigarette is 10 minutes (*2, 6*). This number and the previously calculated average smoking rate indicate that $n_{hs} = 3n_s$.

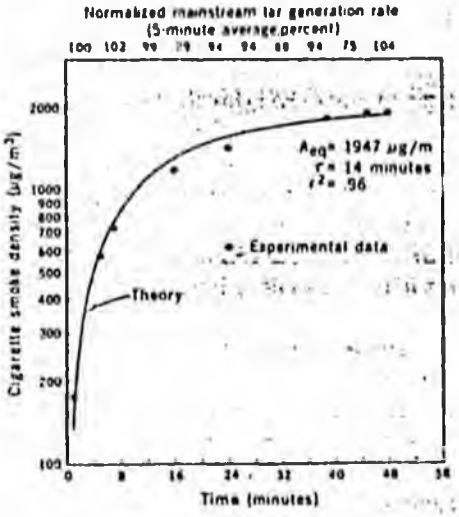

Fig. 2. Theoretical predictions versus experimental results for the attainment of equilibrium A_{eq} for the combined emission of sidestream and exhaled mainstream cigarette smoke from four chain smokers in a 113-m³ conference room with well-mixed ($n \sim 1$) ventilation in a modern office building. Under natural mixing conditions, about 11 habitual smokers would generate an equivalent equilibrated concentration of smoke. This many smokers would be expected in a group of 33 adults (well within the capacity of this 50-person conference room).

From the equilibrated form of E[illegible], we determine that

$$R = A_{eq} = 650 \frac{D_s}{C_a} \quad (4)$$

where R is the smoker-generated equilibrium RSP level in micrograms per cubic meter, D_s is the density of active smokers (number per 100 m³), and C_a is the effective rate for the removal of cigarette aerosol (air changes per hour), with $C_a = 60/\tau$.

The aerosol sampling described in this article was performed from late March through early June 1978 in the Washington, D.C., metropolitan area. The MMD (seasonal average) of the outdoor urban aerosol for Washington in 1970 was 0.5 μm, with 90 percent of the aerosol mass less than 3 μm in aerodynamic diameter and lognormally distributed (*27*).

It is important to note that all of the RSP measurements we report represent time-averaged values.

Factors other than tobacco smoke may contribute to indoor RSP. These include infiltration of outdoor RSP, cooking, dust raised by indoor traffic, and industry. By restricting the sampling to nonindustrial indoor locations where tobacco smoking is absent, the effect of the remaining variables may be assessed. Table 3 gives the RSP levels for several indoor spaces in which smoking did not take place: three restaurants, four private residences, an office building conference room, two libraries, and a church during services. The mean of these measurements is 40 μg/m³. In three instances, fans were mixing the air at a high rate and RSP levels were elevated, apparently because of dust entrainment. No correlation between the volumetric density of people (occupancy) and RSP is evident. Hemperly (*28*), in sampling RSP in Houston, found similar RSP levels in two schools, a library, and a museum—all nonsmoking areas.

Table 4 gives the results of RSP sampling in nonsmokers' automobiles traveling along two major commuter highways (Route 50 in Maryland and U.S. I-295 in Washington) during the rush hour. The samples were taken on different days and were measured in different vehicles. In all cases, the windows were slightly open and the ventilation fans were running. The mean of the data, 38 μg/m³, is not very different from the mean of the indoor readings, 40 μg/m³ (Table 3).

The impact of actual ventilation practice on ambient RSP levels from smoking was investigated at eight restaurants, three cocktail lounges, two bingo games, a dinner-dance, a bowling alley, a sports arena, and a hospital emergency waiting room. For contrast, one unventilated private residence was sampled during a cocktail party. With the exception of the hospital waiting room and the hotel bar, each space sampled represented the major part of the building and was subject to ventilation requirements specified by building codes. Sampling was generally performed well after opening time to ensure that an approximately steady-state level of smoking had been reached.

Table 4. Levels of RSP in nonsmokers' [illegible] during rush-hour traffic on a busy commuter highway in Washington, D.C., measured with the vehicles' windows slightly open and the ventilation fans running. Each car carried four persons and had a volume of 2 m³, so that the occupancy was equal to 200 persons per 100 m³.

Date	Time	Sampling time (min)	RSP level (μg/m³)
23 March	a.m.	10	40
23 March	p.m.	35	20
24 March	a.m.	20	54
28 March	a.m.	26	49
31 March	a.m.	8	25
Mean ± standard error			38 ± 15

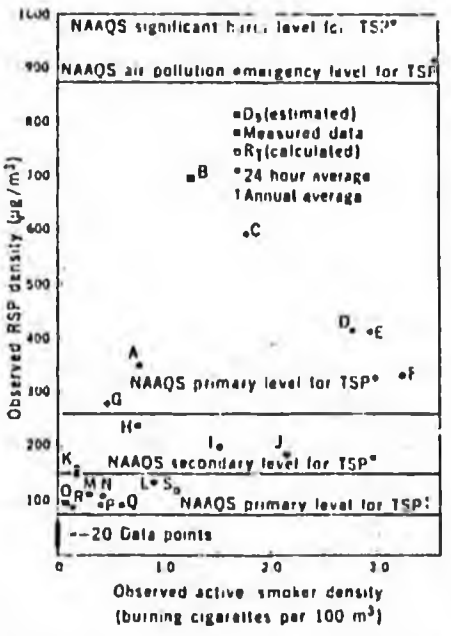

Fig. 3. Results of a field survey of short-term time-averaged levels of RSP in 38 enclosed spaces (see Tables 3, 4 and 5). Levels corresponding to federal standards for TSP are indicated for comparison only. The microenvironments include ten restaurants, three cocktail lounges, two bingo games, a dinner-dance hall, a bowling alley, a sports arena, two libraries, a church, a hospital waiting room, five vehicles, and five residences. The letters A through S correspond to those given in Table 5. The effective air change rates for microenvironments A and S are known from experiments to be C_a = 1.5 and 9.2, respectively.

The piezobalance and a stopwatch were used to take tabletop-level RSP samples for periods ranging from 10 to 50 minutes (mean, 20 minutes). The piezobalance was equilibrated in advance to avoid errors due to changes in temperature or humidity.

The room dimensions were estimated and the number of active smokers was sampled periodically throughout the measurement period and averaged. It was usually not possible to sample the premises when there was no smoking; in most cases, the RSP outside the premises was sampled for comparison. Table 5 gives the results of the measurements. Figure 3 shows the average density of active smokers (defined as the number of burning cigarettes per 100 m³) plotted against the total indoor RSP sampled. As a guide to whether a given datum is "high" or "low," the National Ambient Air Quality Standards (NAAQS) for total suspended particulates (TSP) are also shown. Since a specific averaging time is incorporated into these standards, violation of the standard is not demonstrated here. However, repeated exposure to such elevated levels can lead to "violations" of the annual standards, as will be shown later. Note that all the data for finite smoker density exceeded the level of the annual primary (health-related) NAAQS, whereas none of the data for zero smoker density exceeded this level. Further, the background RSP measured outside the smoking premises suggests that the source of these elevated levels was not the outdoor air. The mean and the standard deviation for the outdoor RSP are 46 ± 13 μg/m³, and in every case the outdoor level is less than the indoor. In certain cases, indoor controls are available. In bingo game 2, held in the nave of a church, the active smoker density was 0.47 per 100 m³, the occupancy was 3.6 persons per 100 m³, and the RSP density was 279 μg/m³ (Table 5). By contrast, during the tobacco smoke-free religious services, despite an occupancy of 7.0 persons per 100 m³, 30 burning votive candles, and several processions, the RSP density was 30 μg/m³. The elevated RSP levels in the bingo game clearly appear to be due to smoking. Similarly, measurements taken in the nonsmoking section of a sandwich restaurant showed considerably lower levels than in the smoking section, indicating that the contribution of smoking to RSP was much larger than the effect of cooking, even at the low cigarette densities shown (Table 5). Figure 3 shows that, in general, RSP levels increase with active smoker density, although there is

considerable scatter in the data. The question now is whether Eq. 1 is useful in explaining this scatter.

We hypothesize that the levels of RSP for finite D_s (Fig. 3) are due to near-equilibrium levels of cigarette smoke adding to much smaller background levels, and that the scatter in the RSP levels for fixed cigarette density is due primarily to differences in the mean ventilation time τ. Analyzing the background corrected data given in Table 5, we use Eq. 4 to calculate a range for C_a between 1.2 and 10.7 air changes per hour; C_a is used instead of τ to facilitate comparison with building code-specified ventilation rates. The range determined for C_a is consistent with two known values of C_a derived from the cocktail party and roadside restaurant experiments (Table 5).

The C_a for tobacco aerosol is affected by the rate of mechanical ventilation and infiltration, the rate of smoke adsorption, and mixing. The range of mechanical ventilation and infiltration can be calculated from tables of standards determined by the American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) (29), the authority specified by the local building code (30). For each premise listed in Table 5, the recommended maximum number of outdoor air changes per hour (based on the estimated floor area, maximum occupancy, and volume) was calculated from the ASHRAE tables; a two-thirds recirculation of air (the maximum permitted by ASHRAE) was assumed. This yielded a range of 0.7 to 9.4 air changes per hour. Infiltration, resulting mainly from the opening of doors, was estimated from the actual occupancy during the sampling (29): we assumed a 100 percent turnover of occupants per hour. This was added to the calculated mechanical ventilation rates, giving a final estimated range of $1.3 \leq C_v \leq 13.4$ air changes per hour, where $C_v = 60/\tau_v$.

The practical range of physical decay from adsorption for cigarette aerosol can be computed from our experiments and the literature. Most establishments possess simple filters that are relatively ineffective at removing tobacco smoke (22). The shortest ideal decay time measured (in experiment 1) was equivalent to six air changes per hour (Table 2). By contrast, Penkala and DeOliviera (15) measured a mean life for tobacco smoke, under uniform mixing in a chamber with unreactive walls, equivalent to one air change per hour. These two extremes given an estimated range of $1 \leq C_d \leq 6$ air changes per hour for RSP from tobacco smoke, where $C_d = 60/\tau_d$.

The range of mixing m appropriate for the spaces listed in Table 5 is $1/4 \leq m \leq 1/2$, as determined from Table 1. By using Eq. 2, a theoretical range of mean air change rates, $1/2 \leq C_{aTh} \leq 10$ air changes per hour, is calculated from the estimated ranges for C_v, C_d, and m. This is consistent with the 1 to 11 air changes per hour determined with our model from the experimental results. In other words, the variations in the observed RSP density for fixed cigarette density can be phenomenologically ac-

Table 5. Field survey of indoor RSP sampled in the presence of smoking. Where the standard deviation is given, the value is an average of 2-minute samples; where it is not given, the sampling time is the averaging time.

Locale	Estimated volume (m^3)	Average number of smokers	Indoor sampling time (min)	Average occupancy (persons)	Active smoker density per 100 m^3	Indoor RSP ($\mu g/m^3$)	Outdoor RSP ($\mu g/m^3$)	Outdoor sampling time (min)	Occupants smoking (%)	Date	Time
A. Cocktail party*	268	2	15	14	0.75	351 ± 38			14	8 April	9:00 p.m.
B. Lodge hall	3,168	40†	50	350	1.26	697 ± 28	60	6	11†	31 March	11:00 p.m.
C. Bar and grill	507	9	18	75	1.78	589 ± 28	63	6	12	21 March	8:00 p.m.
D. Firehouse bingo	541	10.5	16	125	2.77	417 ± 63	51	15	8.4	27 March	10:00 p.m.
E. Pizzeria	170	5	32	50	2.94	414 ± 58	40	5	10	14 April	8:00 p.m.
F. Bar/cocktail lounge	216	7	26	55	3.24	334 ± 120	50	5	13	25 March	10:00 p.m.
G. Church											
Bingo game	4,224	20	8	150	0.47	279 ± 18			13	31 March	10:00 p.m.
Sunday service	4,224	0	31	300	0	30			0	13 May	11:00 a.m.
H. Inn	338	2.5	12	70	0.74	239 ± 9	22	10	3.5	23 March	1:00 p.m.
I. Bowling alley	918	14	20	128	1.53	202 ± 19	49	5	11	25 March	8:00 p.m.
J. Hospital waiting room	93	2	12	19	2.15	187 ± 52	58	6	11	28 March	10:30 p.m.
K. Shopping plaza restaurant											
Sample 1	1,369	2.5	18	95	0.18	153 ± 8	59	5	2.6	24 March	7:30 p.m.
Sample 2	1,369	2.5	18	50	0.18	163 ± 4	36	10	5	24 March	9:30 p.m.
L. Barbeque restaurant	225	2	10	25	0.89	136 ± 17			8	24 March	9:00 p.m.
M. Sandwich restaurant A											
Smoking section	781	2.25	20	30	0.29	110 ± 36	40	5	7.5	25 March	8:00 p.m.
Nonsmoking section	326	0	20	40	0	55 ± 5	40	5	0	25 March	7:30 p.m.
N. Fast food restaurant											
Sample 1	360	1.5	40	30	0.42	109 ± 38			5	26 March	7:00 p.m.
Sample 2	360	0	7	30	0	30			0	26 March	1:30 p.m.
O. Sports arena	823,000	759†	12	6,700‡	0.09	94 ± 13	24	5	11†	29 March	10:00 p.m.
P. Neighborhood restaurant/bar	250	1	12	35	0.40	93 ± 17			2.9	25 March	8:30 p.m.
Q. Hotel bar	169	1	12	25	0.59	93 ± 2			8		2:30 p.m.
R. Sandwich restaurant B											
Smoking section	781	1	8	30	0.13	86 ± 7	55	5	3.3	14 April	11:00 a.m.
Nonsmoking section	326	0	21	50	0	51	55	5	0	14 April	1:30 p.m.
S. Roadside restaurant											
Sample 1	90	1	18	5	1.12	107§			20	29 March	3:00 p.m.
Sample 2	90	0	2	3	0	30			0	29 March	3:00 p.m.

*Only the cocktail party microenvironment was unventilated. †Estimated. See (31). ‡Paid attendance. §Calculated, equilibrium value.

counted for by ventilation, recirculation, infiltration, decay, mixing, and average smoking behavior. We conclude that the finite D_s RSP levels shown in Fig. 3 are indeed generated primarily by cigarette smoke and that this is consistent with the predictions of our model.

The Range of Public Exposure

We can now model the full range of exposure of the nonsmoking public to cigarette smoke. Equation 4 may be rewritten as

$$R = 25.6 \frac{P_s}{C_s} \qquad (5)$$

where P_s is the occupancy (persons per 1000 square feet). (The volumetric measure is implicit, assuming a 10-foot ceiling.) The P_s is three times the density of habitual smokers D_{hs} and nine times the density of active smokers D_s (*31*). A family of RSP curves is generated from Eq. 5 by varying C_s and P_s over their ranges. Representative samples of this family are plotted in Fig. 4. A lower limit for C_s of about one-half to one mean air change per hour has been determined experimentally and theoretically for removal of cigarette aerosol from private dwellings ventilated by infiltration and from commercial establishments whose mechanical ventilation is poor. A realistic upper bound for C_s may be obtained from the well-ventilated environment of the commercial airliner. A mechanical (design) ventilation rate of 15 to 20 air changes per hour with no recirculation is typical of the Boeing 707 (*32*). The best ideal decay rate measured in the experiments was six air changes per hour. Assuming a mixing factor of unity, we calculate an upper limit for C_s of 26 air changes per hour. The practical range for P_s is obtained from the ASHRAE (*29*), which specifies mechanical ventilation rates for typical average occupancies in various structures. For commercial structures, these densities (in persons per 1000 square feet) range from 10 for general office space to 70 for dining rooms to 150 for such places as stand-up bars, auditoriums, arenas, and commercial aircraft. The design ventilation rate C_v is typically determined from both the design occupancy and the intended use of the structure. For example, 15 to 25 cubic feet per minute per occupant is specified for general office space, 10 to 20 for dining rooms, and 30 to 40 for cocktail lounges. In 1975, *ASHRAE Standard 90-75*, "Energy conservation in new building design," decreased these rates by factors of one-half to one-third. *ASHRAE Standard 62-73* is currently being revised to specify higher rates of ventilation for premises in which smoking is permitted. How effective would increases in C_v be in lowering the levels of RSP from tobacco smoke? Equation 5 shows that such levels decrease only exponentially with increasing C_s. Furthermore, as Kalika *et al.* (*33*) observed, "the current practice of recirculation or reuse of air is largely dictated by the economics of heating and cooling, with little regard for changes in indoor air quality." That is, ventilation may be subject to arbitrary reduction by building management or by legislative or bureaucratic fiat; in many nonurbanized areas, it may not even be regulated by building codes (*34*).

Figure 4 illustrates the estimated range

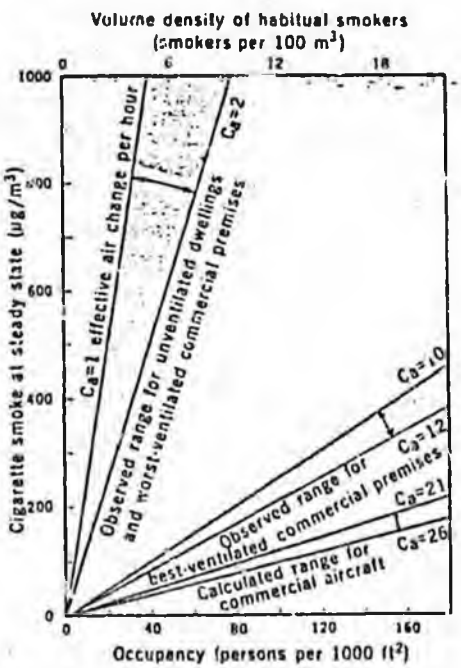

Fig. 4. Theoretical steady-state density of respirable particulates from environmental cigarette smoke in habitable indoor spaces, as related to the design occupancy P_s. On the average, one-third of adults are habitual smokers; for every three such smokers, we calculate that an average of one cigarette burns constantly throughout a 16-hour day. According to standard engineering criteria (*29*), occupancy and the type of microenvironment determine the design rate of mechanical ventilation C_v. The effective air change rate (C_s) for the removal of tobacco aerosol from room interiors is determined by C_v, by mixing, and by the rate of adsorption of tobacco particles on room surfaces. Generally C_v and hence C_s increase with P_s. [Typical P_s (in persons per 1000 square feet) ranges from 10 for office buildings to 70 for restaurants to 150 for bars, sports arenas, and aircraft (*29, 32*).] We estimate the practical range of C_s to be from 1 to 12 air changes per hour. It appears that over the combined practical ranges of P_s and C_s, repeated exposure to tobacco smoke can lead to annual RSP burdens that violate the primary annual NAAQS.

of exposure of the nonsmoking public to RSP from cigarette smoke. The actual dose of RSP is clearly a function of personal activity patterns; differences in respiration rate also affect the dose. Many different scenarios can be imagined. In the following, we express a range of RSP burdens from the cigarette aerosol relative to a typical RSP ambient background level. For an air shed (air quality control region) that is in compliance with the annual secondary (public welfare) NAAQS for TSP of 60 $\mu g/m^3$, the RSP fraction of the ambient aerosol is conservatively estimated at 50 $\mu g/m^3$ and is likely to be composed largely of combustion-produced sulfates (*35*). Since the particle size distributions of this fraction and the cigarette aerosol are both in the respirable range, we first compare them on a mass basis, without regard for differences in the chemical composition of each.

Let A, B, C, and D be nonsmokers who dwell in the same air shed and who breathe at the average rate of 20 m^3/day. All have different occupations and lifestyles that lead, as we shall see, to dramatically different RSP burdens.

Nonsmoker A is a mailman who walks a regular route and is able to live in a completely tobacco smoke-free environment. He is exposed only to the background ambient and therefore inhales 365 mg of RSP annually.

Nonsmoker B is an office worker who works a 40-hour week 50 weeks per year in a 40-m^3 office with two other persons, one of whom is a habitual smoker. Replacing D_s in Eq. 4 with $D_{hs}/3$, we find that B's mass RSP exposure is more than three times that of A (we calculate an expected C_s of 1.1 for office buildings).

Nonsmoker C is a musician who entertains in a popular, poorly ventilated nightclub 8 hours nightly, 5 nights per week, 50 weeks per year. The average P_s in the club is 100 persons per 1000 square feet (about 33 smokers). Further, C shares a 100-m^3 apartment with a roommate who is a chain smoker. C is exposed to the roommate's smoke 5 hours per day, 7 days per week, annually. By using Eqs. 4 and 5 and a C_s of one air change per hour, we find that C's mass RSP burden is more than 15 times that of A.

An alternative way of approaching the excess RSP exposure is in terms of cigarette equivalents. The cigarette with the least tar in the May 1978 FTC scale has 0.55 mg of TPM. In these terms, B's excess RSP burden is equivalent to 5 cigarettes per day and C's burden to 27 cigarettes per day. However, this may un-

derestimate the true impact, since many nonsmokers have greater sensitivity to smoke than smokers (*7*).

Nonsmoker D is a flight attendant who spends 40 hours per week, 50 weeks per year on board a commercial airliner with a C_a of 23 air changes per hour. The average P_a on the plane is 150 persons per 1000 square feet. D's RSP burden is nearly twice that of A. Even with one of the best ventilation systems in use, the high density of smokers causes a substantial increase in mass RSP inhaled by D.

The following three considerations may help to place these scenarios into perspective. First, an annual exposure 1.5 times that of A is sufficient to exceed the primary annual NAAQS; the exposure of D, B, and C to RSP all violate the standard by factors of 1.2, 2, and 10, respectively. Second, pulmonary clearance studies show that the half-life of inert respirable particles (2.8 μm in MMD) in the lungs of nonsmokers is ~ 70 days (*36*): residence of RSP in the lungs is prolonged. Third, in a series of pulmonary lavage studies on 400 nonrandomly selected volunteers (250 nonsmokers and 150 smokers) (*37*), two of the nonsmokers had tarry lavage fluids with pigmented pulmonary alveolar macrophages strikingly similar to those found in smokers. In these two volunteers, the levels of aryl hydrocarbon hydroxylase, an inducible carcinogen-detoxifying pulmonary enzyme, were intermediate in value between the levels found in smokers and most nonsmokers. These findings were attributed to the effects of exposure to tobacco smoke (*38*).

Health Policy Implications

There is good toxicologic evidence that elevated levels of particulates in outdoor air, perhaps in combination with other pollutants, cause illness and death during air pollution episodes (particulate levels in excess of 1000 $\mu g/m^3$ per 24 hours). There is much epidemiologic evidence, some of it conflicting, that lower levels of particulates, perhaps in combination with other pollutants, affect respiratory health adversely when exposure to them is sustained (*39*). (This evidence has been used to establish the thresholds for harm on which the primary annual NAAQS for TSP is based.) There is excellent toxicologic evidence that mainstream cigarette smoke causes chronic obstructive pulmonary disease (*7, 40*). Epidemiological evidence, some of it conflicting, indicates that exposure to tobacco smoke in the home affects respiratory health adversely (*7, 41*). Finally, there is excellent evidence that mainstream cigarette smoke causes cancer in many organs (*7*). Sidestream smoke is chemically identical to mainstream smoke, and typically is more concentrated (*2*). Coke-oven emissions, which chemically are similar to tobacco smoke, are associated with increased rates of many forms of cancer in coke-oven workers (*42*). Animal studies demonstrate that the particulate phase of tobacco smoke contains numerous potent carcinogens and tumor promoters, initiators, and accelerators (*7*). One of these, benzo[*a*]pyrene, was detected at a concentration of 40 parts per million in ambient tobacco smoke (*13*). Strong evidence supports a correlation between the magnitude of long-term exposure to carcinogens and the incidence of cancer (*43*). Therefore, given the efforts by public health authorities to eliminate involuntary public exposure to saccharin and the fire retardant Tris—which have, respectively, one-fifty-thousandth and one-tenth the experimental carcinogenic potency of benzo[*a*]pyrene alone (*44, 45*)—similar efforts to prevent involuntary exposure to ambient tobacco smoke (*46*) appear justified.

Conclusions

We have defined the probable range of exposure of the nonsmoking public to a common pathological aerosol, cigarette smoke. We showed, both experimentally and theoretically, that under the practical range of ventilation conditions and building occupation densities, the RSP levels generated by smokers overwhelm the effects of ventilation and inflict significant air pollution burdens on the public. Our observations show that levels of RSP in places where tobacco is smoked greatly exceed levels found in smoke-free environments, outdoors, and vehicles on busy commuter highways. Our experimental results are consistent with the large differences in 24-hour average RSP levels reported for smoking and nonsmoking homes in the Harvard Six-City Study (*47*), with a survey of short-term RSP levels in commercial and public buildings in Houston (*28*), and with other studies of tobacco-generated TSP (*7, 11-13*).

Attempts to reduce RSP levels from smoking by increasing the rate of mechanical ventilation or the efficiency of filtration yield exponentially diminishing returns for linear increases in ventilation energy (and cost). Moreover, efforts to conserve energy in buildings will decrease ventilation rates (*48*). Therefore, increased ventilation does not appear to be a solution to the problem. Indoor air is a resource whose quality should be maintained at a high level. Smoking indoors may be incompatible with this goal (*33, 49*).

Further research is necessary to define the integrated particulate exposure of various segments of the population; compliance with the NAAQS, as indicated by the establishment of outdoor TSP sampling stations, does not imply protection of the public from excessive RSP burdens. Repeated exposure to ambient cigarette smoke imposes air pollution burdens on nonsmokers that exceed the primary annual NAAQS. It appears that the RSP burdens from ambient tobacco smoke are so large that they must be incorporated explicitly in future epidemiological assessments (*50, 51*) of the relation between particulate levels and morbidity or mortality.

The Clean Air Act of 1970 and its amendments mandate the control of public exposure to outdoor TSP. However, little legislative attention has been devoted to the quality of indoor air—other than the passage of the Public Health Service Act of 1978, which provides for an ongoing study of the health costs of indoor air pollution. Clearly, indoor air pollution from tobacco smoke presents a serious risk to the health of nonsmokers. Since this risk is involuntary, it deserves as much attention as outdoor air pollution.

Note added in proof: A very recent epidemiological study concluded that long-term exposure to tobacco smoke, limited to the work environment only, is deleterious to the nonsmoker and significantly reduces small-airway function to the same extent as smoking one to ten cigarettes per day. This is consistent with scenario B (*52*). *ASHRAE Standard 62-73R*, a proposed standard for ventilation required for minimum acceptable indoor air quality, has been published (see *29*). Using data supplied in the standard, we calculate a C_a of ≤ 1.28 for office buildings where smoking is permitted.

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Paying the piper

When experiments on animals turn up carcinogens in our favorite foods and everyday consumer items, some critics invariably dismiss the data as coming only from animals. The tobacco industry has, of necessity, taken the opposite tack; for years it has argued that the evidence incriminating cigarettes shows merely a "statistical association" because it comes from studies of human deaths, not animal experiments. By now, though, the evidence that cigarettes shorten life is overwhelming; the causal connection is as firmly established as any in medicine. "Indeed," writes John Cairns, a molecular biologist and expert on cancer, "in retrospect, it is almost as if Western societies had set out to conduct a vast and fairly well controlled experiment in carcinogenesis, bringing about several million deaths and using their own people as the experimental animals."

But the cancer connection, which was the most obvious and easiest to establish, is not the major cause of death in smokers. Rather, it is coronary heart disease. Second comes lung cancer. General deterioration of the lung tissue is third. After these three major causes, a variety of other diseases and cancers make a further contribution to the high death rate of smokers. Cancers of the larynx, mouth, esophagus, bladder, kidney, and pancreas are all more common in smokers than nonsmokers. So are ulcers of the stomach and intestine, which are more likely to be fatal in smokers.

Women who smoke during pregnancy run a significant risk that their babies will die before or at birth. The newborns are likely to weigh less, to arrive prematurely, and to be more susceptible to "sudden infant death."

The risk of smoking is, in general, a 70 percent increase in the probability of dying at any age—100 percent for a two-pack smoker. As a rule of thumb, each cigarette knocks about five minutes off the smoker's life. For an average habit, that adds up to six or seven years (more for some, less for others). In the meantime, smokers lose more work days to illness than nonsmokers and spend more time in the hospital.

The ill effects of smoking are mostly, but not entirely, a consequence of the amount of inhaled smoke. Virtually all cigarette smokers inhale, even those who say they do not, and they continue to do so when they switch to pipes or cigars.

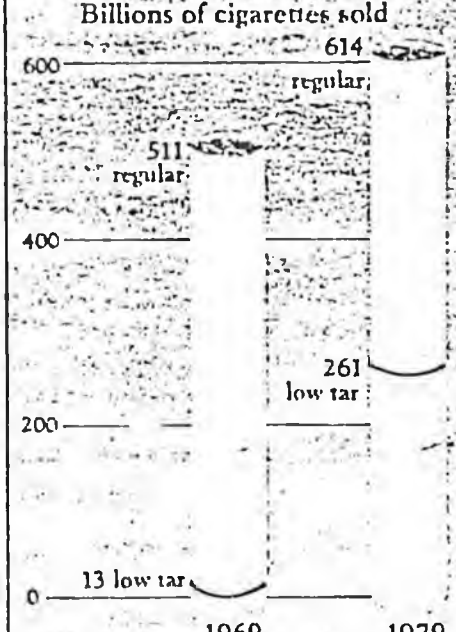
Cigarette smoke is loaded with poisons and carcinogens. The "tars," particles of organic matter, are largely responsible for causing cancer, or, perhaps, for promoting the growth of tumors started by other agents. Nicotine and carbon monoxide are thought to be the main cause of heart disease; there is debate about their relative importance.

In response to public worry about the health hazards of smoking, cigarette manufacturers over the last decade have progressively lowered the tar and nicotine content. These "lighter" brands appear to be effective in reducing rates of lung cancer. There may be a similar effect for heart disease, but the evidence is not as good. Smokers of low-tar cigarettes appear to get no protection from other respiratory illnesses.

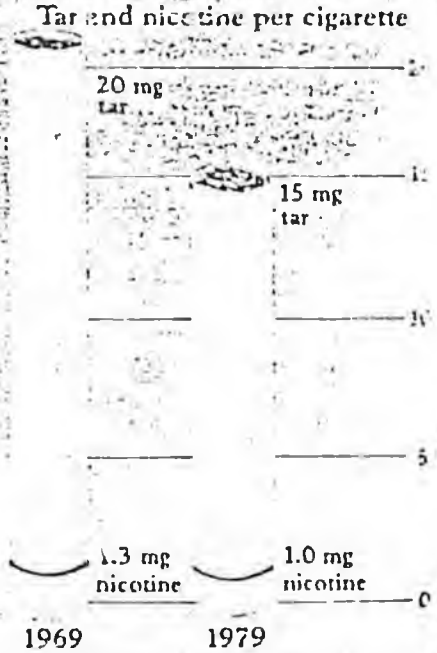
In order to elicit recommendations for his 1981 report on smoking, Surgeon General Julius B. Richmond recently called a conference to set research priorities for low-tar cigarettes. One area of concern was quickly established: The light cigarettes may pose a risk for pregnant women. Dr. Jesse Steinfeld, dean of the Medical College of Virginia in Richmond, speculates that the villain in low-tars may be carbon monoxide, since it "binds hemoglobin and may restrict the oxygen a baby needs from the mother's blood."

Carbon monoxide may turn out to be harmful to adults as well. In fact, questions abound on the safety of light cigarettes. We still do not know whether smokers who switch to low-tar cigarettes smoke more and inhale more deeply. If they do, those smokers are at least partly offsetting the presumed advantage of switching. Another question concerns whether the availability of these "safer" cigarettes has encouraged a large number of young people who otherwise would not have smoked to begin the habit. All in all, we are still far from knowing whether the low-tar, low-nicotine cigarettes will ultimately prove to be a Good Thing.

—*W. D.*



Low tar sales have soared in a decade.

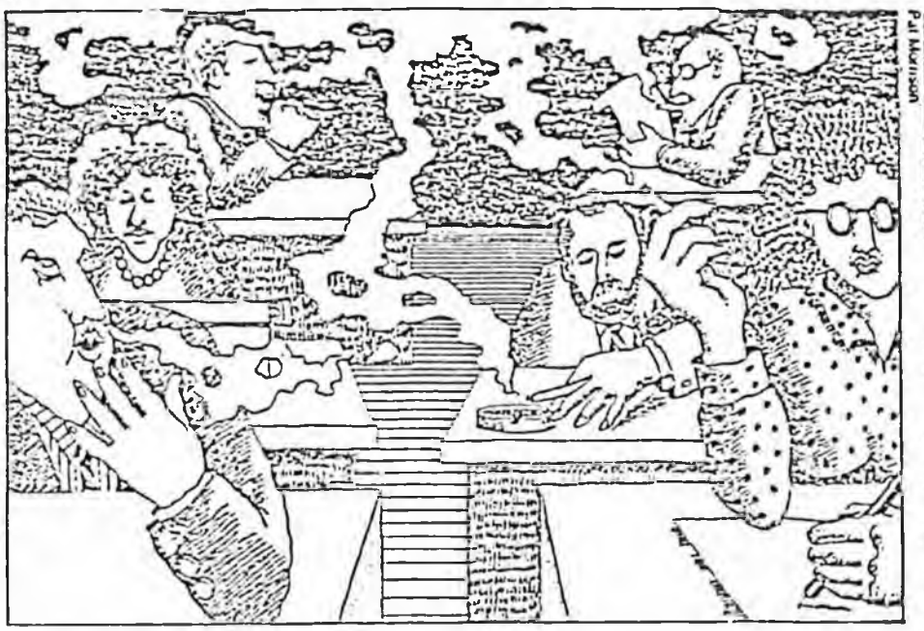


Tar and nicotine content has dropped.

Smoke gets in your eyes . . . and lungs

SAN DIEGO—For years, nonsmokers have endured the tobacco fumes of the smoking public in restaurants, elevators, conference rooms, and the workplace—all the while worrying about the effects on their health. Now there is evidence to back up their fears: A study at the University of California at San Diego shows that the breathing ability of nonsmokers exposed to tobacco smoke on the job is measurably less than that of workers in smoke-free environments.

Researchers James White and Herman Froeb tested 2 100 men and women from the San Diego area by measuring the rates at which they could force air out of their lungs. On average, they found a ten percent lower exhalation rate among nonsmokers who had worked in smoked-filled offices more than 20 years. These so-called passive smokers displayed breathing rates comparable to those of persons who smoked up to ten cigarettes a day.



Paul Madison

There is no evidence as yet that passive smokers risk such illnesses as emphysema and cancer. But their reduced lung capacity does indicate that nonsmokers forced to breathe smoke at work suffer the same kind of damage to small airways deep inside the lungs as do smokers. That damage is in the form of mucous and scar tissue, which block the smooth flow of air and can inhibit the transfer of oxygen into the bloodstream. "This is permanent damage occurring in people who have chosen not to smoke," says White, who studiously avoids the smokers among his colleagues at the university.

Unrestricted smoking at work is permitted by nearly 75 percent of all employers in the United States. Dozens of lawsuits have been filed by nonsmokers and most have been settled out of court. John Banzhaf, executive director of Action on Smoking and Health, knows of only one case that came before a judge, who ruled in favor of the plaintiff. The judge reportedly was impressed by the fact that the plaintiff's company had banned smoking in a nearby computer room because the equipment malfunctioned when exposed to cigarette smoke. He ruled that if smoking could be curtailed for the sake of a machine, it could be curtailed for the sake of a human being.

through sponsorship of the U.S. Department of Agriculture, National Science Foundation, Southwest Border Regional Commission, Four-Corners Regional Commission, and the state of California.

Germplasm collections have been made from wild guayule plants in Mexico and Texas. Plantings have been established to test yields, to increase seed supplies, and to conduct plant breeding work. Test plots have been established to determine desirable planting and cultivating practices. Research is being conducted on the possibility of increasing rubber yield by treating guayule plants with plant growth regulators.

The recent development of a seed coating process to promote germination, and the development of selective herbicides, will make direct seeding in field plantations a possibility. Eliminating nursery or greenhouse propagation could produce considerable savings in production costs.

The only guayule yield figures now available are estimates developed during the ERP. During the life of the ERP the per hectare, approximately 480 kg of guayule rubber per year. Kelly (*15*) obtained yields of approximately 860 kg per hectare per year from one test plot in California. Foster *et al.* (*16*) have outlined the state of the art of guayule technology and described present and projected world rubber market conditions and areas of the United States where conditions favor guayule cultivation.

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Indoor Air Pollution, Tobacco Smoke, and Public Health

James L. Repace and Alfred H. Lowrey

Serious health effects from air pollution have led to federal standards for the regulation of outdoor exposure levels. However, Americans spend about 90 percent of their time indoors (*1*). Thus the levels of indoor air pollution are important in determining total exposure to air pollutants (*2–6*). Indeed, in a recent review article (*4*) it was concluded that indoor air pollution in public office buildings is of greater potential harm than the outdoor variety, and that these exposures may constitute a real threat to the health of many urban people. The U.S. Surgeon General asserted in his report on *Smoking and Health* that tobacco smoke can be a significant source of atmospheric pollution in enclosed areas (*7*). Some 53 million U.S. smokers consumed 615 billion cigarettes in 1978 (*8*). Thus it is apparent that indoor air pollution from tobacco smoke is pandemic.

In the presence of cigarette smoke, many normal nonsmokers experience eye and throat irritation, headache, rhinitis, and coughing; allergic persons report wheezing, sneezing, and nausea as well. Particularly acute symptoms may be found in infants, children, persons with cardiovascular or respiratory disease, and wearers of contact lenses (*7, 9*). Determining the extent of the exposure of nonsmokers to cigarette smoke is important because smoking is a cause of chronic obstructive pulmonary disease, cardiovascular disease, and lung cancer, and is associated with cancers in other parts of the body (*7*); because these diseases also occur in nonsmokers; and because the products of tobacco combustion have been detected in nonsmokers (*10*).

Although measurements of indoor carbon monoxide pollution from smoking are abundant (*7*), published reports of the exposure of the population to the particulate phase of ambient tobacco smoke are rare (*7, 11–13*). Furthermore, a comprehensive theory of the generation and removal mechanisms for tobacco particulates in naturally or mechanically ventilated habitable spaces has not been presented.

We therefore undertook a systematic study of the levels of respirable suspended particulates (RSP) in several common indoor environments in an attempt (i) to determine the relation of these levels to the aerosol from tobacco smoking, (ii) to understand the effect of ventilation on tobacco smoke concentra-

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for estimating the range of the public's exposure. Our goal was to provide a quantitative basis for assessing the health hazards to nonsmokers posed by repeated exposure to tobacco combustion products.

Model Development

To relate the contribution of smoking to indoor RSP requires a model describing the behavior of the tobacco aerosol in indoor spaces. Bridge and Corn (*6*) found that a reduced form of an equation by Turk (*14*) reliably predicts carbon monoxide (CO) concentrations from tobacco smoke in ventilated spaces and so is of major value in assessing the possible hazards in occupied spaces (*11*). The equation is not valid, however, for a pollutant that is affected by physical decay due to adsorption on room surfaces. Penkala and DeOliviera (*15*) showed that decay of the tobacco aerosol in a well-mixed unventilated chamber is exponential.

We modify the Turk equation in differential form by adding a decay term to the removal rate and equating the rate of change of pollutant mass to the algebraic sum of the generation and removal rates. The solution yields the density $A(t)$, in micrograms per cubic meter, of smoke in the room as a function of time:

$$A(t) = A_{eq}(1 - e^{-t/\tau}) \qquad (1)$$

where $A_{eq} = G\tau/V$ is the equilibrium concentration of the pollutant in the room, and where the time constant

$$\tau = \frac{\tau_a \tau_v}{m(\tau_a + \tau_v)} \qquad (2)$$

is the mean ventilation time, or the time for the smoke concentration to decrease to $1/e$ of its value (where e is the base of natural logarithms); V is the room volume in cubic meters; $\tau_v = V/Q$ is the ideal ventilation time, or the time required to replace a volume of air equal to the volume of the room by ventilation and infiltration; Q is the volume rate of ventilation and infiltration; τ_a is the ideal decay time, a time constant associated with the removal of a pollutant from a room through adsorption on surfaces and filtration; and m is the mixing factor, an empirically determined number (*16*) that modifies the ventilation time as τ_v/m, where $m \leq 1$ ($m = 1$ implies ideal mixing). Corn (*11*) suggested values of m for various ventilation systems (Table 1). We postulate that m also modifies the ideal decay time as τ_a/m. The pollution generation rate, in micrograms per minute, is given as $G = nC_0/t_b$, where n is the number of cigarettes being smoked at time t; C_0 is the total particulate matter (TPM) from both sidestream and exhaled mainstream smoke; and t_b is the duration of cigarette smoking.

Equation 1 has two special cases: (i) in the case of ventilation only ($\tau = \tau_v/m$) it becomes the reduced Turk equation of Bridge and Corn (*6*), with $m = 1$; and (ii) in the case of adsorption only (the unventilated room), $\tau = \tau_a/m$. Then, if the generation of smoke ceases at time t_b, prior to equilibrium, A will decay according to

$$A(t) = A_0 e^{-mt/\tau_a} \qquad (3)$$

where A_0 is a constant related to the equilibrium concentration by

$$A_0 = A_{eq}[e^{mt_b/\tau_a} - 1]$$

factor m, after Corn (*11*). The mixing factor is an empirical number that accounts for room-specific effects on pollutant transport. Pollutant removal is more rapid in a well-mixed atmosphere (where m is large) than in a poorly mixed, stable one (where m is small). Factors that affect m include type and placement of ventilation grills, ventilation flow rates, inhomogeneous pollutant distribution, barriers, circulation fans, and room traffic.

Configuration of air supply system	m
Perforated ceiling*	1/2
Trunk system with anemostats	1/3
Trunk system with diffusers	1/4
Natural draft and ceiling exhaust fans	1/6
Infiltration and natural draft	1/10

*This is the best standard condition.

Summary. An experimental and theoretical investigation is made into the range and nature of the exposure of the nonsmoking public to respirable suspended particulates from cigarette smoke. A model incorporating both physical and sociological parameters is shown to be useful in understanding particulate levels from cigarette smoke in indoor environments. Observed levels of particulates correlate with the predictions of the model. It is shown that nonsmokers are exposed to significant air pollution burdens from indoor smoking. An assessment of the public health policy implications of these burdens is presented.

described by Penkala and DeOliviera (*15*) for $m = 1$.

The modified Turk equation (Eq. 1) contains only measurable quantities, and thus in principle can be used to estimate the concentration of TPM or CO from tobacco smoke (or other indoor air pollutants), as a function of time, for any room for which the pollutant generation rate, volume, and mean ventilation time are known.

Controlled Experiments

Equation 2 shows that the mixing factor affects the time constant for decay as well as ventilation. Experiments under conditions of known ventilation were therefore necessary to assess the influence of mixing factors, decay time constants, and generation rates on the growth and decay of tobacco smoke particulates. To increase the usefulness of the experimental values determined for the mean ventilation time for the removal of tobacco smoke, we conducted these experiments in actual occupied spaces rather than in experimental chambers.

A piezobalance (TSI model 3500) (*17–19*) was used in sampling the aerosol. It collects respirable particulates (*20*) between 0.01 and 3.0 micrometers in diameter with near 100 percent efficiency (decreasing to 50 percent at 3.5 μm and to 10 percent at 4 μm). The sampling rate is 1 liter/min (*18*); the sampling time is variable. Factory-calibrated with welding smoke, the detecting crystal in the instrument used has a sensitivity of 5.74 μg/min-m^3 per hertz. The instrument underestimates the mass concentration of tobacco aerosol by about 15 percent compared to measurements made with low-volume filter sampling techniques. Readings can be affected by changes in humidity; the maximum expected error due to changes in relative humidity when sampling a hygroscopic aerosol (such as tobacco smoke) is given as $\pm$ 10 μg/m^3. The overall instrument error is about $\pm$ 10 percent compared with low-volume filter measurements of welding smoke (*19*). The aerosol from sidestream cigarette smoke (that portion emitted by the burning tip), an important component of many indoor aerosols, is lognormal, with 99 percent of the mass < 1 μm in aerodynamic diameter and with an initial mass median diameter (MMD) from 0.2 to 1.5 μm depending on dilution (*20, 21*). The relative particle sizes of fresh sidestream and mainstream smoke (the latter being that portion inhaled by the smoker) are about the same; for ex-

Table 2. Parameters for Eq. 1, as determined with experiments 1 to 3 (unventilated rooms).

Experiment	A_{eq} ($\mu g/m^3$)	A_0 ($\mu g/m^3$)	τ_a/m (min)	m	r^{2*}	C_0† (mg of TPM)	Cigarette condition
1‡	530	503	10	1	.98	12.3	Smoldered
2‡	5178	551	89	1/9	.42	16.0	Smoldered
3§	1773	681	16.4	< 1	.81	23.0	Smoked

*Coefficient of determination for the decay curve. †The estimated amount of TPM liberated if the entire cigarette had been consumed, according to FTC protocol. The FTC mainstream TPM level for this cigarette is 16 mg (24). ‡V = 21.9 m^3. §V = 29 m^3.

haled mainstream smoke, particle size is estimated to be ~ 0.8 μm (MMD). Since the ambient cigarette smoke aerosol is reproducible and coagulates very slowly, it has been used as a test aerosol (*21*) and in evaluation of heating, air-conditioning, and ventilating systems (*22*). [The bulk of the ambient tobacco aerosol is probably due to cigarettes, since less than 15 percent of smokers smoke cigars or pipes (*23*).]

Unventilated Growth and Decay of Tobacco Smoke

Experiments were carried out to determine the usefulness of Eq. 3, which predicts a rapid decay for good mixing and a slow decay for poor mixing; and also to discover the limits of τ_a/m.

Experiments 1 and 2 were conducted in a wood-panelled den in a private residence. In the geometric center of the room (volume, 21.9 m^3), a popular filter cigarette [containing 65 millimeters of tobacco and ranking 94th on the Federal Trade Commission (FTC) scale of tar and nicotine content (17 milligrams of tar and 1 milligram of nicotine) (*24*)] was ignited and allowed to smolder until 89 percent of its tobacco was consumed. During the first experiment, two box fans (51 centimeters in diameter) with antiparallel exhausts were used to ensure ideal mixing; each fan's exhaust, measured with a Velometer, was 55 m^3/min. The growth and decay of the RSP were measured with the piezobalance. Experiment 2 was similar to experiment 1, except that the cigarette was extinguished after 75 percent of its tobacco was consumed and the circulating fans were not used, so that mixing was natural. The results of both experiments are plotted in Fig. 1, with the background levels of RSP subtracted. The data points generally represent 1-minute average values. The differences in mixing dramatically affect the slopes of the decay curves.

The theoretical curves shown in Fig. 1 were generated by fitting the data points from the decay curves to Eq. 3 with a regression analysis; A_0 and τ_a/m were determined and used to calculate the growth curves from Eq. 1, case (ii). The ratio of the slopes of the decay curves for ideal and natural mixing yields the mixing factor for the room. Table 2 gives the values obtained for the various parameters. The value of the mixing factor obtained is in good agreement with the expected value given in Table 1 for the case of infiltration and natural draft. The growth curve for the case of natural mixing (experiment 2) shows a poor fit initially because of the effect of the warm smoke rising to the ceiling and remaining

Table 3. Field survey of indoor RSP in the absence of smoking.

Locale	Room volume (m^3)	Persons per room	Persons per 100 m^3	Indoor RSP level* ($\mu g/m^3$)	Average time per RSP sample (min)	Outdoor RSP level† ($\mu g/m^3$)	Comment
Crepes restaurant (Washington, D.C.)	124	43	35	29	20	44	No smoking section; aroma of frying crepes evident
Sandwich restaurant (Laurel, Md.)	326	40	12	55	21	40	No smoking section; near kitchen; three smokers in smoking section
Sandwich restaurant (Laurel, Md.)	326	55	17	51	21	55	No smoking section; near kitchen; one smoker in smoking section
Fast-food restaurant (Bowie, Md.)	1,400	22	1.6	38	7		Aroma of hamburgers frying
Private residence (Seabrook, Md.)	120	11	9	24	20		Cocktail party; one candle burning 6 m from RSP detector
Private residence (Bowie, Md.)	124	1	0.8	44	15		One hour after sweeping basement floor
Private residence (Greenbelt, Md.)	22	2	9	24	6		Natural mixing‡
	22	2	9	55	1		Two fans moving 110 m^3 of air per minute§
Private residence (Glenn Dale, Md.)	29	7	24	57	5		One fan moving 55 m^3 of air per minute‖
Conference room (Greenbelt, Md.)	113	10	9	53	10		Two fans moving 50 m^3 of air per minute¶
Public library meeting room (Bowie, Md.)	1,415	30	2.1	29	30		During piano recital
Library of Congress (Washington, D.C.)	27,000	130	0.48	30	10		Main reading room
Church (Bowie, Md.)	4,224	300	7	30	42		During Sunday service
Bagel bakery (Yonkers, N.Y.)	510	30	6	25	10	8	Aroma of baking bagels evident
Private residence (Hawthorne, N.Y.)	150	17	11	26	16		During dinner party

*Mean ± standard deviation for the Washington area samples, 40 ± 13 $\mu g/m^3$. †Duration of sampling, 5 minutes. ‡Experiment 2 background. §Experiment 1 background. ‖Experiment 3 background. ¶Experiment 4 background.

out of the detector's range for about 3 minutes. Experiment 3 demonstrated that Eq. 3 is valid under more general conditions, that is, when a cigarette is actually smoked.

We conclude that these experiments show that for the unventilated room, Eq. 3, the reduced form of Eq. 1, is useful in describing the growth and decay of cigarette smoke particulates.

Ventilated Growth and Equilibrium of Tobacco Smoke

An experiment was conducted in a ventilated conference room of a modern office building to test Eq. 1 in the case of removal of uniformly generated tobacco smoke by both decay and ventilation. The experiment involved measuring the growth of cigarette-generated RSP from background levels to near equilibrium. Analysis of the RSP-versus-time curve determines τ, the mean ventilation time, and C_T, the total RSP liberated from the combined sidestream and exhaled mainstream smoke.

The RSP detector was located in the geometric center of the 113-m³ room. Two fans with antiparallel exhausts were used to establish a vigorous circulation of 100 m³/min. The ideal ventilation time τ_v, calculated from the volumetric flow rates of the ventilation system, was 49.2 minutes for a complete change of air. Thirty-two cigarettes were smoked in 49 minutes by a relay of seven smokers, with an average of four persons smoking at any given time. When smoking their own brands, they averaged 9.8 minutes per cigarette; when smoking cigarettes supplied to them, they averaged 5.8 minutes per cigarette. All butts were collected and the amount of tobacco consumed was measured for each cigarette. The estimated mainstream TPM (M) (tar plus nicotine) generated by the 32 cigarettes was determined by weighting the TPM values for each cigarette (*24*) by the fraction of tobacco consumed, and adding the results to obtain M = 418 mg [TPM is emitted from cigarettes at a linear rate after the fourth puff (*25*)].

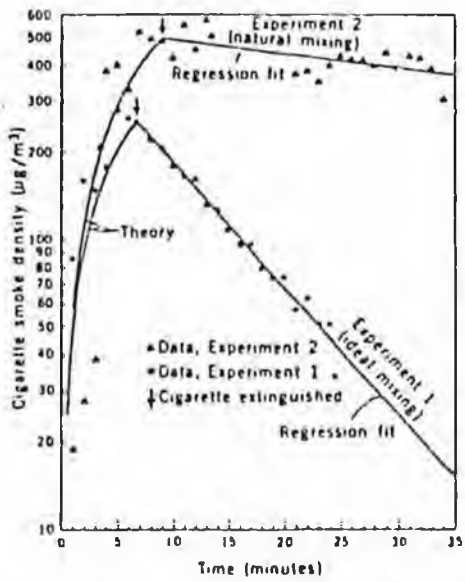

Fig. 1. Theoretical predictions versus experimental results for the growth and decay of RSP from a smoldering, average-tar cigarette in a 22-m³ unventilated room. The dramatic difference in the slopes of the decay curves reflects the difference in room air turbulence (mixing) for the otherwise similar experiments.

Figure 2 shows the growth against time of RSP from the cigarette smoke. The data points are corrected for background RSP levels and are 2-minute averages. A regression analysis using Eq. 1 yields A_{eq} = 1947 µg/m³ and τ = 14 minutes, with a coefficient of determination = .964 (from Eq. 2, τ_v = 19.5 minutes). Finally, C_T, or the total amount of RSP liberated in the room during the entire smoking period, 772 mg, is calculated by using Eq. 1; C_T/M = 1.85. This ratio represents a weighted average for six different brands of filter cigarettes that together commanded a 23 percent share of the market in 1976 (*26*).

From the goodness of fit of the theory to the data and from the observation of predicted interactive behavior among mixing, growth, and decay processes for RSP from cigarette smoke, it appears that all the room-specific factors affecting the removal of tobacco smoke (ventilation, decay, and mixing) can be combined into a single time constant τ, which can be determined for any room by regression analysis of the decay or growth-equilibrium curves, or by calculation from the equilibrium concentration if the smoke generation rate and room volume are known. The ratio of the slopes of the decay curves for ideal and natural mixing yields the mixing factor. We conclude that Eq. 1 is a useful tool for predicting the levels of tobacco smoke in both ventilated and unventilated occupied space.

Field Survey of RSP

We now address the complex problem of surveying the levels of RSP indoors and determining what portion of this aerosol may be attributed to cigarette smoke by means of Eq. 1. The problem is complicated by differences in smoking rates, numbers of smokers, room volumes, effective ventilation rates, and the TPM values of various brands of cigarettes. The problem may be simplified by assuming that smoking is a random process when it occurs among large groups of people. It follows that cigarette smoke RSP values may be treated as equilibrium values; that all of the smokers may be treated as habitual smokers who smoke identical average-tar cigarettes in the same way at the same average rate, uniformly distributed over a 16-hour day. An average smoking rate r of two cigarettes per hour is calculated from the 1975 figures for the number of U.S. smokers and the U.S. domestic cigarette consumption (*8*). In 1978, the sales-weighted average mainstream TPM value M_a was 17.6 mg for all the cigarettes sold in the United States (*7*). The estimated emission rate C_0 (combined sidestream plus exhaled mainstream TPM) from a habitual smoker is given by $E = 1.85\,rM_a$ = 65 mg/hour, where 1.85, used for the ratio C_0/M_a, is taken from the conference room experiment. Physically observable in any field survey of smoking is n_s, the number of burning cigarettes (the number of "active" smokers); n_s can be related to the number of habitual smokers n_{hs} by considering that the average time for smoking a cigarette is 10 minutes (*2*, *6*). This number and the previously calculated average smoking rate indicate that $n_{hs} = 3n_s$.

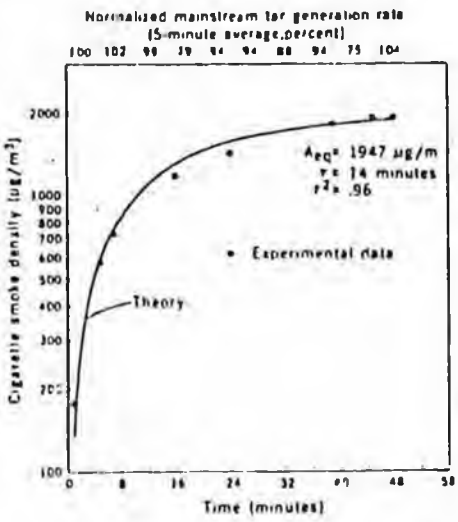

Fig. 2. Theoretical predictions versus experimental results for the attainment of equilibrium A_{eq} for the combined emission of sidestream and exhaled mainstream cigarette smoke from four chain smokers in a 113-m³ conference room with well-mixed ($m \sim 1$) ventilation in a modern office building. Under natural mixing conditions, about 11 habitual smokers would generate an equivalent equilibrated concentration of smoke. This many smokers would be expected in a group of 33 adults (well within the capacity of this 50-person conference room).

From the equilibrated form of Eq. 1, we determine that

$$R = A_{eq} = 650 \frac{D_s}{C_e} \quad (4)$$

where R is the smoker-generated equilibrium RSP level in micrograms per cubic meter, D_s is the density of active smokers (number per 100 m³), and C_e is the effective rate for the removal of cigarette aerosol (air changes per hour), with $C_e = 60/\tau$.

The aerosol sampling described in this article was performed from late March through early June 1978 in the Washington, D.C., metropolitan area. The MMD (seasonal average) of the outdoor urban aerosol for Washington in 1970 was 0.5 μm, with 90 percent of the aerosol mass less than 3 μm in aerodynamic diameter and lognormally distributed (27).

It is important to note that all of the RSP measurements we report represent time-averaged values.

Factors other than tobacco smoke may contribute to indoor RSP. These include infiltration of outdoor RSP, cooking, dust raised by indoor traffic, and industry. By restricting the sampling to nonindustrial indoor locations where tobacco smoking is absent, the effect of the remaining variables may be assessed. Table 3 gives the RSP levels for several indoor spaces in which smoking did not take place: three restaurants, four private residences, an office building conference room, two libraries, and a church during services. The mean of these measurements is 40 μg/m³. In three instances, fans were mixing the air at a high rate and RSP levels were elevated, apparently because of dust entrainment. No correlation between the volumetric density of people (occupancy) and RSP is evident. Hemperly (28), in sampling RSP in Houston, found similar RSP levels in two schools, a library, and a museum—all nonsmoking areas.

Table 4 gives the results of RSP sampling in nonsmokers' automobiles traveling along two major commuter highways (Route 50 in Maryland and U.S. I-295 in Washington) during the rush hour. The samples were taken on different days and were measured in different vehicles. In all cases, the windows were slightly open and the ventilation fans were running. The mean of the data, 38 μg/m³, is not very different from the mean of the indoor readings, 40 μg/m³ (Table 3).

The impact of actual ventilation practice on ambient RSP levels from smoking was investigated at eight restaurants, three cocktail lounges, two bingo games, a dinner-dance, a bowling alley, a sports arena, and a hospital emergency waiting room. For contrast, one unventilated private residence was sampled during a cocktail party. With the exception of the hospital waiting room and the hotel bar, each space sampled represented the major part of the building and was subject to ventilation requirements specified by building codes. Sampling was generally performed well after opening time to ensure that an approximately steady-state level of smoking had been reached.

Table 4. Levels of RSP in nonsmokers' cars during rush-hour traffic on a busy commuter highway in Washington, D.C., measured with the vehicles' windows slightly open and the ventilation fans running. Each car carried four persons and had a volume of 2 m³, so that the occupancy was equal to 200 persons per 100 m³.

Date	Time	Sampling time (min)	RSP level (μg/m³)
23 March	a.m.	10	40
23 March	p.m.	35	20
24 March	a.m.	20	54
28 March	a.m.	26	49
31 March	a.m.	8	25
Mean ± standard error			38 ± 15

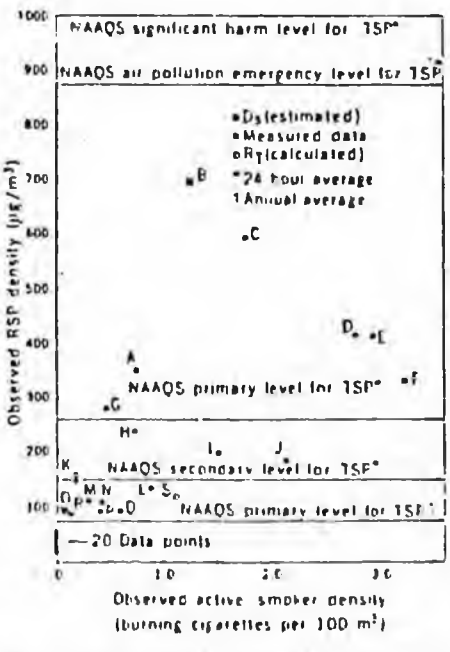

Fig. 3. Results of a field survey of short-term time-averaged levels of RSP in 38 enclosed spaces (see Tables 3, 4 and 5). Levels corresponding to federal standards for TSP are indicated for comparison only. The microenvironments include ten restaurants, three cocktail lounges, two bingo games, a dinner-dance hall, a bowling alley, a sports arena, two libraries, a church, a hospital waiting room, five vehicles, and five residences. The letters A through S correspond to those given in Table 5. The effective air change rates for microenvironments A and S are known from experiments to be C_e = 1.5 and 9.2, respectively.

The piezobalance and a stopwatch were used to take tabletop-level RSP samples for periods ranging from 10 to 50 minutes (mean, 20 minutes). The piezobalance was equilibrated in advance to avoid errors due to changes in temperature or humidity.

The room dimensions were estimated and the number of active smokers was sampled periodically throughout the measurement period and averaged. It was usually not possible to sample the premises when there was no smoking; in most cases, the RSP outside the premises was sampled for comparison. Table 5 gives the results of the measurements. Figure 3 shows the average density of active smokers (defined as the number of burning cigarettes per 100 m³) plotted against the total indoor RSP sampled. As a guide to whether a given datum is "high" or "low," the National Ambient Air Quality Standards (NAAQS) for total suspended particulates (TSP) are also shown. Since a specific averaging time is incorporated into these standards, violation of the standard is not demonstrated here. However, repeated exposure to such elevated levels can lead to "violation" of the annual standards, as will be shown later. Note that all the data for finite smoker density exceeded the level of the annual primary (health-related) NAAQS, whereas none of the data for zero smoker density exceeded this level. Further, the background RSP measured outside the smoking premises suggests that the source of these elevated levels was not the outdoor air. The mean and the standard deviation for the outdoor RSP are 46 ± 13 μg/m³, and in every case the outdoor level is less than the indoor. In certain cases, indoor controls are available. In bingo game 2, held in the nave of a church, the active smoker density was 0.47 per 100 m³, the occupancy was 3.6 persons per 100 m³, and the RSP density was 279 μg/m³ (Table 5). By contrast, during the tobacco smoke-free religious services, despite an occupancy of 7.0 persons per 100 m³, 30 burning votive candles, and several processions, the RSP density was 30 μg/m³. The elevated RSP levels in the bingo game clearly appear to be due to smoking. Similarly, measurements taken in the nonsmoking section of a sandwich restaurant showed considerably lower levels than in the smoking section, indicating that the contribution of smoking to RSP was much larger than the effect of cooking, even at the low cigarette densities shown (Table 5). Figure 3 shows that, in general, RSP levels increase with active smoker density, although there is

considerable scatter in the data. The question now is whether Eq. 1 is useful in explaining this scatter.

We hypothesize that the levels of RSP for finite D_s (Fig. 3) are due to near-equilibrium levels of cigarette smoke adding to much smaller background levels, and that the scatter in the RSP levels for fixed cigarette density is due primarily to differences in the mean ventilation time τ. Analyzing the background corrected data given in Table 5, we use Eq. 4 to calculate a range for C_s between 1.2 and 10.7 air changes per hour; C_s is used instead of τ to facilitate comparison with building code-specified ventilation rates. The range determined for C_s is consistent with two known values of C_s derived from the cocktail party and roadside restaurant experiments (Table 5).

The C_s for tobacco aerosol is affected by the rate of mechanical ventilation and infiltration, the rate of smoke adsorption, and mixing. The range of mechanical ventilation and infiltration can be calculated from tables of standards determined by the American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) (*29*), the authority specified by the local building code (*30*). For each premise listed in Table 5, the recommended maximum number of outdoor air changes per hour (based on the estimated floor area, maximum occupancy, and volume) was calculated from the ASHRAE tables; a two-thirds recirculation of air (the maximum permitted by ASHRAE) was assumed. This yielded a range of 0.7 to 9.4 air changes per hour. Infiltration, resulting mainly from the opening of doors, was estimated from the actual occupancy during the sampling (*29*); we assumed a 100 percent turnover of occupants per hour. This was added to the calculated mechanical ventilation rates, giving a final estimated range of $1.3 \leq C_v \leq 13.4$ air changes per hour, where $C_v = 60/\tau_v$.

The practical range of physical decay from adsorption for cigarette aerosol can be computed from our experiments and the literature. Most establishments possess simple filters that are relatively ineffective at removing tobacco smoke (*22*). The shortest ideal decay time measured (in experiment 1) was equivalent to six air changes per hour (Table 2). By contrast, Penkala and DeOliviera (*15*) measured a mean life for tobacco smoke, under uniform mixing in a chamber with unreactive walls, equivalent to one air change per hour. These two extremes given an estimated range of $1 \leq C_d \leq 6$ air changes per hour for RSP from tobacco smoke, where $C_d = 60/\tau_d$.

The range of mixing m appropriate for the spaces listed in Table 5 is $1/4 \leq m \leq 1/2$, as determined from Table 1. By using Eq. 2, a theoretical range of mean air change rates, $1/2 \leq C_{aTh} \leq 10$ air changes per hour, is calculated from the estimated ranges for C_v, C_d, and m. This is consistent with the 1 to 11 air changes per hour determined with our model from the experimental results. In other words, the variations in the observed RSP density for fixed cigarette density can be phenomenologically ac-

Table 5. Field survey of indoor RSP sampled in the presence of smoking. Where the standard deviation is given, the value is an average of 2-minute samples; where it is not given, the sampling time is the averaging time.

Locale	Estimated volume (m^3)	Average number of smokers	Indoor sampling time (min)	Average occupancy (persons)	Active smoker density per 100 m^3	Indoor RSP ($\mu g/m^3$)	Outdoor RSP ($\mu g/m^3$)	Outdoor sampling time (min)	Occupants smoking (%)	Date	Time
A. Cocktail party*	268	2	15	14	0.75	351 ± 38			14	8 April	9:00 p.m.
B. Lodge hall	3,168	40†	50	350	1.26	697 ± 28	60	6	11‡	31 March	11:00 p.m.
C. Bar and grill	507	9	18	75	1.78	589 ± 28	63	6	12	21 March	8:00 p.m.
D. Firehouse bingo	541	10.5	16	125	2.77	417 ± 63	51	15	8.4	27 March	10:00 p.m.
E. Pizzeria	170	5	32	50	2.94	414 ± 32	40	5	10	14 April	8:00 p.m.
F. Bar/cocktail lounge	216	7	26	55	3.24	334 ± 120	50	5	13	25 March	10:00 p.m.
G. Church											
Bingo game	4,224	20	8	150	0.47	279 ± 18			13	31 March	10:00 p.m.
Sunday service	4,224	0	31	300	0	30			0	13 May	11:00 a.m.
H. Inn	338	2.5	12	70	0.74	239 ± 9	22	10	3.5	23 March	1:00 p.m.
I. Bowling alley	918	14	20	128	1.53	202 ± 19	49	5	11	25 March	8:00 p.m.
J. Hospital waiting room	93	2	12	19	2.15	187 ± 52	58	6	11	28 March	10:30 p.m.
K. Shopping plaza restaurant											
Sample 1	1,369	2.5	18	95	0.18	153 ± 8	59	5	2.6	24 March	7:30 p.m.
Sample 2	1,369	2.5	18	50	0.18	163 ± 4	36	10	5	24 March	9:30 p.m.
L. Barbeque restaurant	225	2	10	25	0.89	136 ± 17			8	24 March	9:00 p.m.
M. Sandwich restaurant A											
Smoking section	781	2.25	20	30	0.29	110 ± 36	40	5	7.5	25 March	8:00 p.m.
Nonsmoking section	326	0	20	40	0	55 ± 5	40	5	0	25 March	7:30 p.m.
N. Fast-food restaurant											
Sample 1	360	1.5	40	30	0.42	109 ± 38			5	26 March	2:00 p.m.
Sample 2	360	0	7	30	0	30			0	26 March	1:30 p.m.
O. Sports arena	823,000	759†	12	6,700‡	0.09	94 ± 13	24	5	11†	29 March	10:00 p.m.
P. Neighborhood restaurant/bar	250	1	12	35	0.40	93 ± 17			2.9	25 March	8:30 p.m.
Q. Hotel bar	169	1	12	25	0.59	93 ± 2			8		2:30 p.m.
R. Sandwich restaurant B											
Smoking section	781	1	8	30	0.13	86 ± 7	55	5	3.3	14 April	11:00 a.m.
Nonsmoking section	326	0	21	50	0	51	55	5	0	14 April	1:30 p.m.
S. Roadside restaurant											
Sample 1	90	1	18	5	1.12	107§			20	29 March	3:00 p.m.
Sample 2	90	0	2	3	0	30			0	29 March	3:00 p.m.

*Only the cocktail party microenvironment was unventilated. †Estimated. See (*31*). ‡Paid attendance. §Calculated, equilibrium value.

infiltration, decay, mixing, and average smoking behavior. We conclude that the finite D_s RSP levels shown in Fig. 3 are indeed generated primarily by cigarette smoke and that this is consistent with the predictions of our model.

The Range of Public Exposure

We can now model the full range of exposure of the nonsmoking public to cigarette smoke. Equation 4 may be rewritten as

$$R = 25.6 \frac{P_s}{C_s} \quad (5)$$

where P_s is the occupancy (persons per 1000 square feet). (The volumetric measure is implicit, assuming a 10-foot ceiling.) The P_s is three times the density of habitual smokers D_{hs} and nine times the density of active smokers D_s (*31*). A family of RSP curves is generated from Eq. 5 by varying C_s and P_s over their ranges. Representative samples of this family are plotted in Fig. 4. A lower limit for C_s of about one-half to one mean air change per hour has been determined experimentally and theoretically for removal of cigarette aerosol from private dwellings ventilated by infiltration and from commercial establishments whose mechanical ventilation is poor. A realistic upper bound for C_s may be obtained from the well-ventilated environment of the commercial airliner. A mechanical (design) ventilation rate of 15 to 20 air changes per hour with no recirculation is typical of the Boeing 707 (*32*). The best ideal decay rate measured in the experiments was six air changes per hour. Assuming a mixing factor of unity, we calculate an upper limit for C_s of 26 air changes per hour. The practical range for P_s is obtained from the ASHRAE (*29*), which specifies mechanical ventilation rates for typical average occupancies in various structures. For commercial structures, these densities (in persons per 1000 square feet) range from 10 for general office space to 70 for dining rooms to 150 for such places as stand-up bars, auditoriums, arenas, and commercial aircraft. The design ventilation rate C_v is typically determined from both the design occupancy and the intended use of the structure. For example, 15 to 25 cubic feet per minute per occupant is specified for general office space, 10 to 20 for dining rooms, and 30 to 40 for cocktail lounges. In 1975, *ASHRAE Standard 90-75*, "Energy conservation in new building design," decreased these rates by factors of 2 to 3. The current *Standard 62-73* is currently being revised to specify higher rates of ventilation for premises in which smoking is permitted. How effective would increases in C_v be in lowering the levels of RSP from tobacco smoke? Equation 5 shows that such levels decrease only exponentially with increasing C_s. Furthermore, as Kalika *et al.* (*33*) observed, "the current practice of recirculation or reuse of air is largely dictated by the economics of heating and cooling, with little regard for changes in indoor air quality." That is, ventilation may be subject to arbitrary reduction by building management or by legislative or bureaucratic fiat; in many nonurbanized areas, it may not even be regulated by building codes (*34*).

Figure 4 illustrates the estimated range

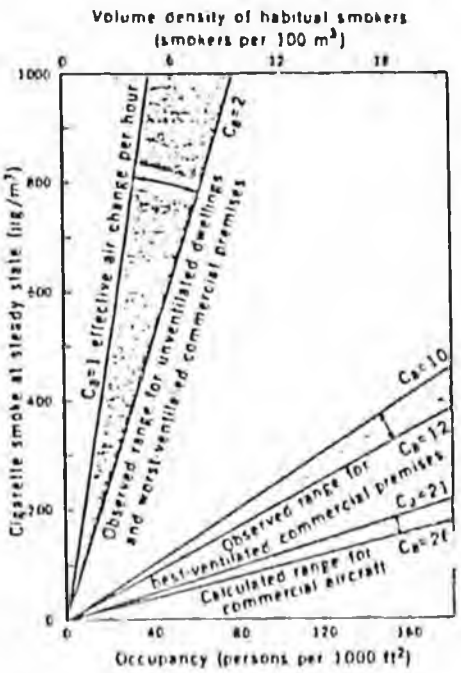

Fig. 4. Theoretical steady-state density of respirable particulates from environmental cigarette smoke in habitable indoor spaces, as related to the design occupancy P_s. On the average, one-third of adults are habitual smokers; for every three such smokers, we calculate that an average of one cigarette burns constantly throughout a 16-hour day. According to standard engineering criteria (*29*), occupancy and the type of microenvironment determine the design rate of mechanical ventilation C_v. The effective air change rate (C_s) for the removal of tobacco aerosol from room interiors is determined by C_v, by mixing, and by the rate of adsorption of tobacco particles on room surfaces. Generally C_v, and hence C_s, increase with P_s. (Typical P_s (in persons per 1000 square feet) ranges from 10 for office buildings to 70 for restaurants to 150 for bars, sports arenas, and aircraft (*29*, *32*).) We estimate the practical range of C_s to be from 1 to 12 air changes per hour. It appears that over the combined practical ranges of P_s and C_s, repeated exposure to tobacco smoke can lead to annual RSP burdens that violate the primary annual NAAQS.

RSP from cigarette smoke. The actual dose of RSP is clearly a function of personal activity patterns, differences in respiration rate also affect the dose. Many different scenarios can be imagined. In the following, we express a range of RSP burdens from the cigarette aerosol relative to a typical RSP ambient background level. For an air shed (air quality control region) that is in compliance with the annual secondary (public welfare) NAAQS for TSP of 60 $\mu g/m^3$, the RSP fraction of the ambient aerosol is conservatively estimated at 50 $\mu g/m^3$ and is likely to be composed largely of combustion-produced sulfates (*35*). Since the particle size distributions of this fraction and the cigarette aerosol are both in the respirable range, we first compare them on a mass basis, without regard for differences in the chemical composition of each.

Let A, B, C, and D be nonsmokers who dwell in the same air shed and who breathe at the average rate of 20 m^3/day. All have different occupations and lifestyles that lead, as we shall see, to dramatically different RSP burdens.

Nonsmoker A is a mailman who walks a regular route and is able to live in a completely tobacco smoke-free environment. He is exposed only to the background ambient and therefore inhales 365 mg of RSP annually.

Nonsmoker B is an office worker who works a 40-hour week 50 weeks per year in a 40-m^3 office with two other persons, one of whom is a habitual smoker. Replacing D_s in Eq. 4 with $D_{hs}/3$, we find that B's mass RSP exposure is more than three times that of A (we calculate an expected C_s of 1.1 for office buildings).

Nonsmoker C is a musician who entertains in a popular, poorly ventilated nightclub 8 hours nightly, 5 nights per week, 50 weeks per year. The average P_s in the club is 100 persons per 1000 square feet (about 33 smokers). Further, C shares a 100-m^3 apartment with a roommate who is a chain smoker. C is exposed to the roommate's smoke 5 hours per day, 7 days per week, annually. By using Eqs. 4 and 5 and a C_s of one air change per hour, we find that C's mass RSP burden is more than 15 times that of A.

An alternative way of approaching the excess RSP exposure is in terms of cigarette equivalents. The cigarette with the least tar in the May 1978 FTC scale has 0.55 mg of TPM. In these terms, B's excess RSP burden is equivalent to 5 cigarettes per day and C's burden to 27 cigarettes per day. However, this may un-

derestimate the true impact, since many nonsmokers have greater sensitivity to smoke than smokers (*7*).

Nonsmoker D is a flight attendant who spends 40 hours per week, 50 weeks per year on board a commercial airliner with a C_v of 23 air changes per hour. The average P_v on the plane is 150 persons per 1000 square feet. D's RSP burden is nearly twice that of A. Even with one of the best ventilation systems in use, the high density of smokers causes a substantial increase in mass RSP inhaled by D.

The following three considerations may help to place these scenarios into perspective. First, an annual exposure 1.5 times that of A is sufficient to exceed the primary annual NAAQS: the exposure of D, B, and C to RSP all violate the standard by factors of 1.2, 2, and 10, respectively. Second, pulmonary clearance studies show that the half-life of inert respirable particles (2.8 μm in MMD) in the lungs of nonsmokers is ~ 70 days (*36*): residence of RSP in the lungs is prolonged. Third, in a series of pulmonary lavage studies on 400 nonrandomly selected volunteers (250 nonsmokers and 150 smokers) (*37*), two of the nonsmokers had tarry lavage fluids with pigmented pulmonary alveolar macrophages strikingly similar to those found in smokers. In these two volunteers, the levels of aryl hydrocarbon hydroxylase, an inducible carcinogen-detoxifying pulmonary enzyme, were intermediate in value between the levels found in smokers and most nonsmokers. These findings were attributed to the effects of exposure to tobacco smoke (*38*).

Health Policy Implications

There is good toxicologic evidence that elevated levels of particulates in outdoor air, perhaps in combination with other pollutants, cause illness and death during air pollution episodes (particulate levels in excess of 1000 $\mu g/m^3$ per 24 hours). There is much epidemiologic evidence, some of it conflicting, that lower levels of particulates, perhaps in combination with other pollutants, affect respiratory health adversely when exposure to them is sustained (*39*). (This evidence has been used to establish the thresholds for harm on which the primary annual NAAQS for TSP is based.) There is excellent toxicologic evidence that mainstream cigarette smoke causes chronic obstructive pulmonary disease (*7*, *40*). Epidemiological evidence, some of it conflicting, indicates that exposure to tobacco smoke in the home affects respiratory health adversely (*7*, *41*). Finally, there is excellent evidence that mainstream cigarette smoke causes cancer in many organs (*7*). Sidestream smoke is chemically identical to mainstream smoke, and typically is more concentrated (*2*). Coke-oven emissions, which chemically are similar to tobacco smoke, are associated with increased rates of many forms of cancer in coke-oven workers (*42*). Animal studies demonstrate that the particulate phase of tobacco smoke contains numerous potent carcinogens and tumor promoters, initiators, and accelerators (*7*). One of these, benzo[*a*]pyrene, was detected at a concentration of 40 parts per million in ambient tobacco smoke (*13*). Strong evidence supports a correlation between the magnitude of long-term exposure to carcinogens and the incidence of cancer (*43*). Therefore, given the efforts by public health authorities to eliminate involuntary public exposure to saccharin and the fire retardant Tris—which have, respectively, one fifty-thousandth and one-tenth the experimental carcinogenic potency of benzo[*a*]pyrene alone (*44*, *45*)—similar efforts to prevent involuntary exposure to ambient tobacco smoke (*46*) appear justified.

Conclusions

We have defined the probable range of exposure of the nonsmoking public to a common pathological aerosol, cigarette smoke. We showed, both experimentally and theoretically, that under the practical range of ventilation conditions and building occupation densities, the RSP levels generated by smokers overwhelm the effects of ventilation and inflict significant air pollution burdens on the public. Our observations show that levels of RSP in places where tobacco is smoked greatly exceed levels found in smoke-free environments, outdoors, and vehicles on busy commuter highways. Our experimental results are consistent with the large differences in 24-hour average RSP levels reported for smoking and nonsmoking homes in the Harvard Six-City Study (*47*), with a survey of short-term RSP levels in commercial and public buildings in Houston (*28*), and with other studies of tobacco-generated TSP (*7*, *11*-*13*).

Attempts to reduce RSP levels from smoking by increasing the rate of mechanical ventilation or the efficiency of filtration yield exponentially diminishing returns for linear increases in ventilation energy (and cost). Moreover, efforts to conserve energy in buildings will decrease ventilation rates (*48*). Therefore, increased ventilation does not appear to be a solution to the problem. Indoor air is a resource whose quality should be maintained at a high level. Smoking indoors may be incompatible with this goal (*33*, *49*).

Further research is necessary to define the integrated particulate exposure of various segments of the population; compliance with the NAAQS, as indicated by the establishment of outdoor TSP sampling stations, does not imply protection of the public from excessive RSP burdens. Repeated exposure to ambient cigarette smoke imposes air pollution burdens on nonsmokers that exceed the primary annual NAAQS. It appears that the RSP burdens from ambient tobacco smoke are so large that they must be incorporated explicitly in future epidemiological assessments (*50*, *51*) of the relation between particulate levels and morbidity or mortality.

The Clean Air Act of 1970 and its amendments mandate the control of public exposure to outdoor TSP. However, little legislative attention has been devoted to the quality of indoor air—other than the passage of the Public Health Service Act of 1978, which provides for an ongoing study of the health costs of indoor air pollution. Clearly, indoor air pollution from tobacco smoke presents a serious risk to the health of nonsmokers. Since this risk is involuntary, it deserves as much attention as outdoor air pollution.

Note added in proof: A very recent epidemiological study concluded that long-term exposure to tobacco smoke, limited to the work environment only, is deleterious to the nonsmoker and significantly reduces small-airway function to the same extent as smoking one to ten cigarettes per day. This is consistent with scenario B (*52*). *ASHRAE Standard 62-73R*, a proposed standard for ventilation required for minimum acceptable indoor air quality, has been published (see *29*). Using data supplied in the standard, we calculate a C_v of ≤ 1.28 for office buildings where smoking is permitted.

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Nonsmokers and Cigarette Smoke: A Modified Perception of Risk

The article by Repace and Lowrey (*1*) is an interesting and useful aid for evaluating the exposure levels of nonsmokers to cigarette smoke. The article, however, is flawed because it compares the levels to those of smokers of cigarettes delivering the lowest amount of tar of any brand on the market. This cigarette delivered 0.55 mg of tar in 1978 and accounted for less than 2 percent of the cigarettes consumed that year and for less than 3 percent of the cigarettes sold in 1979 (*2*). The exposure of various nonsmokers was found to be the equivalent of smoking 2 to 27 such cigarettes per day. Twenty years ago, smoking as few as ten cigarettes daily was hazardous (*3*). Hence, the article implies that the health of exposed nonsmokers is seriously jeopardized.

The consequences of smoking on health have not been measured for consumers of the brand yielding the lowest tar, but rather for all smokers. In 1977 the average tar yield of American cigarettes, based on sales, was about 16 mg (*4*). Thus, "nonsmoker B" was exposed to the equivalent of one-sixth of the average 1977 cigarette per day, "nonsmoker C" to one per day, and "nonsmoker D" to one-sixteenth. It might be more pertinent to consider the tar delivery of cigarettes that were smoked about 20 years ago. The average cigarette in 1959 delivered about 29 mg in ten puffs. Thus, the model nonsmokers then would have been exposed to the equivalent of one-third to five *puffs* per day.

To be sure, we do not know that any level of cigarette smoke is harmless. The model nonsmokers were exposed to levels exceeding the primary annual National Ambient Air Quality Standards. Small amounts of smoke are irritating to many nonsmokers and may physically impair some. Such effects by themselves are sufficient cause for concern about passive exposure to cigarette smoke. Risk of cancer and other diseases for which dose is important should not, however, be imputed from comparisons of nonsmokers with the least affected 2 percent of the smoking population.

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Bock raises an important issue: How is the estimated range of exposure of nonsmokers to cigarette smoke translated into an increased risk of incurring the diseases of smoking? However, Bock both misinterprets our analysis and understates the risks. First, we did not derive an exposure-response relation between tobacco smoke and risk on the basis of low-tar cigarette equivalents inhaled by nonsmokers. Rather, we expressed the range of exposure in terms of such equivalents and confined our assessment of risk to the statement that such exposure represents a serious risk to the health of nonsmokers. We justified this not only by comparison with low-tar cigarettes, but also by references to outdoor air standards, pulmonary lavage experiments, coke-oven emissions, and carcinogenic potency. Second, we do not agree with Bock's assertion that comparisons with low-tar cigarettes are inappropriate because such cigarettes are smoked by the "least affected 2 percent of the smoking population." The latest report of the Surgeon General (*1*) advises that "there is no safe cigarette and no safe level of consumption" and that although lower yields of tar and nicotine reduce the risk of lung cancer and "to some extent improve the smoker's chance for longer life . . . it is not clear what reductions in risk may occur in the case of diseases other than lung cancer."

More to the point, Bock's comparisons of nonsmokers' exposures to those of inhaling smokers of high-tar cigarettes are misleading. As we have shown, the exposure of certain nonsmokers to tobacco smoke appears to be similar to the exposure of low-tar cigarette smokers. The cloud of pollution surrounding low-tar smokers appears to be not very different from the cloud surrounding high-tar smokers who are noninhalers. In fact, our low-tar cigarette produces sidestream emissions that are nearly 80 percent of those of the 1977 cigarette and nearly 40 percent of those of the 1959 cigarette (*2*). This is significant because a study begun in 1959 (*3*) has shown that 45- to 54-year-old male smokers who were noninhalers suffered a 41 percent higher mortality rate than male "nonsmokers" (*4*). A number of studies now indicate that carcinogenic, respiratory, and cardiovascular effects result from nonsmokers' exposure to indoor tobacco smoke (*5*).

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sponding to the average reflectivity is 11 ± 4, a moderately high value for a dry planetary surface at radar frequencies. For other parts of Venus where the reflectivity approaches 0.4, K calculated by assuming negligible conductivity and loss rises to 20.

How can these relatively high reflectivities be explained? From Eq. 2 we see that both the real and imaginary parts of ϵ contribute to the reflectivity. It seems extremely unlikely that dielectric losses (L) of naturally occurring dry rocks could be large enough to dominate the reflection mechanism; they normally control only the attenuation with depth (*9*). For the conductivity, S, to be important in reflection, not only must it exceed about 0.1 mho/m at our radar's operating frequency, but the conductive region must extend over an area with dimensions comparable to a wavelength or larger. If such large areas of conductive surface exist, it would seem likely that, at least occasionally, reflectivities near unity would be encountered, since occurrence of the precise frequency-dependent threshold value of S necessary to yield reflectivities consistently between 0.3 and 0.4 seems highly fortuitous.

We are left, then, with the necessity for explaining how the real part, K, of the dielectric constant can be raised to values between 11 and 20. The most likely mechanism is the presence in the rock of conducting inclusions much smaller than the observing wavelength. Meteorites containing relatively large amounts of free iron-nickel mixtures and sulfides display values of K ranging up to 100 or more (*9*). Free metals seem unlikely and in any case could not exist for long in any part of the Venus surface exposed to the atmosphere; highly conducting metallic sulfides would also be unstable to atmospheric exposure (*11*). But if overturning of the first few centimeters of surface proceeds slowly enough, or if material is being steadily stripped off the surface and blown elsewhere, atmospherically unstable minerals could be maintained sufficiently near the surface to be effective in raising the dielectric constant.

Nozette and Lewis (*12*) suggested that chemical erosion takes place at high elevations on Venus, where winds are comparatively strong and atmospheric densities and temperatures relatively low. The fine-grained eroded material is subsequently delivered to lower elevations, where it is chemically modified and possibly compacted. In the process, the putative conducting inclusions are transformed into gases and nonconducting minerals. If the "original" Venus surface contained on the order of 15 percent free metal—or, more likely, pyrite (FeS_2), which has been postulated independent of radar observations to explain the observed atmospheric chemical composition (*11*)—the high values of reflectivity seen at high elevations could be readily explained. Pyrite is one of very few minerals with the necessary high conductivity, having values between 1 and 10^5 mho/m (*8*).

The model that emerges from this discussion requires that the surface of Venus in the vicinity of the highly reflecting regions contain a significant amount of conducting mineral as inclusions in the rock. From a consideration of the present lower atmospheric composition, this material is likely to be pyrite and may be widespread in the original crustal rock, lying in radar view only at higher elevations, where new surface is constantly being exposed. It is also possible that pyrite is preferentially produced in sulfur-rich volcanic material.

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Cigarette Smoke Contains Anticoagulants Against Fibrin Aggregation and Factor XIIIa in Plasma

Abstract. *Gas-phase, water-soluble components of cigarette smoke cause delayed fibrin self-assembly and prevent fibrin cross-linking by inactivation of factor XIIIa (plasma transglutaminase). These anticoagulant properties of smoke are demonstrable in plasma, suggesting they play a role in the pathophysiology of smoking.*

There is little information on the effects of cigarette smoke on important biochemical interactions, though considerable information on its physiologic effects is available. Studies on interactions of cigarette smoke with certain proteins have provided biochemical evidence that specific functions of such proteins are inhibited (*1*, *2*) or augmented (*3*) by smoke components. In view of the shortened half-life of radioactively labeled fibrinogen in dogs exposed to cigarette smoke (*4*) and of the reported increase in fibrinogen in human smokers (*5*), we examined the possible effect of smoke on certain fibrinogen functions in vitro. We found that water-soluble smoke components include two types of anticoagulants: one is directed against fibrin self-assembly and the other inactivates factor XIIIa, thereby preventing cross-link formation (that is, stabilization) in fibrin clots.

Fibrinogen, a plasma glycoprotein, consists of three pairs of disulfide-bridged polypeptide chains termed Aα (molecular weight, ~ 70,000), Bβ (~ 60,000), and γ (~ 50,000). Cleavage by thrombin of arginyl-glycine peptide bonds at positions Aα16 and Bβ14 results in release of small polar peptides A and B from their respective chains: the resulting fibrin monomers polymerize noncovalently and form the fibrin gel.

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factor XIIIa which catalyzes the formation of ε-(γ-glutamyl)lysine cross-links between adjacent fibrin molecules. Certain other proteins are also cross-linked in this way to fibrin, for example, fibronectin (*6*) and fast-reacting α_2-plasmin inhibitor (*7*).

In this work, the effect of cigarette smoke on fibrin aggregation was investigated by the use of water-soluble, gas-phase components of smoke, obtained (*8*) by bubbling the smoke produced from one cigarette through 3 ml of buffer or distilled water. This extract (SE) was incorporated in varying dilutions in the buffer to which fibrin monomer solution (in 0.25 percent acetic acid) was added in order to initiate fibrin aggregation, which was monitored turbidimetrically at 350 nm (*9*). A dose-dependent delay in fibrin aggregation was observed (Fig. 1A). Increasing the amount of smoke extract resulted in decreased absorbance of the clot (1:10 and 1:5 dilution) and (1:5 dilution) delayed the onset of fibrin aggregation. The decreased absorbance (indicating a less opaque or more transparent clot) remained undiminished for several hours. These effects were partly decreased in dialyzed smoke solutions and could not be corrected by incorporating calcium chloride (20 m*M*) in the buffer (not shown).

The fibrin aggregation inhibitor was also examined by use of two differing fibrin preparations with α chains lacking COOH-terminal segments. One fraction, termed I-6 and isolated from plasma, lacked approximately one-fifth of the native peptide from most of its α chains [that is, its α chains were of similar size (*10*)]. Another fraction, termed I-9D, was obtained from an 88 percent coagulable plasmic digest of fibrinogen and lacked longer segments, approximately two-thirds of the native peptide, from most of its α chains (*11*). Neither preparation displayed a delay in fibrin aggregation in the presence of smoke extract (Fig. 1, B and C). In addition, the clot absorbance of I-6 was decreased, suggesting that aggregation was partially inhibited or that this effect was distinct from that of the delay in fibrin aggregation (Fig. 1A). These results implied that the fibrin aggregation inhibitor required the presence of intact α chains to exert its effect. What is more, they suggested that the inhibitor interacted either directly with the α chains of fibrin or with another fibrin site which lost its capacity to interact with the inhibitor following conformational changes induced by the loss of intact α chains.

We also determined whether the Smoke extract consistently prolonged the thrombin clotting times of plasma (Fig. 1D), but this effect could be abolished by decreasing the amount of extract added. In related analyses the clotting times of isolated fibrinogen fractions were examined in plasma containing the extract. To fresh plasma that had been depleted of its fibrinogen by heat precipitation, band I fibrinogen and thrombin and determined clotting times. Consistent with the results on fibrin aggregation (Fig. 1A), the smoke extract prolonged clotting times in samples containing band I fibrinogen, and this defect was not corrected by the presence of calcium chloride (Fig. 1E). Samples containing fraction I-6 fibrinogen exhibited no

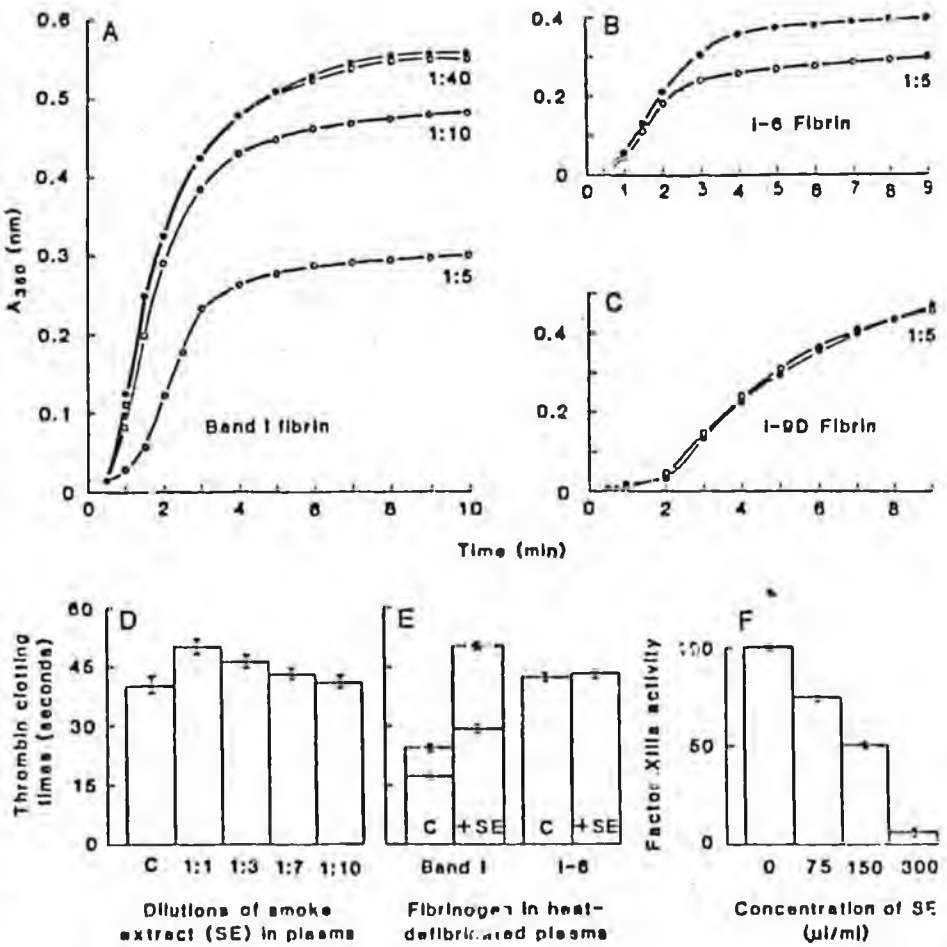

Fig. 1. Anticoagulant effects of smoke extract (*SE*) on fibrin and factor XIIIa. (A) Effect of different dilutions (open circles) on the time course of aggregation of (band I) fibrin which has intact α chains (*10*); closed circles indicate buffer control. After dilution of the fibrin solution with 20 volumes of buffer (0.025*M* tris-hydrochloride, *p*H 7.4, 0.135*M* NaCl) (*9*) containing SE, the final concentration of fibrin was 0.5 mg/ml. The *p*H remained constant during the experiments; in certain control experiments buffer containing SE alone or SE plus human albumin (0.5 mg/ml) in buffer was monitored for several hours and displayed no increase in absorbance (not shown). (B and C) Effect of SE on the aggregation of fibrin which lacks COOH-terminal segments from its α chains: fraction I-6 lacks approximately one-fifth (*10*) and fraction I-9D lacks approximately two-thirds (*11*) from their α chains. The final concentration was 0.4 mg/ml; the buffer was as in (A) and the SE dilution was 1:5. (D) The effects of SE on the clotting times of fresh human plasma (*9*). The height of each column reflects the mean and the brackets the range of at least three determinations in the presence of different dilutions of SE as shown; control plasma is designated *C*. Human thrombin (in 0.01*M* tris-hydrochloride, *p*H 7.4, and 0.15*M* sodium chloride) was added to citrated plasma (to 0.4 U/ml) and the clotting times were determined. (E) Histograms of clotting times for two differing fibrinogen preparations, showing that the inhibitory effect of SE on band I is not present when I-6 fibrinogen is clotted. The experiments were carried out in plasma that had previously been heat-treated (56°C, 10 minutes) to remove its own fibrinogen [which is heterogeneous (*10*)]. To this treated plasma, fibrinogen (either band I or I-6) was added (to 2 mg/ml) and clotting times were determined. Final thrombin concentration was 0.5 U/ml and SE dilution was 1:3 in all experiments (*C*, control). Columns with solid or interrupted lines indicate the presence and absence of calcium chloride (20 m*M*), respectively. (F) Effect of SE on the activity of isolated factor XIIIa, showing inhibition of [^{14}C]putrescine incorporation into casein. The conditions of the assay (*16*) as applied here were as follows: To 0.5 ml of 0.01*M* tris-acetate buffer, *p*H 7.4, containing calcium chloride (20 m*M*), dithiothreitol (5 m*M*), Hammerstein casein (1 percent), different concentrations of SE (as shown), and [^{14}C]putrescine (0.1 μCi/μmole), we added 5 μg of XIIIa. The mixture was incubated for 60 minutes at 37°C, and the reaction was stopped by adding 4 ml of 7 percent cold trichloroacetic acid. The precipitate was assayed by liquid scintillation (*16*).

change in their clotting times in the presence of the extract (Fig. 1E), again in agreement with the fibrin aggregation results. These findings indicated that smoke extract inhibits fibrin aggregation in the plasma environment. Moreover, the similarity of its dose-response whether plasma or isolated fibrin was used implied that competitive interaction between the smoke inhibitor and nonfibrinogen plasma proteins was minimal.

We then examined factor XIIIa–catalyzed fibrin cross-linking in the presence of the smoke extract. Fresh whole blood or plasma fibrin clots are cross-linked and therefore insoluble in 5*M* urea. When such clots were obtained in the presence of smoke extract (1:3 dilution) they were soluble in 5*M* urea (not shown). Similarly soluble were clots obtained from isolated fibrinogen and factor XIIIa in the presence of the extract. Cross-linking inhibition was also demonstrated by subjecting reduced fibrin samples to polyacrylamide gel electrophoresis in the presence of sodium dodecyl sulfate (*12*). In this analysis cross-linking can be shown by the characteristic depletion of α and γ chains and the concurrent appearance of more cathodal electrophoretic bands, termed α-polymers and γ-γ dimer, respectively (Fig. 2, gel 1). The presence of smoke extract resulted in a concentration-dependent inhibition of α-polymer and γ-γ dimer formation (Fig. 2, gels 2 and 3). Moreover, higher concentrations of the extract were required to inhibit the [more rapid (*13*)] formation of γ-γ dimer than that of α-polymers.

The possibility that this inhibitor was directed against (or binding to) fibrin cross-linking sites was examined. Non-cross-linked fibrin clots (*14*) exposed to smoke extract, followed by extensive washing and subsequent exposure to XIIIa, resulted in unimpaired fibrin cross-linking, indicating that the inhibitor did not bind to fibrin. In addition, dialysis removed the cross-linking inhibitor from the smoke extract (Fig. 2, gel 4), and its activity remained in freeze-dried samples.

Factor XIIIa–catalyzed incorporation of monodansyl cadaverine (fluorescence) (*15*) and of ^{14}C-labeled putrescine (*16*) into casein was used to assess the smoke inhibitor effect on XIIIa. These amines compete with the ε-amino group of peptide-bound lysine and prevent its cross-linking to peptide-bound γ-glutamine; thus measurement of casein-bound fluorescence or radioactivity serves to assay XIIIa by use of substrates other than fibrin. Both assays disclosed irreversible inhibition of XIIIa which depended on the concentration of smoke extract and required calcium chloride. Fig. 1F illustrates the results of the experiments with [^{14}C]putrescine. Direct inactivation of XIIIa was demonstrated after it was incubated with smoke extract. That is, the loss of XIIIa activity was proportional to the concentration of the smoke extract, and it could not be reversed by removal of the smoke inhibitor by dialysis or by gel filtration (G-25 Sephadex). By contrast, when the inactive zymogen (XIII) was subjected to the same treatment, it displayed no loss of activity following dialysis and activation to XIIIa.

Thus cigarette smoke contains two distinct coagulation inhibitors: one prolongs the clotting times of plasma by inducing delayed fibrin aggregation and requires the COOH-terminal region of fibrin α chains to exert its effect; the other inactivates XIIIa, preventing the cross-linking of fibrin polymers. By extension, this second smoke inhibitor may similarly affect other physiologically important proteins (*6*, *7*) also known to be cross-linked to fibrin by XIIIa.

We believe that these results permit speculation on potential pathophysiologic effects that may result from the exposure of lungs or other tissues to cigarette smoke. Diminished fibrin cross-linking could result in premature removal of fibrin, by proteolytic enzymes, thereby impairing the role of fibrin in initiating the provisional or temporary extracellular matrix during normal tissue repair. Besides protective fibrin-to-fibrin cross-links, other proteins [such as fibronectin (*6*) and α_2-plasmin inhibitor (*7*)] are cross-linked by XIIIa to fibrin, the latter conferring additional resistance to proteolysis (*7*). The presence of the smoke inhibitor is therefore likely to result in an initial wound matrix lacking the protective effect of such cross-links and consequently susceptible to early degradation. Blood complement abnormalities, suggesting ongoing low-grade inflammation in cigarette smokers (*17*), are consistent with this possibility, and the elevated fibrinogen levels (*5*) as well as the shortened half-life of radioactively labeled fibrogen (*4*) associated with smoking can be explained as a response to such an inflammatory process. What is more, that fibrin(ogen) degradation fragments induce a leukocyte-mediated increase in fibrinogen synthesis in cultured hepatocytes (*18*) is also consistent with this speculation.

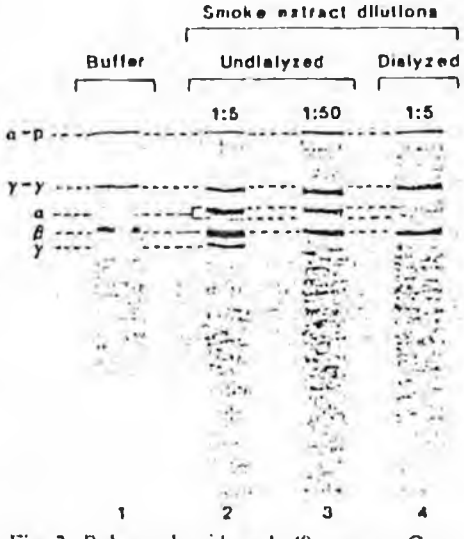

Fig. 2. Polyacrylamide gels (9 percent, Coomassie stained) of cross-linked fibrin clots (*9*) which had been washed and reduced prior to electrophoresis (*12*), showing non-cross-linked α-polymer (gels 2 and 3) and γ chains (gel 2) in clots obtained in the presence of smoke extract (SE). The absence of γ chains in gel 3 reflects the higher rate of γ-γ dimer (than α-polymer) formation (*13*) and indicates that the rate of XIIIa inactivation by the 1:50 SE dilution was lower than that of γ-γ dimer formation. The cross-linking mixture (*9*) contained dialyzed (gel 4) or undialyzed (gels 2 and 3) SE, isolated (band 1) fibrinogen, and factor XIII.

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2 April 1982; revised 28 May 1982

Metabolism of 2,4',5-Trichlorobiphenyl by the Mercapturic Acid Pathway

Abstract. *Carbon-14–labeled 2,4',5-trichlorobiphenyl was found to be metabolized by the mercapturic acid pathway to metabolites that are excreted in bile. About 57 percent of the carbon-14 was excreted in the bile; 30 to 35 percent was present as mercapturic acid pathway metabolites. Mercapturic acid was also isolated from the urine (0.3 percent of the dose).*

Although polychlorinated biphenyls (PCB's) have not been shown to be metabolized by the mercapturic acid pathway (MAP), there is evidence that the MAP may be involved. Biphenyl and 2,2',5,5'-tetrachlorobiphenyl are metabolized to dihydrodiols (*1*, *2*), and the NIH shift occurs in the metabolism of 4-chloro- and 4,4'-dichlorobiphenyl (*3*, *4*). Both of these metabolic routes usually indicate that an arene oxide precursor was formed, and compounds that form arene oxides are often metabolized in part by conjugation with glutathione, that is, by the MAP. Also, biphenyl is known to be metabolized by the MAP (*5*).

The most common indication that a xenobiotic was metabolized by the MAP is the isolation of the appropriate mercapturic acid from the excreta; however, this may also be indicated by formation of metabolites that contain metabolically introduced thiol, *S*-glucuronyl, methylthio, methylsulfinyl, or methylsulfonyl groups (*6–8*). Several chlorinated biphenyls were found to be excreted by mice as metabolites containing methylthio and methylsulfonyl groups (*9*), and chlorinated biphenyl methyl sulfones were also isolated from various tissues (*10–12*) and from milk from a lactating female (*13*). The radioactivity from intraperitoneally administered [^{35}S]cysteine was incorporated into 2,4',5-trichlorobiphenyl (triCB) methyl sulfones that accumulated in the lungs of mice given oral doses of triCB (*14*).

The evidence cited above indicated that some chlorinated biphenyls are metabolized by the MAP and that the common products of this pathway (the corresponding mercapturic acid and its precursors) are metabolized further before excretion. The mechanism was thought to be similar to that described for pentachlorothioanisole, where the biliary MAP metabolites were excreted mainly in the feces as bis-(methylthio)tetrachlorobenzene and nonextractable residues (*15*) and about 1 percent of the dose was present in the urine as *N*-acetyl-*S*-(methylthiotetrachlorophenyl)cysteine. The excretion of triCB methyl sulfide and methyl sulfone in feces from mice given triCB (*9*) prompted a search for MAP metabolites in bile from rats given ^{14}C-labeled triCB. In addition, triCB is a significant component of technical PCB containing 42 to 48 percent chlorine.

Bile collected from four bile duct–cannulated rats given single oral doses of ^{14}C-labeled triCB (*16*) (4 mg, 3.94 μCi per rat) contained 52.7 ± 19.2 percent of the dose after 48 hours, and 84 to 90 percent of the radioactivity was extracted from the bile (*17*). The radioactivity in the extract was separated into six fractions by reversed-phase high-performance liquid chromatography (HPLC) (*18*). The fractions were examined for possible MAP metabolites by converting the xenobiotic moieties to the corresponding triCB-*S*-acetates (*19*). Fractions 4 and 5, which contained 4.5 and 33.5 percent of the biliary ^{14}C, respectively, yielded significant quantities of triCB-*S*-acetates. Small quantities were obtained from fractions 1, 2, and 3. Two isomeric triCB-*S*-acetates were separated by gas chromatography (*20*) and found to have retention times and mass spectra identical with those of authentic triCB-3-*S*-acetate and triCB-4-*S*-acetate (*21*). After derivatization (*22*) of fraction 4, the derivatized triCB-*S*-cysteinylglycine and -cysteine conjugates were isolated by HPLC. After derivatization of fraction 5, the methyl ester of triCB-*S*-(*N*-acetyl)cysteine was isolated by HPLC. From the mass spectral data (*23*), structures were assigned to these derivatives and to the underivatized mercapturic acid as outlined in Fig. 1 (*21*).

About 30 to 35 percent of the radioactivity in the bile was present as MAP metabolites, showing that the MAP is a major metabolic pathway for this chlorinated biphenyl and that significant quantities of the metabolites are available for further metabolism by intestinal enzyme systems.

The fate of biliary triCB MAP metabolites in the intestine could not be deduced from the identities of the metabolites reported previously (*24*); therefore, the metabolic fate of ^{14}C-labeled triCB in

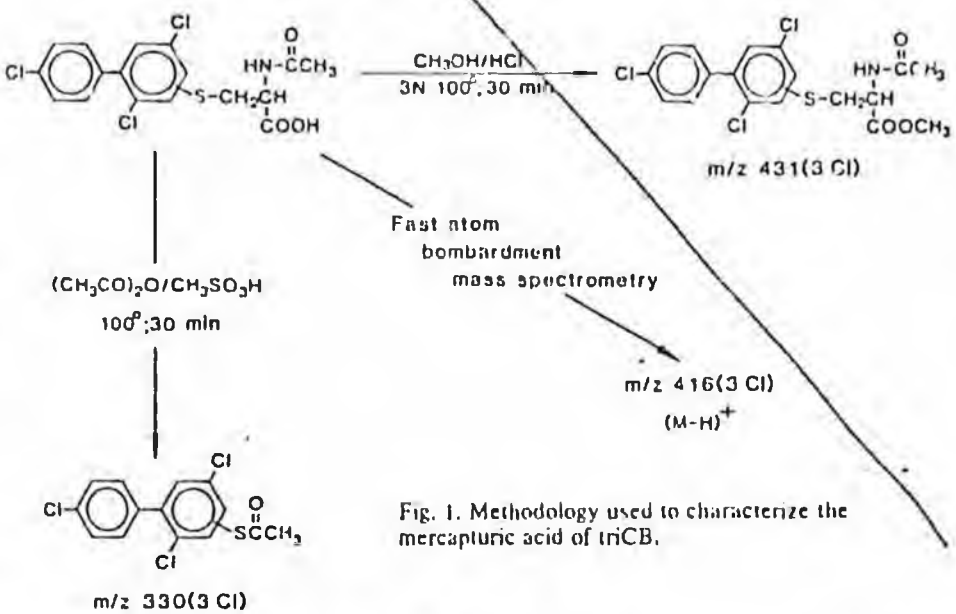

Fig. 1. Methodology used to characterize the mercapturic acid of triCB.

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Anchorage Daily News - 22 Oct 82

Non-smoker gets disability pay

The Associated Press

SAN FRANCISCO — A non-smoking federal worker transferred to an office with several smokers is entitled to about $20,000 in disability pay because she developed breathing difficulties, a federal appeals court ruled Thursday.

Irene Parodi cannot "perform her job due to its location in a smoke-filled office," the 9th U.S. Circuit Court of Appeals wrote in what the woman's lawyer called a landmark ruling.

"Unlike a person with a physical limitation, a person with an enviromental limitation can physically perform the assigned work in a proper environment," the court said.

"She is as disabled for her job at her assigned worksite as she would be had she actually suffered permanent and severe chronic bronchitis or another physically disabling disease," the court said.

The court said that unless Parodi is offered a job in a smoke-free office within 60 days, she will become eligible for disability retirement benefits of about $500 a month.

Mike Copies for Committee.

The Menace of Indoor Pollution

As we insulate, weatherstrip and seal our homes to conserve energy, we are also sealing in pollutants that are odorless, invisible and sometimes highly dangerous

By Lowell Ponte

On a brisk autumn day in 1979, James L. Repace, a policy analyst for the Environmental Protection Agency (EPA), used himself as a guinea pig. He carried a sophisticated air-monitoring device with him throughout the day—at home and at work. It was at his side as he drove through rush-hour traffic in smoggy Washington, D.C. Like him, it took in gusts of bus exhaust as he lugged it along city streets. To Repace's surprise, the monitor revealed that he suffered the most severe air pollution while waiting for his dinner to cook in his own kitchen.

Many scientists warn that pollution *inside* our homes and offices frequently reaches concentrations that would be illegal out-of-doors. Since the typical American spends 90 percent of each day indoors, pollution here is clearly a serious problem. And it is aggravated by the well-motivated efforts of energy-conscious consumers to insulate, weatherstrip and otherwise seal living areas to reduce fuel costs.

Unfortunately there have been few, if any, efforts on the part of either government or environmentalists to publicize the problem or to warn citizens of possible danger. The information presented here is published to acquaint readers with the facts so that they can take individual measures to reduce the risks.

Gas appliances. Most Americans live today in homes with fires burning night and day—the tiny blue pilot lights in furnaces, water heaters, clothes dryers, stoves and ovens fueled by natural gas. Gas burns far cleaner than the coal and wood stoves it replaced; it almost never pours visible smoke into homes. But where there's fire, there's smoke. Scientists are finding that some combustion products emitted by gas appliances may be more hazardous than we suspected.

Researchers at California's Lawrence Berkeley Laboratory measured the pollution output of gas stoves cooking at 350° F. and found that in kitchens with poor ventilation, the room's air could soon contain as much carbon-monoxide and nitrogen-dioxide gas as the Los Angeles sky on a smoggy day.

Carbon monoxide can be deadly. Just before last Halloween, a Los Angeles father turned on his home gas heater, apparently neglecting to check its vent. Days later he and his two daughters were found dead, asphyxiated by carbon monoxide. Each year in the United States, roughly 1700 people die in similar accidents.

When carbon monoxide enters our lungs, it binds to the red blood cells' hemoglobin 210 times more strongly than oxygen does. Thus even a small amount of carbon monoxide impairs our blood's ability to carry oxygen.

The EPA has set a standard of nine "parts per million" (ppm)—nine molecules of carbon monoxide mixed with every million molecules of air—as the maximum safe exposure to carbon monoxide during an eight-hour period. Victims of cardiovascular disease can suffer the pains of angina at this level. When carbon monoxide reaches 15 ppm, some subjects experience ominous changes in heart rhythm. At 30 ppm some will suffer distortions in vision and physical dexterity that impair driving.

Yet a study by the National Academy of Sciences reports that the air in home kitchens over short periods of up to one hour can reach up to 50 ppm carbon monoxide "as a result of ordinary use of a gas range, especially when the cooking utensils divert or quench the flame."

Gas flames also create oxides of nitrogen, including nitrous oxide (laughing gas) and nitrogen dioxide. The EPA's permissible annual pollution average for nitrogen dioxide in the air is only 0.05 ppm. At 0.1 ppm, it will cause breathing difficulties. At 0.5 ppm, this gas has been shown to lead to a greater susceptibility to disease in animals.

Some scientists suspect that nitrogen dioxide is the prime cause of increased respiratory illness among people in homes with gas stoves. Dr. Frank E. Speizer of Harvard University Medical School studied 8000 children, ages six to nine, half of whom lived in homes where parents cooked with gas and half where electricity was used. He found evidence that children from homes using gas stoves suffered up to 15 percent more respiratory illness than did the other group.

Kerosene heaters. In 1982 Americans bought more than 5 million portable heaters fueled by kerosene. Used to warm single rooms, the heaters vent the chemical by-products of their flame directly into the surrounding air. Test results published by *Consumer Reports* last October revealed that in a small room kerosene heaters put an average of 13 ppm of carbon monoxide into the air, and as much as 1.8 ppm of nitrogen dioxide (many times the EPA permissible level). A kerosene heater could also produce up to 12 ppm of sulfur dioxide in room

air—as much as 85 times the EPA health standard for outdoors. At even one ppm of sulfur dioxide, mild asthmatics can suffer breathing problems within 10 minutes, and at 5 ppm they can experience asthma attacks.

Home fireplaces. Many Americans have reverted to fireplaces for winter warmth. But fireplace chimneys are often poorly vented, and the fires built in them can raise carbon-monoxide levels in the home and produce exotic gases such as benzo[*a*]pyrene, a known cancer-causing chemical. Family members often toss into a fireplace anything that will burn, without knowing that the colored inks of the Sunday funnies, for example, can put a trace of arsenic vapor into the air. Other objects such as wood treated with preservatives can emit toxic fumes.

Insulation. In the 1970s more than half a million Americans fought the energy crunch by insulating their homes with urea-formaldehyde foam. Formaldehyde vapors in concentrations as high as 0.5 ppm or more have since been measured. At as little as 0.1 ppm of formaldehyde, some persons are hit by throat and upper-lung irritation, and at 0.25 ppm asthmatics and children face a serious health risk.

Fire-resistant asbestos, another widely used insulating material, is now recognized as a serious threat to health. Its fiber dust is easily inhaled into the lungs, where its crystals will cause development of fibrous tissue. Now banned in industry, fraying asbestos insulation continues to pollute the air in schools where an estimated three million American children attend classes. Replacing asbestos-backed vinyl tile floor covering can also be a hazard: if the tile is ripped up incorrectly, asbestos dust is released into the air.

Air-conditioning systems. These are sources of living air pollution—germs, fungi and amoeba. The bacterial infection that is known as Legionnaire's disease infected its victims in Philadelphia in 1976 and in other places since then by spawning in air-conditioning systems.

Air-conditioning systems and humidifiers often spread lung ailments through office buildings—especially modern, energy-efficient ones that have windows that can't be opened. These white-collar enclosures also absorb pollution from underground garages.

While factory workers have government agencies regulating pollution in their work place, most white-collar workers have not received such attention. Yet even photocopiers give off a common smog ingredient, ozone, along with other chemicals. Typically these machines are put in backroom offices with little ventilation.

Aerosol sprays. When we disperse insecticides indoors with spray cans, tiny droplets can remain in the air for a few hours, after which the chemicals will settle into the carpet and furniture. Each time an

infant crawls across a rug, subtle clouds of hazardous dust may waft up. Hair and deodorant sprays, oven cleaners and other aerosols also create clouds of chemicals that can be inhaled and ingested into the bloodstream.

Tobacco smoke. While smoking one cigarette, a person inhales more than 3600 different chemicals, including carbon monoxide, formaldehyde and nitrogen dioxide. Young children in homes of smokers suffer significantly higher rates of respiratory illness and receive hospital treatment more often than other children.

In offices where people smoke, scientists have measured levels of inhalable particles many times more concentrated than a factory would be allowed to put into the surrounding outside air in a 24-hour period. The heavy smoker also poses a special hazard in closed automobiles, where airspace is so limited that carbon-monoxide levels can quickly approach 35 ppm, an amount that could affect the driver's ability by distorting his hearing, perception and motor skill.

Radiation. The soil and common building materials such as granite, brick, cement and concrete can be naturally rich in radioactive radon and its by-products polonium 218, lead 214 and bismuth 214. Ironically these are the preferred construction materials for energy-efficient passive solar homes. What has alarmed scientists is that the houses act like closed jars, trapping natural radon gas and causing it to build up.

In Mount Airy, Md., the National Association of Homebuilders created a prototype for the ideal energy-efficient home of the future. Here the old-fashioned, drafty American house was replaced by a dwelling so airtight it exchanged air with the outdoors only about twice in 24 hours.

Trouble was, Mount Airy happens to have a concentration of radon gas, which was producing radioactivity inside the prototype at levels several times higher than might be found inside an ordinary home—levels eight times higher than those deemed safe by government guidelines.

In 1981 an editorial in the *Journal of the American Medical Association* called for "a temporary halt to our home-energy conservation programs" until we knew more about the risks of indoor air pollution. One of the facets needing study is how *mixtures* of pollutants can combine to become more dangerous. Those who smoke and are exposed simultaneously to either radiation or asbestos dust, for example, are apparently at greater risk than separate exposure could cause.

But just when we need to know more about synergistic effects and other unknown dangers, the federal government has cut by more than half its meager research budget for indoor pollution. Until more is known, it is simple prudence for individual Americans to take what-

ever steps they can to protect themselves from the already proven dangers. What can *you* do to reduce indoor pollution?

• Be alert when using a fireplace, kerosene heater or gas stove. A poorly adjusted gas stove can give off carbon monoxide at a greater rate than a properly adjusted one. One sign of a gas range needing adjustment: the tip of the flame is yellow instead of blue. Have a service man correct it.

Ideally you should vent stoves and ovens *outside*, preferably with an exhaust fan that switches on automatically whenever the appliance is in use. If you use a kerosene heater, set it inside your fireplace and let the chimney vent its combustion gases.

• Smokers should confine their air pollution to one room of the house and make sure the window is open. Better still, they shouldn't smoke inside at all.

• If there is a risk of radon gas in your area (a special threat in basements), consider sealing off any leaks or cracks in the foundation of your house. Basement drains are also a potential entry point.

• Consider using an "indoor-outdoor heat exchanger." These devices, which cost from $300 to $1000 and can be installed like a window air conditioner, use the heat from air that they pump *out* of your home to warm the fresh outdoor air that is simultaneously pumped in. They bring in fresh air on a cold winter day while conserving up to 70 percent of a home's costly heat. They can also be installed with filters to remove smoke and other pollutants.

Our home is our castle, but in trying to make it a perfect environment, we've filled it with some dangerous conveniences and made it a bit too snug to breathe in safely. Short of new gadgetry to clear inside air, good ventilation is the quickest remedy for indoor pollution.

For information on reprints of this article, see page 214

Flight Paths

At the Nashville airport I purchased a ticket to Dallas. The flight left from Gate 7, and I asked the ticket agent how to get there. "Take the escalator to the upper level and turn right," he said. "Gate seven is about halfway to Dallas."

—Contributed by Ronald Sanders

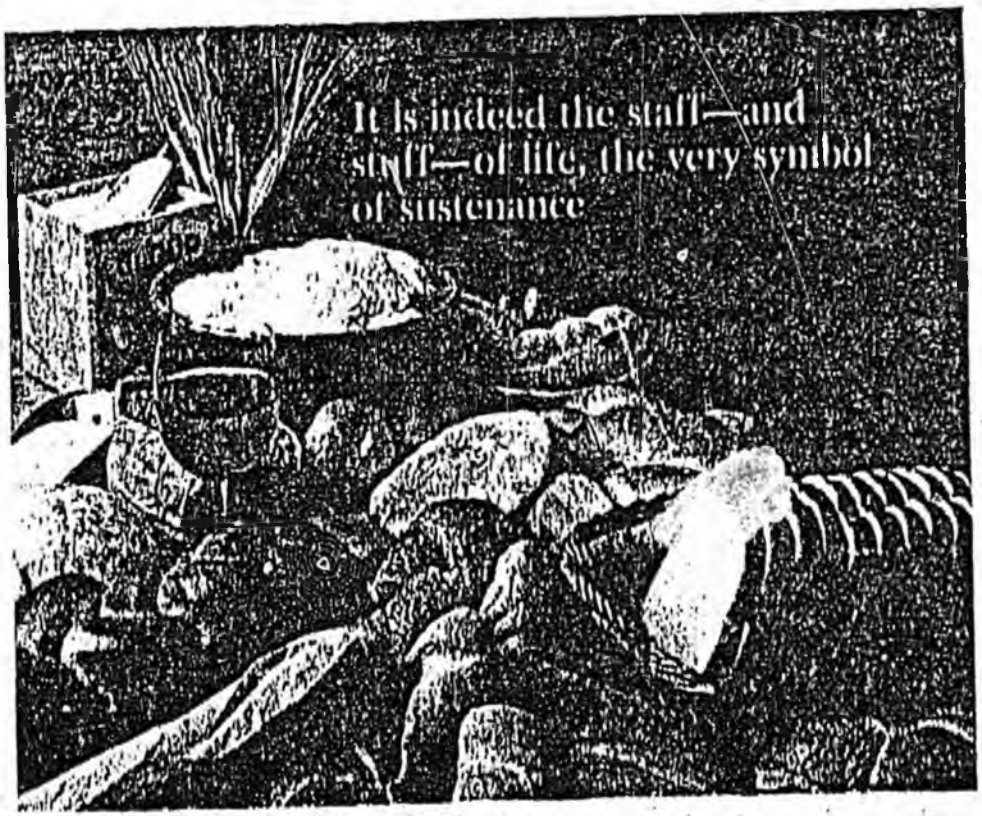
I was in an airport bar with a friend when we were approached by a slightly tipsy stranger. He asked my friend, who has an accent, where he was from. "Central America," my friend said.

"Really? So am I!" the man said in a booming voice.

"What part?" my friend asked.

"Kansas City."

—Contributed by Garry M. Schaeffer



Our Daily Bread

By Jack Denton Scott

The slightest whiff of baking bread starts taste buds blossoming. Its siren scent has even driven men to acts of madness: refused bread that he smelled baking in the little town of Byron, Mich., an enraged Indian launched one of the worst massacres in the state's history in 1827.

Like a country's flag, bread signals nationality at the world's tables: Mexico's tortilla; India's flat *chapati* and puffy *puri;* Scottish scones and oatcakes; England's light cottage loaves; Ireland's soda bread; the flat, pocketed Middle Eastern pita; America's corn bread.

And loaves appear in as many

PHOTO: IRENE STERN

Anti-smoking bill becomes hot issue

HB84

Times 3-18-83

Associated Press

Juneau — A representative who puffed on a cigarette during hearings regarding new smoking regulations apparently was not blowing smoke into the face of the law.

Rep. Ramona Barnes, R-Anchorage, says she was not violating the state's new smoking regulations during a recent House Judiciary Committee meeting, as was charged in a complaint filed by the Alaska Department of Environmental Conservation.

Roberta Banko of Juneau filed a complaint after seeing Barnes smoking during the committee hearing on a bill to prohibit smoking in a variety of public places. The bill being considered would increase the number of public places in which smoking is banned.

Barnes was cited for violating a year-old Alaska statute prohibiting smoking "in a room, chamber, place of meeting or public assembly under auspices of the state, or a department or agency of the state while public meeting held under the auspices of the state, or a department or agency of the state, is in progress." The penalty, if convicted, is $15.

The House majority leader said she was smoking in an approved smoking area. She also said that others were smoking during the meeting.

Barnes denied Banko's charge that she (Barnes) "was being very theatrical — obviously very arrogantly smoking in the meeting. It was apparent to everyone there that she was making a statement about her distain for the proceedings," said the woman, an active supporter of smoking regulations.

Barnes said, "I smoke all the time."

Banko said she was upset that Barnes was smoking in the same room as several people who had come to testify about how smoking aggravated their health problems.

Barnes said a smoke-eater was sitting next to her to prevent the smoke from bothering others.

HB 84

Smoke signals

IF ONE'S ACTIONS can be deemed a statement of that person's position on any given topic, Rep. Ramona Barnes can smoke to her heart's content at legislative committee hearings — and do so under constitutional immunity.

A Juneau woman filed a complaint with the Department of Environmental Affairs against Mrs. Barnes, accusing her of theatrically and arrogantly smoking a cigarette during a meeting of the House Judiciary Committee. At the time, the committee was considering a bill to ban smoking in public places.

BUT ALREADY on the books, as Roberta Banko protested in her complaint, is a year-old law that prohibits smoking "in a room, chamber, place of meeting or public assembly under auspices of the state, or a department or agency of the state while a public meeting held under the auspices of the state, or a department or agency of the state, is in progress."

Said the complaint by the Juneau woman, an active supporter of smoking regulations: "She was being very theatrical — obviously very arrogantly smoking in the meeting. It was apparent to everyone there that she was making a statement about her disdain for the proceedings."

AH, BUT there's the rub. By her own admission, Ms. Banko acknowledges that Rep. Barnes "was making a statement."

And the Alaska constitution specifically says: "Legislators may not be held to answer before any other tribunal for any statement made in the exercise of their legislative duties while the legislature is in session. Members attending, going to, or returning from legislative sessions are not subject to civil process and are privileged from arrest except for felony or breach of the peace."

For Rep. Barnes, apparently, politics still means a smoke-filled room now and then — notwithstanding any law to the contrary.

HB84

Smoking mad about lawsuits

He said. She said. They said. You said. And now I say I'm sick of the Alaska state government using my tax dollars for trivial lawsuits such as the one mentioned in your paper of March 15 regarding Rep. Ramona Barnes having a complaint filed against her by DEC for smoking in a ". . . room, chamber, place of meeting . . . under auspices of the state . . ."

How much is this nonsense going to cost the taxpayer? Wouldn't it have been simpler to ask her to please stop smoking rather than letting it slide so the state could secure solid "evidence" that she abused the law? Being as Juneau is all state government, why not just stop selling cigarettes altogether. That way there would be no chance of abusing the law.

With the ridiculousness that is going on in Juneau these days most of us are wonderng what is really being smoked!

— *Laura M. Schafer*

4/13/83

Letters

Smoking

Dear Editor:

House Bill 84 "An act relating to smoking in public places and vehicles," is not a bill just for the health conscious person who is into aerobic exercise, jogging, etc. It is more than a statement by the 2/3 population, which does not smoke, to the 1/3 that does, to give us a place to breathe smoke-free air. The bill: addresses the health hazards of second hand smoke, acknowledges the recent court decision concerning rights of nonsmokers, and takes note of the cost of smoking to the public.

It has been estimated the State of Alaska pays increased expenses for its smoking employees in excess of three million dollars annually. Alaska also pays workers compensation to people who lose work time because of smoke conditions making them too ill to work.

Some private industry is addressing the smoking problem now. IBM, 3M, and AT&T have set up separate areas for their nonsmoking employees. Some employers have set up separate areas because of court, decisions.

In 1976, a New Jersey court ruled against New Jersey Bell Telephone Co. stating that companies have a duty to give workers a smoke free work area if they want it. In August 1982, Lanny L. Vickers vs. the Veterans Administration, the United States District Court, Western District of Washington, ruled that a person who is physically sensitive to smoke qualifies as handicapped person as defined in 29 U.S.C. 706(7) (B). In October 1982, the United States Court of Appeals, ninth circuit, (Irene C. Parodi vs. Merit Protection Board) ruled a person is eligible for voluntary disability retirement benefits if that person is medically not able to perform the job because of sensitivity to smoke in the work place and the employer does not offer "... Suitable employment in a safe environment ..." Werner H. Peterke, a former Social Security administration worker, was awarded approximately $700 every two weeks in employee compensation payments on the grounds that he suffered from the smoke of fellow employees at work.

Medical research has linked ambient smoke to medical problems in nonsmokers. Children of parents who smoke are more prone to bronchial ailments. Nonsmoking wives of smokers have higher lung cancer rates than nonsmoking wives of nonsmokers.

House bill 84 represents an issue whose time has come. It has moved from arguments between smokers and nonsmokers to a bill before the legislature. It could very well go on to become law. If you have an interest, contact your legislator at Pouch V, Juneau, Alaska, 99811.

Gary Miller
P.O. Box 2436
Juneau, Alaska

Anchorage Daily News photo illustration by Jim Lavrakas

Stickers like this are available from the Alaska Lung Association, and obstetrician Lydia Eastburn is always pleased when she can give one to a patient who's kicked the habit.

Pregnant smokers are 'smoking for two'

By GWEN BARCUS
Daily News reporter

☐ "Warning: The surgeon general has determined that cigarette smoking is dangerous to your health."

For the expectant mother that precautionary notice on every package of cigarettes is doubly important. When she smokes she is smoking for two.

Dr. Lydia Eastburn, an Anchorage obstetrician and gynecologist, says about 40 percent of the pregnant women she sees are smokers.

"When I'm seeing a new patient, one of my first questions is 'Do you smoke?' The next is 'How much?' "

If the answer to the first question is yes, Eastburn says she goes on to explain why now is the time to quit. About one-fifth of her patients heed her advice, and probably an equal number cut down on the number of cigarettes they smoke.

Eastburn is trying to increase those numbers. "I know most mothers want to do everything possible to assure their baby's health," she says. "There are some things over which they have no control, but stopping smoking is one thing they can do, and it's very important in the development of the fetus."

The pregnant woman who smokes increases the risk of having a miscarriage — smokers are almost twice as likely to miscarry as women who don't smoke, according to Peter Fried and Harry Oxorn in their book "Smoking for Two."

Eastburn says another risk is that the baby will die during pregnancy, and low birth weight also appears to be closely related to smoking during pregnancy.

Although there may be several reasons why some newborns are smaller than average, virtually all scientific studies on the subject show a clear relationship between cigarette smoking and lower infant birth weight. And, according to Eastburn, smaller babies mean smaller heads and smaller heads mean smaller brains. She is convinced that the incidence of learning disabilities is higher in babies of smokers.

There are some 200 chemicals in cigarettes, many of which are harmful even for the adult smoker. For the unformed developing life, dependent on its mother for the oxygen and nourishment it needs to develop properly, Eastburn says the results of smoking by the mother can be tragic.

"A couple of years ago I had a patient whose fetus was not growing at the proper

See Page F-3, SMOKING

pared to her husband, arriving in Palmer in 1936 to work as a surgery nurse at the Matanuska Valley Hospital. It was a "far cry from Chicago," where she had taught school, "but I fell in love with the country — and also with Walter. We were married in Seward in 1938."

Walter had gone to Palmer in 1935 while the Matanuska Colonists were enroute there. Following his graduation from Washington State College, now Washington State University, he was working for the Alaska Rural Rehabilitation corporation.

Elsie recalls their honeymoon prospecting trip to Lake Chalatna — with a friend of Walter's. She refers to the trip as "Three on a Honeymoon." They flew into the remote area with a dismantled river boat which they planned to reassemble on arrival. The river ran in a narrow bed between high bluffs and the two men pulled Elsie upstream from the top of the

The Rondy King and Queen Regent will celebrate their 45th anniversary this year, and except for a year spent Outside when Elsie's mother was ill, and time spent in New York while Walter was earning his master's degree at Hunter College, the two have lived in Alaska all that time.

During World War II, Walter served as paymaster and timekeeper with the Army Transport Service based in Seward. He spent 15 years as a teacher at Seward High School, and when the family moved to Anchorage in 1966, taught history in evening classes at the University of Alaska, Anchorage.

Prior to his retirement five years ago, he was Command Historian for the U.S. Army Alaska, and still pursues his interest in history, particularly Alaska history.

Work for Elsie has included teaching, nursing and administration, and

Anchorage Daily News/Paul Brown

Walter and Elsie Blue were chosen by the Pioneers of Alaska to be King and Queen Regent of the 1983 Fur Rendezvous.

13 years operating "The Blue Shop," a women's and children's clothing store in Seward. She served as administrative assistant at Seward General Hospital and was administrator of the Careage House and Ridgeview Manor, both former nursing homes in Anchorage. She retired in 1975, and devotes much of her time to the activities of The Pioneers of Alaska Auxiliary 4. Vitally interested in the welfare of Alaska's long-time residents, she serves as a member of the State of Alaska Pioneer Homes advisory board.

Virginia, their only child, was born in Seward, and now lives in New York City where she is a writer. She was an original member of the Anchorage Community Theater.

When asked what sort of vehicle they would like to ride in during the Rondy parade Feb. 19, Walter Blue replied, his blue eyes twinkling, "a garbage truck."

"Our entire life in Alaska has been like this," Elsie laughs. "There's never been a dull moment."

Smoking and pregnancy don't mix — for you or your baby

Continued from Page F-1

rate," the doctor recalls. "I felt sure it was because she was a heavy smoker. When she went into labor she was rushed to the hospital and in her nervousness smoked almost a package of cigarettes on the way. Her baby was born dead."

The most active and powerful ingredient in cigarette smoke is nicotine. The amount of nicotine in a single cigarette, if injected rather than inhaled, would paralyze the brain centers controlling the heart and breathing and cause rapid death. In the case of a pregnant smoker, her nicotine-constricted veins narrow the blood vessels that supply the fetus, thus decreasing the amount of oxygen and "food" it receives. In addition, nicotine actually crosses the placenta and directly affects the fetal cardiovascular system.

Frier and Oxorn say "for every cigarette smoked by the mother, the fetus gets the equivalent effects of two."

Eastburn says another danger for the pregnant smoker becomes evident if the baby has to be delivered by cesarian section. The procedure requires a general anaesthetic, which presents a risk to both mother and baby if the mother is a heavy smoker, especially if smoking continues right up to the time of birth.

It helps to stop smoking even a week before the baby is born, says Eastburn. And the new mother should work hard to maintain her resolve, she adds, because the "involuntary smoker" — anyone breathing the air around the smoker — is exposed to even more of tobacco's harmful constituents than the smoker. The smoke from the burning end of a cigarette has *twice* as much nicotine as the inhaled smoke, and *five* times as much carbon monoxide.

According to the Alaska Lung Association, babies in the first year of life have higher rates of pneumonia and bronchitis if their parents smoke at home. And children of smoking parents are twice as likely to smoke as children of non-smokers.

The Lung Association distributes an informational packet for the prospective mother who smokes, and Eastburn gives one to each of her smoking patients. (They are also available from the association at 406 G St., telephone 272-2332. A $1 donation is requested.) She files their charts in blue folders so they're easily distinguishable, and everytime she sees a "blue folder" patient she encourages her to quit.

Other than quitting "cold turkey," what can the smoking mother-to-be do to lessen the danger to her baby?

"If she has tried to quit and has failed, she should at least cut down to no more than 10 cigarettes a day," says the doctor. "If lighting up a cigarette is important to her, perhaps she could light up, take one puff, then throw the cigarette away."

But it would be even better, Eastburn says, if she could say what is printed on a sticker distributed by the lung association: "I quit because I love my baby."

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Edited by
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SMOKING AND PREGNANCY

JONATHAN E. FIELDING, M.D., M.P.H.

In 1957 Simpson published her original finding that babies born to women who smoke during their pregnancy are on the average 200 g lighter than neonates born to nonsmokers.[1] More than 100 articles on this relation have led to general acceptance that, on the average, smokers' babies weigh 150 to 250 g less than nonsmokers' babies, and twice as many of the former weigh less than 2500 g. Differences in weight are in direct proportion to the number of cigarettes smoked and are independent of other infant and maternal factors known to influence birth weight.[2]

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Until recently, however, it had been difficult to explain why observed perinatal mortality ratios (smokers to nonsmokers) varied from lows of 1.01 and 1.03 in sample sizes of 11,700 and 9800[3-5] to highs of 1.28 and 1.40 in respective sample sizes of 15,800* and 12,500,†[6,7] with an intermediate value of 1.12 in a sample of 19,000.[8]

A series of articles analyzing data from the Ontario Perinatal Mortality Study of all single births in 10 Ontario teaching hospitals during 1960-61 indicates progress in untying this Gordian knot.[2,9,10] The study of 51,490 births, including 701 fetal deaths and 655 early neonatal deaths, supplemented usual clinical records with interviews of mothers in the hospital, interviews with anesthetists and attending physicians and autopsy records.[11] Results relate perinatal mortality to social, demographic and physical maternal factors, prenatal care, histories of prior pregnancies, complications of pregnancy, details of anesthesia, delivery and hospital course and infant survival up to eight days. The interviews of mothers included questions on the maximum amount smoked during pregnancy, expressed as packages per day. The study found that perinatal mortality was 27 per cent higher in infants of smokers as a group than in those of nonsmokers. Risk, however, varied greatly according to other factors, including maternal height, weight before pregnancy, hospital pay status (public or private), birthplace, age, parity, previous pregnancy history and sex of the child. When simultaneous adjustments for these variables had been made, smoking less than one package per day increased perinatal mortality risk by 20 per cent versus 35 per cent for smoking more than one package per day.

Controlling for the same variables, the authors found that the increased risk of both in utero deaths and neonatal deaths is concentrated in the period of 20 to 28 weeks' gestation. Increased neonatal mortality rates seem to result from the increased risk of very early delivery rather than lower survival at a particular gestational age. The authors estimate that 30 per cent of the excess perinatal death associated with maternal smoking is due to increases of 25 and 24 per cent respectively in placenta praevia and abruptio placentae for those who smoke less than one package per day and of 92 and 68 per cent for heavier smokers. Other studies have also found an average increase of 28 to 48 per cent in these conditions for smokers.[12-15]

Crucial to an understanding of risk is the differential risk of smoking based on maternal characteristics. The Ontario study found that the excess perinatal death risks of less than 10 per cent were associated with low parity, young age and normal hemoglobin. By contrast, older mothers of higher parity who were of public pay status or who suffered from anemia (hemoglobin less than 11 g per deciliter) had in-

*For neonatal deaths only.

†Approximate.

creased perinatal mortality risks of 70 to 100 per cent when they were heavy smokers.[2,11] The characteristics other than smoking that are associated with differential risk may be important in reconciling the varying risks to smokers observed in the large-scale studies. Differences in population selected for study, as well as different methods of adjustments to match smokers and nonsmokers, appear to have contributed to the wide variation in perinatal mortality ratios for smokers vis-à-vis nonsmokers.[4] In addition to the growing evidence for adverse late outcomes of pregnancy, a recent article in the *Journal* provided convincing demonstration that pregnant smokers have a significantly increased risk of spontaneous abortion, 1.8 to 1.0, when compared to nonsmokers.[16] Not yet fully explored are the combined risks of smoking and alcohol ingestion, although there is no reason to believe that this combination yields fewer adverse effects than the sum of those two behaviors. Also unresolved is whether the effects of maternal smoking on fetal growth and development carry over into childhood.

Although the risks recently reported need confirmation by others, the growing evidence linking maternal smoking to increased perinatal mortality argues strongly for augmented attempts to apprise women of childbearing years of the potential risk of smoking in the outcome of pregnancy. The Surgeon General's 1973 report on the Health Consequences of Smoking estimated 4600 perinatal deaths in the United States annually as a result of maternal smoking.[17] Unfortunately, despite a drop from 52 to 39 in the percentage of men over 21 who smoke, the percentage of women of childbearing age who smoke is growing.[18] A careful cohort study of teen-age girls found that the proportion of regular smokers among girls 15 to 16 years of age had risen from 9.6 per cent in 1968 to 20.2 per cent in 1974, and among girls 17 to 18, an increase from 18.6 to 25.9 per cent in the same period.[19]

Although too little is known about what motivates smokers to stop, some evidence suggests that pregnant women may be particularly susceptible to educational and motivational technics aimed at cessation of smoking. Several studies indicate that most women are sufficiently concerned about the outcome of pregnancy to adhere to medical advice regarding helpful and harmful activities.[20-22] One survey showed that 62 per cent of young women smokers believed that smoking can harm an unborn child; most reported that they either had cut back (32 per cent) or had stopped smoking (35 per cent) during pregnancy.[23] The frequency of contact between pregnant women and health professionals provides an unusual opportunity for education about the dangers of smoking and positive reinforcement for reduction or elimination of this habit.

The Massachusetts Department of Public Health has made the following recommendations to help reduce smoking among pregnant women:

1. In the initial prenatal visit, physicians and nurses should include cigarette smoke in the usual list of drugs that are known to affect adversely the outcome of pregnancy and are to be avoided. Mention may be made that during pregnancy, when use of all pharmacologic agents is to be kept to a minimum, cigarette smoke, which contains over 1000 "drugs," is of special concern.

2. Pregnant women should be told that evidence to date suggests that they are at especially high risk for detrimental effects of smoking on the pregnancy if they have had a history of previous perinatal loss, bleeding or placental complications, if they are anemic or if they are in the older age group.[24]

3. Physicians should consider obtaining a carboxyhemoglobin or expired carbon monoxide level on every patient during the first prenatal visit. If this test is performed, the patient should be shown abnormal laboratory results and told that the high level, which poses a risk to the fetus, can be lowered to the normal range only by cessation of smoking.[25] The success of cessation efforts can be monitored by repeat testing at later prenatal visits. When levels remain elevated, physicians should reinforce their previous strong advice.

4. Any woman with bleeding at any time during pregnancy should be questioned closely on whether or not she smokes, and the dangers of smoking to the fetus re-emphasized.

5. Physicians should obtain a list of reputable local smoking cessation clinics and provide this information to pregnant patients who smoke, with the suggestion that these peer groups may help the patients stop smoking.

6. Health facilities and physician offices should not permit smoking in any areas where staff and patients come in contact. Prominent "No Smoking" signs should be displayed in all patient areas, and abstinence enforced.

7. Blue Cross and commercial insurers should consider inclusion of coverage for approved smoking cessation activities in all subscriber contracts. At a minimum, such activities for pregnant women should become a part of standard master medical coverage. Medicaid should also cover such programs. Insurers should develop reasonable criteria for vendors of these services that address cost, program content, personnel and standardized reporting on results. As an alternative, insurers could consider reinforcing information on the dangers of smoking generally by charging lower premiums to those individuals and families who would sign a certified affidavit that they are nonsmokers. (This approach has worked with life insurance.)

The Massachusetts Department of Public Health is recommending to the Federal Trade Commission that the warning on cigarette packages be revised to read as follows: "Cigarette smoking is hazardous to your health and can cause fatal cancer, heart disease and lung disease. Smoking during pregnancy increases the [illegible] risk of death of the un-

Abstract from Early Human Development

RANTAKALLIO, P., University of Oulu, Finland

"The effect of maternal smoking on birth weight and the subsequent health of the child." (Early Human Development 2/4: 371-82, 1978)

"The effect of maternal smoking during pregnancy on birth weight was studied in 12,068 births, the mothers in 1819 of which were regarded as smokers. The children of the smokers were compared with those of controls of similar age, parity, marital status and place of residence. Maternal smoking reduced birth weight in a dose-related manner. Birth weight was least affected among young, primiparous mothers who smoked only slightly, a difference which was, however, entirely caused by those mothers who stopped smoking for the last 3 mth of pregnancy, and this subgroup showed similar figures for postneonatal mortality and morbidity up to the age of 5 to those of their controls.

Postneonatal mortality was higher in the total group of the smokers than among their controls, the primiparous or young mothers not differing in this respect from the others. Morbidity up to the age of 5 was significantly higher ($P < 0.001$) among the children of the smokers, the children of the primiparas and young mothers being affected in a similar way to the others. The low birth weight infants of the smokers had a higher mean birth weight and lower perinatal mortality than the low birth weight infants of the controls, but morbidity up to age of 5 and postneonatal mortality were higher among the smokers in respect of both low birth weight infants and others."

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Current Comments®

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Nicotine Addiction Is a Major Medical Problem: Why So Much Government Inertia?

Number 31 July 30, 1979

Readers of *Current Contents*® are probably familiar with my feelings about the smoking habit.[1,2] I particularly dislike being forced to suffer the choking pollution so casually created by most smokers. On the other hand, I'm especially appreciative of smokers who understand my concern. Many other non-smokers feel the same way. It doesn't make sense to us that smokers maintain their habit in obvious disregard of its dangers and costs. Nevertheless, this is not without an understanding that the smoker has a need which can at best be delayed only temporarily.

The dangers and costs attributed to smoking are legion. Smoking is linked to a number of diseases: cardiovascular disease; cancers of the throat, lung, mouth, pancreas, and urinary bladder; peptic ulcers; decreased fertility; and increased still-births and spontaneous abortions.

Society loses the most from the smoker's habit. A few years ago the National Institute of Drug Abuse (NIDA) published an important volume on smoking behavior research. In this work, on which I will draw heavily below, Bryan Luce and Stuart Schweitzer (UCLA School of Public Health) state that smoking results in a major drain of the nation's economic resources in both direct health care costs and the costs associated with lost earnings due to sickness and death.[3] The NIDA estimates that smoking-related illnesses cost the American public nearly $8 billion in 1975. Lost earnings resulting from smoking-related illnesses and deaths totalled nearly $18 billion. By comparison the $170 million cost of smoking-caused fire damage seems small. But the National Fire Prevention and Control Administration says that smoking-caused fires account for 47% of all fire-related deaths and injuries.[4]

Most smokers will agree that their habit is dangerous and costly. The majority of them *want* to quit but few are successful. A 1967 survey of adult and adolescent smoking habits in Britain indicated that 77% of current smokers want to stop.[5] However, only one in five stops permanently.

Many non-smokers find it especially ironic and annoying that smokers often dislike the habit. There is a common view that smokers are simply weak people with little will-power. This is a distorted view of a *significant medical problem*. Smoking is more than just a bad habit that is socially and physically unacceptable to non-smokers. Smoking is a *powerful physical addiction*. I agree with M.A.H. Russell, senior lecturer and honorary consultant psychiatrist at Maudsley Hospital Addiction Control Unit, UK, who asserts, "Cigarette-smoking is probably the most addictive and dependence-producing form of... self-administered gratification known to man."[6] Unfortunately the evidence is

growing that the statement is now equally applicable to women.

I cringe when I recall a recurring scene from my youth. A beautiful young woman, dressed as a cheerleader, would stand on the corner outside my high school giving away sample packets of cigarettes. Her supply was inexhaustible: she was never empty-handed. She and her colleagues were the predecessors of the drug-pushers who now pervade the educational establishment. Whether she realized it or not, she was pushing the powerful addictive drug called nicotine.

Unfortunately, most people do not acknowledge this *addictive* aspect of smoking. In fact, the 1964 Surgeon General's Report alleged that smoking is not an addiction because "there are no withdrawal symptoms, no tolerance is developed, and no anti-social behavior is elicited."[7]

Not surprisingly, the Tobacco Institute in Washington, DC, the chief lobbying group for the tobacco industry, also maintains that smoking is not an addiction. Ann Browder, assistant to the president of the Institute, explains: "If nicotine is addictive, we wonder why 30 million people have given up the habit since 1964. Cigarette smoking is not addicting, as opposed to being habituating, in that an individual who began smoking a pack or a pack and a half a day doesn't have to increase that consumption in order to satisfy the desire to smoke. Our stand is certainly that it is habituating but not addictive."[8]

The distinction the Tobacco Institute draws between habituation and addiction has been called artificial by the World Health Organization (WHO). In its reports, WHO has replaced both terms with a single term: drug dependence.[9] Since the 1964 Surgeon General's Report was issued, there has been convincing research to show that at least one component of tobacco, nicotine, is a dependence-producing drug, as defined by WHO: "a drug having the capacity to produce a state of psychic or physical dependence, or both."[7]

WHO defines drug dependence as "a state, psychic and sometimes physical, resulting from the interaction between a living organism and a drug, characterized by behavioral and other responses that always include a *compulsion* to take the drug on a continuous or periodic basis in order to experience its psychic effects, and sometimes to avoid the *discomfort of its absence* [withdraw]. *Tolerance* may or may not be present."[9]

Tolerance is indicated when the addict becomes accustomed to the initial effects of the drug. Also, it usually involves a physical change of some kind in the addict. In its third report on smoking and health, the Royal College of Physicians stated that nicotine fits the definition of tolerance: "When inhaling cigarette smoke for the first time most people have symptoms such as palpitations, dizziness, sweating, nausea and vomiting.... If they continue to smoke, they acquire a tolerance to nicotine, and over a period of two or three years the smoking pattern usually changes so as to allow a high intake of nicotine.... The metabolic type of tolerance also occurs, in that smokers metabolize nicotine more efficiently than do nonsmokers."[10]

Withdrawal symptoms are those "which follow sudden withholding of a drug to which a person has become addicted."[11] (p. 1483) The regular smoker feels withdrawal symptoms an hour or two after his last cigarette. Stanley Schachter, professor of psychology at Columbia University, observes, "Restrained smokers appear to be chronically more irascible, to nibble more, and to have poorer concentration than unrestrained smokers."[12] Russell adds, "Such [withdrawal] symptoms as depression, anxiety, restlessness [and] intense craving...have frequently been described.... More recently, objective physical withdrawal effects have been clearly demonstrated and include sleep disturbance, sweating, gastrointestinal changes, drop in pulse rate and blood pressure, disturbed time perception..., impaired performance at simulated driving...and EEG changes."[5]

Jerome Jaffe, professor of psychiatry at Columbia, points out that society is unwilling to recognize these withdrawal symptoms as signs of illness. Jaffe reported, "Even where severe withdrawal phenomena do occur...society generally has taken the view that such signs and symptoms are 'normal' and to be expected under the circumstances. While tremulousness following abrupt withdrawal of alcohol, or autonomic disturbances from withdrawal of opiates are equally to be expected under the circumstances, they are, for some reason, not regarded as equally 'normal under the circumstances' and are viewed as representing signs of illness."[13] In short, alcohol addiction is now viewed as an illness (it wasn't always) but smoking has not yet reached that stage in the public's perception of the problem.

Smokers are compelled to smoke to avoid the disagreeable symptoms of withdrawal. Compulsion is "an irresistible impulse to perform some act contrary to one's better judgment or will."[11] (p. 332) Not only did 77% of the British smokers surveyed in 1967 want to quit but they also gave good reasons for quitting.[5] Smoking causes physical harm and discomfort, is expensive, and is increasingly disapproved of by other people. Since only one in five of those surveyed quit permanently it is clear that the majority of smokers act against their will and better judgment. For them, cigarette smoking is a compulsion.

I can't avoid mentioning here a certain woman Ph.D. who makes frequent TV and public appearances on behalf of the Tobacco Institute. She is one of those one in five who smokes out of free will. She is well paid to convince smokers that they should cling to their habit in spite of the statistical evidence that smoking is disease related. None of those statistics applies to them, she says. My contempt for such misuses of science is difficult to express briefly. While it may not matter what she says to today's smokers who can't quit even if they want to, it does matter that she convinces individuals who haven't started smoking that they may be exempt from the laws of probability.

Like other addictive compulsions, cigarette smoking leads to antisocial behavior when the available supply is restricted. Gwenda Blair, writing in *Mother Jones*, claims: "Data from Germany after World War II indicates that even under conditions of extreme deprivation, and in situations where food rations were under 1,000 calories a day, smokers still bartered eats for smokes.... Smokers' need for cigarettes was so overwhelming that some also... prostituted themselves or stole other goods that could be traded for cigarettes."[14]

It should be obvious that cigarette smoking is not a minor vice. Nicotine, "the most powerful pharmacological agent in cigarette smoke,"[15] is in the same chemical family as the poison strychnine, the medicine quinine, the hallucinogen mescaline, and the addictive pain relievers cocaine, opium, morphine, codeine, and heroin. Russell suggests that nicotine is addictive because it stimulates the hypothalamus, which is considered to be the "pleasure center" in the brain. He states, "It is likely that the special feature of dependence-producing potential possessed by some psychoactive drugs [including nicotine] rests in their ability to either directly or indirectly influence the hypothalamic reward system."[5] He claims that stimulation of the hypothalamus is a more powerful reinforcer of behavior than hunger, thirst, and sex.[5]

Some researchers now believe that nicotine is the *most* powerful addictive drug. Russell also quotes a survey indicating that 85% of those who smoke

more than *one* cigarette develop a dependence on nicotine.[5] Thus, nicotine addiction is established even more quickly than heroin addiction.

Robert Dupont, former director of the NIDA, estimates that nearly 70% of people who ever smoked (and are still alive) still smoke on a regular basis.[14] He compares this to less than 15% of people who ever used heroin and are still alive and addicted. Once established, the smoking habit is harder to break than addiction to heroin. In fact, heroin addicts consider nicotine to be more "needed" than heroin. In 1974, 278 British opiate users were asked to list a number of drugs in subjective order of greatest personal need. They rated nicotine above heroin, methadone, amphetamine, barbiturates, LSD, cannabis, alcohol, and tea or coffee.[5] I'll have more to say about coffee in a future essay.

There are several reasons why nicotine is more addictive than other dependence-producing drugs. First, the beginning smoker inhales around 200 puffs of nicotine-rich smoke in his or her first pack of cigarettes. The heroin user starts off with only one or two shots a week. The pack-a-day smoker "shoots up" more than 50,000 nicotine puffs in a year! Behavioral psychologists agree that the strength of a habit increases with the frequency of its reinforcement.[5, 16] Nicotine forms the most addictive habit because it is reinforced most frequently.

A second factor affecting habit strength is the timing of reinforcement. After heroin enters the bloodstream through intravenous injection, it takes almost 14 seconds for it to reach the brain. Nicotine enters the bloodstream through the lungs—it takes only eight seconds for it to reach the brain and pay off its pharmacological rewards.[5] The Australian Council on Smoking and Health says this rapid absorption explains why nicotine is so much more dependence-producing than alcohol and other drugs.[16] I suppose when you are addicted, six seconds faster can seem like a vast improvement.

Third, cigarette smoking is a socially accepted habit. The Royal College of Physicians contrasts the role of cigarette smoking with alcohol and barbiturate use in society. "Most people who drink alcohol or take sleeping pills are able to do so in moderation or on special occasions and can tolerate periods without them. It is only a small minority who become alcoholics or addicts. Furthermore, dependence on alcohol or barbiturates usually occurs in settings of psychological or social difficulty. With cigarette smoking the situation is altogether different. The most stable and well-adjusted person will, if he smokes at all, almost inevitably become dependent on the habit."[10]

As any cigarette smoker will tell you, it is much harder to kick the habit than to acquire it. Since the 1950s, when public withdrawal clinics first opened in the Scandinavian countries, there have appeared almost as many smoke control programs as there are brands of cigarettes. Jerome Schwartz, chief of health care research at the California Department of Health, Sacramento, classifies the variety of smoke control programs into nine categories: individual counseling by health professionals; educational programs sponsored by schools or commercial groups; group control activities sponsored by volunteer associations, foundations, commercial groups, and health departments; medications used to help smokers overcome their habit and withdrawal symptoms; hypnosis; behavioral conditioning; self-control procedures; mass-media programs on the risks of smoking and ways to kick the habit; and community efforts to involve neighborhoods and cities in educational programs.[17]

Given the evidence for physical dependence on nicotine, you would think that smoke control programs concentrate on handling withdrawal problems. However, most do not. It is no coincidence that these smoke control programs show disappointingly low cure rates. Schwartz surveyed 123 smoking cessation programs and found that only one-fifth had success rates of 40% or better. Nearly one-half of the programs had success rates of 21% or *less*. This would seem to be the expected rate based on the British data. Only three of the programs in the survey claimed success rates higher than 70%.[17]

Edward Lichtenstein, professor of psychology at the University of Oregon, Eugene, admits that behavioral programs are generally unsuccessful because they ignore nicotine dependence as one of the more important clinical aspects of smoking behavior. He says, "Most social learning workers, including myself, consistently ignore the implications of a large body of research which suggests that nicotine is a primary reinforcer for smoking and that, at least for heavy smokers, there are internal or physiological stimuli that drive the smoking habit.... The challenge for social learning workers is to incorporate this information on physiological processes into treatment programs. At least, we should probably cease trying to persuade smokers that their habit is entirely or even largely under external stimulus control."[18] (Incidentally, a social learning worker is a psychologist who studies the situational and environmental factors influencing a person's behavior.)

The Five-Day Plan, organized by the Seven-Day Adventist Church, uses the educational and group approach. While it acknowledges nicotine as an addictive drug, the Plan encourages people to overcome their habit by changing their attitudes toward both themselves and cigarettes. V.E. Gardner, medical director of the Five-Day Plan at the Philadelphia Better Living Center, explains, "We strongly follow concepts of nicotine as an addictive drug with very definite withdrawal symptoms. The plan is designed to minimize these. The withdrawal symptoms clear up in less than one week if no cigarettes are used. We get together each day to support each other during the few days of withdrawal. During this time, focus is placed on changing attitudes—toward the cigarette and towards oneself. The cigarette must not be looked upon as a reward or its denial a deprivation. New health promoting behavior gives a new self image in which smoking is out of character. It is these changed attitudes that are of real help in preventing a return to smoking, for the psychological craving persists even after the physical withdrawal symptoms have cleared."[19]

Smokers in the Five-Day Plan attend five consecutive group-therapy sessions, each lasting one and one-half hours. Sessions include lectures, films, and discussions on the psychological aspects of the smoking habit and ways to overcome it. The Five-Day Plan also encourages physical fitness exercises, balanced diet, hot and cold showers, and abstention from tea, coffee, and alcohol. A series of monthly follow-up meetings is organized to make sure that those who have quit or cut down on their smoking continue to do so.

During the five days of treatment, between 70% and 100% of the participants stop smoking. But, Schwartz points out, "Follow-up reports indicate that recidivism is high.... In-residence treatment at the Seventh Day Adventist Church's facility in St. Helena, California, showed 35% cure rates a year afterward."[17] Data from ten Five-Day Plan programs in different parts of the world show cure rates ranging from 16% to 40% after periods ranging from six months to five years.[17] Again we find that only about one in five can break the habit. Evidently, the changes in attitude and self-image are less permanent than the persistent physiological craving for nicotine.

The Smokenders program uses a behavior modification approach to help

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the smoker kick the habit. The program is designed with nicotine addiction in mind. Participants are given "assignments"—things to do which physically and psychologically recondition them for withdrawal. Lois Rafalko, vice president for program and training at the Smokenders headquarters in Phillipsburg, New Jersey, explains, "We do things to deal with the physiological withdrawal in advance. It happens gradually so that on the day when the member stops smoking he doesn't have the physical trauma of 'cold turkey.'"[20] Cold turkey is the abrupt cessation of drug intake, at which time the drug user begins to feel symptoms of withdrawal.

Participants in the Smokenders program attend eight weekly sessions, each lasting two hours. The first five sessions are smoking sessions—participants can smoke as much as they choose. But participants must stop smoking completely on the day after the fifth session. The last three sessions are reinforcing meetings that prepare reformed smokers to live without the habit. As long as people are not smoking they can return after the program ends for continued reinforcement.

The gradual and "painless" modification of smoking behavior makes the Smokenders program one of the more successful methods. Rafalko claims that 80-85% of those who complete the program are not smoking at all at the end of eight weeks. At the end of one year, 70% of those who quit smoking in the program are still not smoking. These data have not been confirmed by independent reviewers. Let's hope it is true.

Any program that tries to change smoking behavior faces a number of problems from the start. First, little has been done to identify tried and true smoke control techniques. Instead, researchers are influenced by currently popular ideas. Lichtenstein states, "The complexity of smoking permits a wide variety of social learning strategies and tactics to be applied. Unfortunately, choices often seem to depend on current fads or trends rather than flowing from a clinical and empirical analysis of smoking behavior itself."[18]

Also, behavioral smoke control programs must treat a large number of variables. Phoebus Tongas, chief psychologist at the Kaiser-Permanente Medical Center, says, "[Smoking] is under the control of such a great number and variety of discriminative stimuli and reinforcers that the task of eliminating it for long periods of time is immensely difficult as every research study has shown."[21] Lastly, the number of variables is multiplied by the number of individual smokers. John Pinney, director of Health, Education, and Welfare's Office of Smoking and Health, remarks, "If we've got 53 million smokers, we've got 53 million different kinds of smokers."[22]

Treating the cigarette habit as a physical addiction makes the problem of kicking the habit simpler. Instead of balancing hundreds of behavioral stimuli and reinforcers, the researcher can concentrate on the relatively few physiological motivations behind nicotine dependence. Pinney says, "If we can find some common denominators between [smokers]—biochemical or otherwise—then that will help."[22]

Schachter, suggests one such biochemical common denominator. He was interested in the smoker's claim that smoking is calming in stressful, anxious situations. Using a number of experimental subjects and strategies, Schachter found that "the smoker's mind is in the bladder."[12] Stress results in making the urine more acidic. When the urine is acidic nicotine is flushed out of the body. The smoker begins to feel withdrawal symptoms as the level of nicotine in the body decreases. "When the urine is alkaline, only one fourth as much nicotine is excreted as when the urine is acid; this is explained by the fact that nicotine base is reabsorbed from an alkaline urine."[12]

Psychologist James Fix at the University of Nebraska in Omaha thinks this biochemical fact can be practically applied to kick the smoking habit. Fix and co-workers divided a total of 42 subjects into three groups. One group took daily supplies of sodium bicarbonate, which decreases the acid content of the digestive system. Another group took vitamin C, which raises the acid content. The third group took a placebo.[23]

After five weeks, Fix reported "totally astonishing" results: "The bicarbonate group's average daily consumption dropped drastically to 0.14 cigarettes, while the vitamin C group and placebo groups went to 7.8 (up slightly from the fourth week); moreover, bicarbonate takers proved more likely to abstain from smoking for a 48-hour period."[22] Fix cautions that these results are preliminary, since many variables were not controlled in the experiment. However, they are encouraging enough to support the original idea that increased alkaline content makes the craving for nicotine less compelling.

There is good reason to be excited by Fix's advance in smoking control research. Any advance that does not depend upon a behavior modification program is bound to be valuable. It should encourage basic research scientists and others involved in clinical research because a cure for the disease is attainable. Whether we are coming closer to a cure is debatable. The Australian Council on Smoking and Health asserts, "An effective cure [for smoking] would rank with the discovery of penicillin in its effect on the health of mankind."[16] Russell adds, "The effective control of cigarette smoking is potentially the most important health measure that is likely to be open to us for the rest of this century."[5]

But it will not be enough to cure smokers of their addiction to nicotine. It is even more important to prevent people from getting hooked in the first place. This involves changing society's attitude toward the smoking habit. Recently, WHO's Expert Committee on Smoking Control recommended that "nonsmoking be regarded as 'normal social behavior' and [that] governments ...step' up antismoking legislation to establish that objective.... The purpose of smoking-control measures is not to punish the smoker but to encourage recognition of non-smoking as the norm."[24] Perhaps when the smoke clears a bit society may see the wisdom in this position.

Not only would it be illegal for cigarette companies to dispense samples to adolescents, but the government would be ready to discourage tobacco growing. One cannot ignore the economic significance of the cigarette industry. Cigarettes will be produced even if all forms of advertising and promotion are banned. Tobacco production will stop only when a better use for tobacco crop land is found. This is difficult to achieve because thousands of small farmers derive high income from a few acres of tobacco. The complicated nature of agricultural politics makes it doubtful that we would curtail tobacco farming to eliminate "the most lethal of all breathable pollutants."[16] Consider how difficult it has been to prevent production of the poppy plants needed to produce heroin.

The tobacco industry is well aware of society's changing attitude toward the cigarette habit and of the increasing restrictions on cigarette advertising and smoking in public places. As a result, it has turned to Third World countries in order to bolster declining sales. Blair points out that the Third World market is ripe for exploitation because it is "eager for symbols of Western affluence and still unencumbered by health and advertising regulations."[14]

Another recent article noted that the US government is helping the tobacco industry market cigarettes to the Third World at the same time it helps to alleviate malnutrition there. "Perhaps the most cynical foreign use of tobacco by the United States has been the inclusion of tobacco in the...'Food for Peace'

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program of concessional agricultural sales to needy countries. This supposedly humanitarian aid program has been manipulated...to get rid of unwanted domestic tobacco surpluses, to introduce foreigners to US tobacco in hopes of nurturing a future commercial market, and to provide aid to politically favored governments."[25]

But the Third World countries are not as ignorant as the tobacco industry hopes. China, the world's leader in cigarette production, recently acknowledged the link between cigarette smoking and cancer. "The government's first anti-smoking effort is aimed at discouraging the young from taking up the habit.... The reason for the push may not be hard to find: the World Health Organization estimates that cancer is the leading cause of death in China."[4] In Egypt, where smoking has increased *tenfold* in the last two years, cigarette advertising has been banned from television and smoking has been restricted in theaters and on planes. Cairo's English-language newspaper, *The Gazette*, angrily observes, "The tobacco industry, like the arms industry, is looking increasingly to the vast Third World for what sensible people would call suckers with a death instinct."[4]

While the tobacco industry here and abroad will not be dissuaded from pushing nicotine in the Third World, the US government can be. Lest anyone be left with the impression that it is only the profit-minded capitalists who are guilty of indirect mass murder through smoking-induced disease, let me remind you that the smoking habit is equally tolerated, supported, and prevalent in the socialist countries. Clearly it is a problem that is not going to be solved by cliches.

It is also clear that the amount of research directed at a solution is pitifully small. The total research budget this fiscal year for the NIDA is $48 million; only $1.7 million of that is going into smoking research.[26] Not only should there be direct allocations to basic and applied research from tobacco revenues, there should also be allocations for prevention programs and treatments. Two bills, now in the Pennsylvania General Assembly, would allocate funds from the sales of cigarettes for such worthwhile programs. One would place a 1 cent per pack tax on cigarettes to be paid to the Pennsylvania Cancer Control and Research Fund.[27] The other would place a 1 cent per pack tax on cigarettes to be paid to the state's Department of Health for grants and low interest loans for the payment of cancer treatment.[28] Let us hope that these bills and others like them will be passed.

I do not deny that I feel strongly about this issue. My father and three of my best friends died of smoking-related diseases. Every one of them would have supported every effort, financial and otherwise, that would assure future generations would be spared the agony of emphysema and other smoking-induced diseases.

At the same time we need to recognize that the smoking problem is a very complicated issue. Simplistic formulas for resolving the complex issues involved are not enough. While I would never support legislation to outlaw cigarette production, there is not adequate moral justification for cigarette production merely because people want it. The tobacco industry has an obligation to help support research that will produce disease-free alternatives for future generations.

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*Reprinted in Garfield E. *Essays of an information scientist.* Philadelphia: ISI Press, 1977. 2 vol.

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My Turn

Juneau Empire - 4-5-83

In Defense of Smokers

By MICHAEL COHERN

During the last week I have been inundated by propaganda from anti-smoke lobbyists. I would like to tell you what has transpired to me during this last week that has me wondering why no one has looked at the other side of this one-sided debate.

Late last week as I sat having my morning coffee downtown, I heard two people discussing House Bill 84 (an amendment to existing law on smoking in public places, to include places of employment). I asked if they smoked. They most certainly did not. I asked if any of them drove a motor vehicle. One wouldn't answer, but the other told me she owned a '74 VW. I asked if it used oil. She said it smoked a little.

"Isn't this a little hypocritical?" I asked. "A car puts out more pollution and smoke in one hour than a smoker does in a month! Ever have an accident? How much property is damaged each year by drivers? How many 'innocent' people are hurt every year? Ever known anyone to die from an accident? These are typical complaints of non-smokers to smokers."

She suddenly she realized she was late for work, and promptly left.

The next morning, I heard a young man voice the opinion that smoking in public was terrible, as he had to smell it.

"Have you ever had to set next to a drunk? Does he smell better?" I asked him. "What about a woman who uses so much perfume she makes you choke at twenty feet? Ever smell someone who doesn't shower very often? What about sitting next to someone with bad breath?" He wasn't so nice. Instead of him leaving, he told me where to go.

A few days later, I met two ladies talking about the "foul air around people that smoke." One said she had "rights" to clean air. I told her foul air came with industry, cars, big cities and even refuse burning — as well as a tiny bit from cigarettes.

Rights? Doesn't everyone have rights? It was quoted (incorrectly) that only 40 to 45 percent of the population smoked. What is it that they are not saying? Only that this figure represents everyone, regardless of age. The true fact is well over 50 percent of the adult population smokes. The minors don't count, as they can't legally purchase cigarettes anyway. So why is the minority (of voting age) telling the majority what it can or cannot do? Doesn't the majority rule?

Smokers are already cornered, restricted, punished and regulated. Are non-smokers? Can non-smokers get a ticket for abstaining in certain public places? Are there smoking areas where non-smokers are not permitted? Do airlines save the majority (and the best) seats for smokers? Are non-smokers ridiculed for not smoking? Are non-smokers harassed and hounded to smoke by smokers? Do smokers get the best tables in restaurants segregating smokers from non-smokers? Can smokers

... It was at this point that I finally realized that they were no longer sitting next to, or even near me — they had moved!

Yesterday morning, again having my morning coffee - I was handed a petition for support of the anti-smoke HB 84. Ah-ha! In it, was the statement that smoking was harmful, increased employer costs (through higher costs for health, fire and life insurance, as well as worker compensation coverage.) The person that handed it to me must have already heard of me, though, because he didn't stay to debate the petition. A clear case of hit-and-run!

Now about the petition; someone hasn't done their homework, obviously. Anyone who ever smoked knows that it is far more dangerous to tell a smoker he can't smoke than to let him smoke! Who wants an angry smoker, suffering from withdrawal, to work with?

And who will pay all the lost man-hours a worker will have if he or she has to stop working to run to an obscure, hidden-away smoke room for 5 to 10 minutes every time he or she wants to smoke? What smoker can concentrate on their work properly when they are suffering physical as well as mental withdrawal? The costs for these lost minutes will far outweight any minor additional insurance costs.

Today, at lunch, I listened to a nurse describe her patients. (Not personal information, just general information.) I heard her say that more than 75 percent of the patients at her hospital do not smoke. Hmmm Perhaps people should smoke to lower overcrowding of our hospitals. Maybe non-smokers are the principal cause of exhorbitant hospital costs. (If they smoked, there wouldn't be as many in the hospitals.)

I don't really believe that last paragraph, but any honest person must admit, the statements are just as reasonable as most complaints non-smokers have against smokers. Come to think of it, if you check our hospital you will find the majority of ill patients is there because of auto accidents, alcohol-related diseases and accidents and infections. Very few patients are there from tobacco related problems.

What have I been saying? Listen to me, please. I firmly believe that we all should take a closer look at the other side of HB 84. Is this a minority trying to force a majority to do their will? Will this bill hurt more than it helps? Will it save Alaska money?

I believe each of us has some habit — eating too much, drinking, foul or abusive language, drugs, unmentionable sexual diversities, or any number of other problems. How would you like to be harassed because of yours?

I find it incredible that a society that accepts the murder of the unborn, the public drunkenness and many other far more serious problems should attack the personal rights of an individual to smoke.

Let's quit finding fault with others or their habits. Everone has a habit. What's yours? Cars? Alcohol? Drugs? Are these really safer than smoking? I think not.

Cigarettes are not the No. 1 preventable killer we've been led to believe. Cars are. Followed closely by alcohol. Then abortions. Then possibly tobacco. So should we ban the car? What about alcohol? (That was done once. Anyone remember prohibition? Increased crime, violence, even bloodshed. Did it help? Will prohibition of cigarettes produce the same effects? You bet!)

Perhaps we should ban obnoxious perfumes, outlandish cosmetics, people with bad breath, or maybe blue-eyed people with blue suits. Tax collectors are offensive — let's ban them! How about nasty teachers? Why not make cuss-rooms for all our foul-mouthed friends?

Or should we just stop to realize that the anti-smoke campaign is just as ridiculous as those already mentioned?

Honorable members of the Legislature, I ask that you re-evaluate this highly volatile topic. I ask for rejection of HB 84 in its attack on personal freedom. If you feel you cannot support this opinion, at least leave it up to the people, those who elected you. Let us decide.

Again I ask, let this be put before the people — or reject this willful destruction of personal freedom.

Every Alaskan concerned with the erosion of our personal human rights, should be upset with this House Bill 84, not just smokers. Today, it's smoking. What rights will be under attack tomorrow? If you feel this is an attack on your personal freedoms, contact your local representative and senator today. Let him know how you feel — today! Tomorrow may be too late. The address to write is:

Senator John Q. Public
Alaska Legislature
Pouch V
Juneau, 99811

related activities. Again, we

Juneau

1980 – Labor Relations Yearbook

scope of the major issues that the participation teams are empowered to address. As the parties' statement of the terms of their Experimental Agreement puts it:

"Appropriate subjects, among others, which a Team might consider include: use of production facilities; quality of products and quality of the work environment; safety and environmental health; scheduling and reporting arrangements; absenteeism and overtime; incentive coverage and yield; job alignments; contracting out; and energy conservation and transportation pools."

St. John cautioned in his Arden House remarks that "the bottom line for the plan won't be determined for many years." He also said that a "crucial" element in the plan's success will be "effective problem solving through the better communications approach" of the teams.

An advantage of the stress on resolving these issues at the local plant level, he noted, is the flexibility of response this provides. The teams "will have the ability to deal with problems at the local level that may be unique or otherwise special to that particular department."

In the plan, the parties suggest that their experience in collective bargaining, although "successful" in many respects, has been disappointing in the resolution of "many problems . . . at the work site." These "are not readily subject to resolution under existing contractual programs and practices . . .," according to the parties.

They indicated that the joint team attack on such problems "is an essential ingredient in any effort to improve the effectiveness of the company's performance and to provide employees with a measure of involvement adding dignity and worth to their work life."

Study on Employer Anti-Smoking Programs

About 15 percent of the nation's businesses have programs to encourage and help workers stop smoking, according to a study prepared for the Department of Health and Welfare.

The study, which surveyed top-level management and medical officials in 3,000 U.S. firms, found that one-third of the companies are interested in developing or expanding smoking and health programs for workers.

Employer Cost

The National Interagency Council on Smoking and Health (NICSH), which conducted the survey, said cigarette smoking accounts for $5 billion to $8 billion in direct health care expenses and another $12 billion to $18 billion in indirect costs for lost productivity, wages and absenteeism.

A significant number of responding companies have policies that restrict or prohibit smoking in the workplace, said NICSH, a coalition of more than 30 public and private health groups concerned with reducing smoking.

"Most policies had been initiated by management, and policies were most frequently applied to blue-collar work areas, where smoking often poses a safety as well as a health hazard," NICSH said. According to the survey, 65 percent of the firms established those policies after release of the first Surgeon General's report in 1964.

Companies responding to the NICSH survey ranked smoking programs third among health education and promotion efforts, following high blood pressure and weight control programs. Larger firms are more likely to have smoking programs, NICSH said.

Most workplace smoking and health programs are operated in-house, with distribution of materials on how to stop smoking, physician counseling and other health professional counseling, the study found. Most firms use their own staff to administer the programs and support activities with existing medical resources rather than separate funds, the report said.

Of the companies interested in developing or expanding worker anti-smoking programs, more than 70 percent said they would like assistance. One-third of the companies responding to the survey said they are unsure at present whether they want to begin or expand worker health programs.

More information on anti-smoking efforts and copies of the survey are available from NICSH, 291 Broadway, Suite 1005, New York, N.Y. 10008, Tel. 212-227-4390.

HB

85

STATE OF ALASKA
PRELIMINARY STATEMENT OF FISCAL IMPACT

Bill No: House Bill 85 Date on Bill: 1/20/83
Title: "An Act relating to the Alaska permanent fund (AS 37.13) and repealing the permanent fund
Sponsor: divident program (AS43.23); and providing for an effective date."
Requestor: St. Affairs, Judiciary and Finance Sponsor: Rep. Szymanski

1. Estimated fiscal impacts on:

a. Expenditures:

Millions
(Thousands of Dollars)

			FY 83	FY 84	FY 85	FY 86		
Capital			$117 mill.					
Operating								
Total			$117 mill.					

b. Revenues:

Revenue								

2. Source of funds to offset fiscal impact of bill:

3. Assumptions:

Bill takes effect immediately, and is retro to January 1, 1983. The $117 million figure is an approximate of the assets from the PFD that would now transfer to the Alaska permanent fund upon the bills effective date, taking in funds from FY 80, FY 81, FY 82, and FY 83 to date.

4. Disclaimer:
This statement has not been reviewed by the OMB in the Office of the Governor. It does not represent the policy of the Sheffield Administration or the final estimate of fiscal impact.

Prepared By: Mary Relford Phone: 465-2300
Division: Commissioner's office Date: 2/10/83

Approved by Commissioner: Robt A Smith Date: 2/10/83
Department: Revenue

5. Distribution:
Original to Legislative Finance
Copy to OMB
Copy to Sponsor
Copy to Requestor

2/8/83

AMENDED TITLE: SSHB 85

AN ACT RELATING TO THE USE OF THE INCOME OF THE ALASKA PERMANENT FUND (AS 37.13); REPEALING THE PERMANENT FUND DIVIDENT PROGRAM (AS 43.23); AND PROVIDING FOR AN EFFECTIVE DATE

PRIME SPONSOR: SZYMANSKI.

CO-SPONSORS:

CURRENT STATUS: 2/15/83 IN (H) JUDICIARY REFERRAL: FINANCE

DATE	SEQ	PAGE	LEGISLATIVE ACTION
02/11/83	01	0225	FIRST READING -- COMMITTEE REPORTS
02/15/83	02	0259	S.A. -- DP03, NR03
02/15/83	03	0259	F/NOTE HSE SUPPL #9
			JUDICIARY
			FINANCE
			RULES
****	**	**	*** *** ***

HB

87

ALASKA NETWORK ON DOMESTIC VIOLENCE AND SEXUAL ASSAULT

P.O. BOX 809
JUNEAU, ALASKA 99802
586-3650

POSITION PAPER
HB87: An Act prohibiting publication of the identity of persons charged with certain sexual offenses

The Alaska Network on Domestic Violence and Sexual Assault is a non-profit corporation composed of twenty-one programs statewide that provide domestic violence, sexual assault, and adult crisis intervention services to members of their respective communities. Network programs are funded in part through grants and contracts awarded by the Council on Domestic Violence and Sexual Assault.

The Network was established in 1978, and has as one of its primary focuses the elimination of domestic violence and sexual assault through provision of shelter, advocacy, and education/prevention services.

The Network supports the intent of HB87 to protect the privacy of the victim and reduce the potential for additional trauma.

HB 87

STATE OF ALASKA
THE LEGISLATURE

POUCH Y - STATE CAPITOL
JUNEAU, ALASKA 99811
907-465-3800

LEGISLATIVE AFFAIRS AGENCY

M E M O R A N D U M January 3, 1983

SUBJECT: Publication of identity of certain sex offenders (Work Order #13-0213)

TO: Representative Robert H. Bettisworth

FROM: James H. Lear
Legislative Counsel JHL

You have requested this office to draft a bill that would withhold names of individuals from public access in the case of certain sexual offenses such as incest in an effort to protect the victim from adverse publicity.

Your request could be satisfied by closing the criminal proceedings and records surrounding those proceedings similar to the methods employed in children's proceedings (AS 47.10). However, such a statutory scheme would in all likelihood be found violative of the right of the accused to a public trial guaranteed by Article I, sec. 11 of the Alaska Constitution. Additionally, the due process clause of the 14th Amendment to the U.S. Constitution prohibits secret trials in criminal proceedings according to the Alaska Supreme Court decision in RLR v. State, 487 P.2d 27 (1971). It might also present some problems with respect to Article 1, sec. 5 of the Alaska Constitution and the First Amendment of the U.S. Constitution (freedom of speech).

Although the absolute closing of trial proceedings and surrounding records would not be constitutionally permissible, a good argument can be made in support of prohibiting the press from identifying the defendant in conjunction with prosecution of incest charges. One could assert that the state has a compelling interest in such a limited protection

of the victim without affecting the right of the accused to a public trial or the right of the press to otherwise monitor the criminal proceedings.

Accordingly, I have drafted a bill that would subject the news media to misdemeanor charges if they reveal the identity of such a criminal defendant and would impose the same penalty as that provided in AS 47.10.090(b) and (c) pertaining to publication of identity in conjunction with juvenile delinquent status.

I would be willing to elaborate upon the legal principles and court decisions that underly the above conclusions if you so desire. Please feel free to contact me at 465-2450.

JHL:csh

STATE OF ALASKA
PRELIMINARY STATEMENT OF FISCAL IMPACT

Bill No: HB 87 Date on Bill: 1-20-83
Title: An Act prohibiting publication of the identity of persons charged with certain sexual offenses
Sponsor: Rep. Bettisworth
Requestor: House Judiciary

1. Estimated fiscal impacts on:

a. Expenditures:

(Thousands of Dollars)

	FY 83	FY 84	FY 85	FY 86
Capital				
Operating				
Total	-0-	-0-	-0-	-0-

b. Revenues:

Revenue				

2. Source of funds to offset fiscal impact of bill:

Source of funds not identified by sponsor

3. Assumptions:

NO FISCAL IMPACT

4. Disclaimer:
This statement has not been reviewed by the OMB in the Office of the Governor. It therefore does not represent the final estimate of fiscal impact.

Prepared By: Paul Conger Phone: 465-4338
Division: Administrative Services Date: 3-4-83

Approved by Commissioner: [signature] Date: 2/8/83
Department: Public Safety

5. Distribution:
Original to Legislative Finance
Copy to OMB
Copy to Sponsor
Copy to Requestor

2/15/83

Bill/Resolution No. HB 87

Title "An Act prohibiting publication of the identity of persons charged with certain sexual offenses; and changing Rule 8(1), Rules of Criminal Procedure."

Requested by House Judiciary Committee Date 2/3/83

II. FISCAL DETAIL

Agency Affected Department of Law

Program Category Affected Administration of Justice

BRU, Program, Or Subprogram(s) Affected Prosecution

(Note: If more than one budget component is affected, separate line-item amounts and funding for each component in the analysis section.)

EXPENDITURES (Thousands of Dollars)

	FY 83	FY 84	FY 85	FY 86	FY 87	FY 88
100 PERSONAL SERVICES						
200 TRAVEL						
300 CONTRACTUAL						
400 COMMODITIES						
500 EQUIPMENT						
600 LAND & STRUCTURES						
700 GRANTS,CLAIMS,ETC.						
TOTAL		-0-	-0-	-0-		

FUNDING (Thousands of Dollars)

GENERAL FUND		-0-	-0-	-0-		
FEDERAL FUNDS						
OTHER (Specify Source)						

POSITIONS

FULL TIME						
PART TIME						
TEMPORARY						

III. ANALYSIS (See Fiscal Note Preparation Instruction, Section III)

The Department of Law believes that this bill has serious constitutional problems and in light of those problems the department does not believe there will be any fiscal impact.

RECEIVED

FEB 8 1983

LEGISLATIVE FINANCE

IV. DATE February 4, 1983 PREPARED BY Richard I. Pegues, Dir. Adm. Svcs.

AGENCY Department of Law

Original: Legislative Finance PHONE 465-3672

cc: Budget and Management

Prime Sponsor (First Legislator Named)

33-001 (Rev. 12/82)

OMB Reviewed by: Guy Bell

THE LEGISLATURE OF THE STATE OF ALASKA
THIRTEENTH LEGISLATURE

FISCAL NOTE

[X] Expenditure Type
[ ] Revenue Type

I. REQUEST
Bill/Resolution No. HB 87
Title ~~Act prohibiting publication of identity of persons....~~
Requested by House Judiciary Date

II. FISCAL DETAIL
Agency Affected Department of Public Safety
Program Category Affected Crime ID and Apprehension
BRU, Program, Or Subprogram(s) Affected Troopers & Council on Domestic Violence
(Note: If more than one budget component is affected, separate line-item amounts and funding for each component in the analysis section.)

EXPENDITURES (Thousands of Dollars)

	FY 83	FY 84	FY 85	FY 86	FY 87	FY 88
100 PERSONAL SERVICES						
200 TRAVEL						
300 CONTRACTUAL						
400 COMMODITIES						
500 EQUIPMENT						
600 LAND & STRUCTURES						
700 GRANTS,CLAIMS,ETC.						
TOTAL		-0-	-0-	-0-		

FUNDING (Thousands of Dollars)

GENERAL FUND						
FEDERAL FUNDS						
OTHER (Specify Source)						

POSITIONS

FULL TIME						
PART TIME						
TEMPORARY						

III. ANALYSIS (See Fiscal Note Preparation Instruction, Section III)

NO FISCAL IMPACT

RECEIVED
FEB 9 1983
LEGISLATIVE FINANCE

IV. DATE 2-2-83 PREPARED BY Paul Conger Phone 465-4338
DIVISION Admin Services Initials
DEPARTMENT OF PUBLIC SAFETY Initials

Original: Legislative Finance
cc: Budget and Management
Prime Sponsor (First Legislator Named)

33-001 (Rev. 1/82) OMB Reviewed by: Eric Jaschever

HB

91

HB 91

"Vital statistics" refers to records of birth, death, fetal death, marriage, divorce, adoption and related data. With specific exceptions, vital statistics are not available for inspection.

The language of HB 91, taken from the 'Model State Vital Statistics Act' published by the Public Health Service, opens records to the public: birth records - 100 years after date of birth; and marriage, death, divorce, dissolution of marriage or annulment records - after 50 years.

Passage of HB 91 will make public approximately 33,000 of the 675,000 records held by the State Registrar of Vital Statistics. The bill addresses the concerns of geneological researchers. A zero fiscal note is attached to the bill. Committee of next referral is the Judiciary Committee.

Dave Palmer
March 4, 1983

18.50.310 DOCUMENT= 1 OF 1 PAGE = 1 OF 2
CHAPTER = 18.50
SECTION = 18.50.310
TITLE = 18
EADINGS TITLE 18.
HEALTH AND SAFETY.
CHAPTER 50.
VITAL STATISTICS ACT.
ARTICLE 4.
RECORDS.
ITATION SEC. 18.50.310.
ATCH LINE
DISCLOSURE OF RECORDS.
EXT (A) TO PROTECT THE INTEGRITY OF VITAL STATISTICS RECORDS, TO INSURE THEIR PROPER USE, AND TO INSURE THE EFFICIENT AND PROPER ADMINISTRATION OF THE VITAL STATISTICS SYSTEM, IT IS UNLAWFUL FOR A PERSON TO PERMIT INSPECTION OF, OR TO DISCLOSE INFORMATION CONTAINED IN VITAL STATISTICS RECORDS, OR TO COPY OR ISSUE A COPY OF ALL OR PART OF A RECORD, EXCEPT AS AUTHORIZED BY REGULATIONS ISSUED UNDER THIS CHAPTER.

(B) THE BUREAU MAY PERMIT THE USE OF DATA CONTAINED IN VITAL

18.50.310 DOCUMENT= 1 OF 1 PAGE = 2 OF 2
STATISTICS RECORDS FOR RESEARCH PURPOSES.

(C) INFORMATION IN VITAL STATISTICS RECORDS INDICATING THAT A BIRTH OCCURRED OUT OF WEDLOCK SHALL NOT BE DISCLOSED EXCEPT UPON ORDER OF A SUPERIOR COURT OR AS PROVIDED BY REGULATIONS.

(D) APPEALS FROM DECISIONS OF THE CUSTODIANS OF LOCAL RECORDS REFUSING DISCLOSURE UNDER (A) AND (B) OF THIS SECTION SHALL BE MADE TO THE STATE REGISTRAR, WHOSE DECISION IS BINDING UPON THE CUSTODIAN OF LOCAL RECORDS.

(E) THE DEPARTMENT MAY BY REGULATION PROVIDE FOR THE RELEASE OF INFORMATION TO AUTHORIZED REPRESENTATIVES OF ORGANIZATIONS OR FOUNDATIONS THAT COUNSEL THE NEXT OF KIN OF VICTIMS OF INFANT SUDDEN DEATH SYNDROME.

STORY (SEC. 27 CH 118 SLA 1960; AM SEC. 1 CH 132 SLA 1978)

601 * END OF DOCUMENTS IN LIST - ENTER RETURN OR ANOTHER COMMAND.

FISCAL NOTE

I. REQUEST
Bill/Resolution No. House Bill Number 798 Item 82
Title "An Act relating to disclosure of vital statistics records"
Requested by Representative Bettisworth Date

II. FISCAL DETAIL
Agency Affected Department of Health and Social Services
Program Category Affected Division of Administrative Services
BRU, Program, Or Subprogram(s) Affected Bureau of Vital Statistics
(Note: If more than one budget component is affected, separate line-item amounts and funding for each component in the analysis section.)

EXPENDITURES (Thousands of Dollars)

	FY 82	FY 83	FY 84	FY 85	FY 86	FY 87
100 PERSONAL SERVICES	0	0	0	0	0	0
200 TRAVEL	0	0	0	0	0	0
300 CONTRACTUAL	0	0	0	0	0	0
400 COMMODITIES	.4	0	0	0	0	0
500 EQUIPMENT	0	0	0	0	0	0
600 LAND & STRUCTURES						
700 GRANTS,CLAIMS,ETC.						
TOTAL	.4	0	0	0	0	0

FUNDING (Thousands of Dollars)

GENERAL FUND	.4	0	0	0	0	0
FEDERAL FUNDS						
OTHER (Specify Source)						

POSITIONS

FULL TIME	0	0	0	0	0	0
PART TIME						
TEMPORARY						

III. ANALYSIS (See Fiscal Note Preparation Instruction, Section III)

1. 8 foot table $225.00
2. Chairs (2) $175.00

$400.00

IV. DATE Feb. 23, 1982 PREPARED BY Paul P. Brooks
AGENCY Dept. of Health & Social Services
PHONE 465-3391

Original: Legislative Finance
cc: Budget and Management
Prime Sponsor (First Legislator Named)

33-C01 (Rev. 12/81)

STATE OF ALASKA
PRELIMINARY STATEMENT OF FISCAL IMPACT

Bill No: House Bill Number 91 Date on Bill:
Title: "An Act Relating to disclosure of vital statistics records"
Sponsor:
Requestor: Representative Bettisworth

Estimated fiscal impacts on:

a. Expenditures:

(Thousands of Dollars)

			FY 83	FY 84	FY 85	FY 86		
Capital								
Operating								
Total			0	0	0	0		

b. Revenues:

Revenue								

. Source of funds to offset fiscal impact of bill:

Responsibility for identification of funding is that of the author of the bill.

. Assumptions:

. Disclaimer:
This statement has not been reviewed by the OMB in the Office of the Governor. It does not represent the policy of the Sheffield Administration or the final estimate of fiscal impact.

Prepared By: Joan Brooks by D. Lee 1st. Phone: 465-3391
Division: Planning, Policy and Evaluation/Vital Statistics Date: February 22, 1983

Approved by Commissioner: Robert Loeber Smith Date: 2/22/83
Department: H & SS

. Distribution:
Original to Legislative Finance
Copy to OMB
Copy to Sponsor

2/8/83

HB 91 TITLE & SPONSOR SUMMARY 1c-01 3/08/83 PAGE 1 OF 2

AMENDED TITLE:
AN ACT RELATING TO DISCLOSURE OF VITAL STATISTICS RECORDS
AND INFORMATION;
AND PROVIDING FOR AN EFFECTIVE DATE

PRIME SPONSOR: BETTIWORTH.
CO-SPONSORS:
CURRENT STATUS: 3/07/83 IN (H) JUDICIARY

HB 91 HOUSE ACTION 1A:02 3/08/83 PAGE 2 OF 2

DATE	SEQ	PAGE	LEGISLATIVE ACTION
01/20/83	01	0069	FIRST READING -- COMMITTEE REPORTS
03/07/83	02	0418	HESS -- DPOS
03/07/83	03	0418	HESS F/NOTE EQUALS ZERO
			JUDICIARY
			RULES

(7)

COMMITTEE REPORT

3/7

HOUSE

1/20/83

FURTHER: JUDICIARY

Date: March 4, 1983

Mr. Speaker:

The Committee on HESS has had HB 91

An Act relating to disclosure of vital statistics records and information; and providing for an effective date.

under consideration and reports it back as follows:

[V] do pass [ ] do not pass

[ ] do pass with attached amendments(s)

[ ] replace with CS for _____ [ ] same title [ ] new title

and recommends _____

[ ] AND attaches a "Letter of Intent" [ ] New Fiscal Note [X] Zero Fiscal Note Attached

[ ] reports it back without recommendation

[ ] referred to the _____ Committee

MEMBERS SIGNING DO PASS

Peter Jacobs

Mike Bowen

Bette Cate

Mike DAVIS

MILO FRITZ

MEMBERS HAVING OTHER RECOMMENDATIONS:

MILO FRITZ

CHAIRMAN

CO-

H 60

February 24, 1983

House Health, Education and
Social Services Committee
Room 112, Capitol Building
Pouch V
Juneau, AK 99811

attn: Mr. Dave Palmer

Although I earnestly wish to testify in person on HB91, I am unable to postpone a trip on March 4th and, therefore, am submitting the following testimony in favor of the bill:

I am both a family and professional genealogist (Alaskan Records Research, Business License number BL000201); hold a BLA with history emphasis from the University of Alaska, Juneau; am a full-time student in the Master of Arts in Teaching program at Alaska Pacific University; am employed half-time at the UAJ library; tutor history, english, geography, and logic at UAJ; am Alumni Representative on the UAJ Assembly; have published a family history and am currently working (in my spare time) on an historical novel.

With this variety of efforts underway, you can understand why I have little time to spend writing back and forth to prospective clients to obtain the written permissions required before I can access vital statistics data as my client's personal representative.

As it stands now, individuals can obtain vital statistics on their own ancestors but professionals or those working on far distant lateral relatives may not easily do so. A business must be cost-effective. Repeated correspondence is not (unless I raised my fees to cover it, which would price me out of the market). Thus, I simply refer many inquiries to the office of Vital Statistics and so lose the fees I could obtain for doing the research myself.

Passage of this bill will help all who are searching for long-lost relatives who "went up north to the goldrush and were never heard of again."

Being the first of my family to reside in Alaska, I have no family interest in opening Alaska's records. However, I would personally like to see my state on a par with the rest of the country. After tracing several thousand of my own ancestors, lateral, and collateral relatives through most of the northern states and Ontario, and collecting hundreds of official documents dated from 1738 to 1978, I have never been refused a record because of access restrictions except hospital patient records. I feel that this bill is a step in the right direction but could go further. People researching in Alaska should meet with no more roadblocks than I have experienced in other states. I would like to see the time limits of 100 and 50 years reduced to about 30 years, if the purpose is genealogical research. The most persuasive argument against opening birth records is the problem of illegitimacy. Besides the fact that it no longer carries the stigma it once did, a professional genealogist couldn't care less what the records say, all he or she is interested in

is locating them for the client. The client or individual researcher is a member of the illegitimate child's family and thus has a personal interest in keeping the fact of illegitimacy within the family.

I feel a person's right to know about their own heritage outweighs any "right to privacy" of their ancestors and this includes a person's right to hire a researcher if unable or unwilling to travel to Juneau to do their own research. Therefore, I highly recommend passage of this bill, preferably with an amendment allowing disclosure to genealogists of those vital records not made public by AS 18.50.310(f).

Sincerely

Kit Stewart

Kit Stewart
9119 Nagoon Lane
Juneau, AK 99801
789-9411

a false status, occupation, membership, license, privilege, or identity himself or another person shall be fined not more than $[ ] imprisoned not more than [ ] years, or both.

(c) Any person who uses any such document to commit a crime shall punished by fine or imprisonment or both equal to that required by tute for the accompanying offense. Such sentence shall be served consecutively with that of the accompanying offense.

Section 4. [*Severability.*] [Insert severability clause.]

Section 5. [*Repeal.*] [Insert repealer clause.]

Section 6. [*Effective Date.*] [Insert effective date.]

Model State Vital Statistics Act (1977 Rec. Act.)

#291

The Model State Vital Statistics Act is a document designed to be used by state registrars of vital statistics and state legislators when considering revision of the vital statistics laws. The main objectives of the 1977 revision of the model act are (1) to incorporate current social customs and practices and current technology into the policies and procedures of the vital statistics system in the states, (2) to promote the uniformity of these policies and procedures to the end that all vital records will be readily acceptable in all places as prima facie evidence of the facts therein recorded, (3) to enhance the level of comparability of vital statistics data among the states, and (4) to minimize duplication within the vital statistics system and thereby achieve maximum administrative economy.

The historical philosophy of the vital statistics systems in the United States is that vital events be registered only in the state in which they occur. This concept is maintained in this revision of the model act. The jurisdiction of the state registrar extends only to boundaries of his state, and standards for registration may be set and enforced only for those events occurring within those boundaries. This is a very important concept in maintaining the validity of vital records in their use for legal purposes. If it is to be respected, the appropriate procedures for recording birth and death information for United States citizens born or dying in foreign countries and certification of birth information for aliens adopted by United States citizens must continue to be the responsibility of those federal agencies which retain jurisdiction over recording these events.

While this revision of the model act does not constitute an abrupt departure from earlier acts, there are several modifications that should be noted. The most significant change relates to the establishment of a centralized system for the collection, processing, registration, and certification of vital records in each state, whereby all vital events are reported directly to the State Office of Vital Statistics. However, the model act contains authorization for local offices to perform those functions the state registrar may direct, including the receipt and processing of vital records and the issuance of certified copies, when such offices can be shown to be an aid to efficient and effective operation of the system. The model act further provides for the options of allowing such local offices to work with records only for their designated geographic area or to be given access to the entire state file and allowing them to issue certified copies without regard to where the event occurred within the state. The important concept, however, is that these offices are part of the State Office of Vital Statistics and are under the direct control of the state registrar.

The recommendation for a change from a locally oriented vital statistics system to a centralized system is based on several considerations: (1) a centralized system produces more timely registration of the records, thereby improving the timeliness of all operations, including publication of statistical data as well as fulfillment of citizens' needs for vital records services; (2) it decreases duplication and cost since many activities presently performed at local vital records offices are repeated at the state office; (3) it reduces the opportunity for fraudulent use of certified copies because amendments to the records will be easier to control and certified copies will

...records of each [divorce, dissolution of marriage, or annulment] decree ...mitted during the preceding calendar month.

...(c) [Provision for a recording fee may be added here if desired.]]

Section 22. [*Amendment of Vital Records.*]

(a) A certificate or report registered under this act may be amended only in accordance with this act and regulations adopted by the state agency to protect the integrity and accuracy of vital records.

(b) A certificate or report that is amended under this section shall be marked "Amended," except as otherwise provided in this section. The date of amendment and a summary description of the evidence submitted in support of the amendment shall be endorsed on or made a part of the record. The state agency shall prescribe by regulation the conditions under which additions or minor corrections may be made to certificates or records within one year after the date of the event without the certificate or record being marked "Amended."

(c) Upon written request of both parents and receipt of a sworn acknowledgment of paternity signed by both parents of a child born out of wedlock, the state registrar shall amend the certificate of birth to show such paternity if paternity is not already shown on the certificate of birth. Such certificate shall not be marked "Amended."

(d) Upon receipt of a certified copy of an order of [court of competent jurisdiction] changing the name of a person born in this state and upon request of such person or his or her parents, guardian, or legal representative, the state registrar shall amend the certificate of birth to show the new name.

(e) Upon receipt of a certified copy of an order of [court of competent jurisdiction] indicating the sex of an individual born in this state has been changed by surgical procedure and that such individual's name has been changed, the certificate of birth of such individual shall be amended [as provided in Regulation 10.8(a)(5)] to reflect such changes.

(f) When an applicant does not submit the minimum documentation required in the regulations for amending a vital record or when the state registrar has reasonable cause to question the validity or adequacy of the applicant's sworn statements or the documentary evidence, and if the deficiencies are not corrected, the state registrar shall not amend the vital record and shall advise the applicant of the reason for this action and shall further advise the applicant of the right of appeal to [court of competent jurisdiction].

(g) When a certificate or report is amended under this section, the state registrar shall report the amendment to any other custodians of the record and their record shall be amended accordingly.

Section 23. [*Reproduction of Vital Records.*] To preserve vital records, the state registrar is authorized to prepare typewritten, photographic, electronic, or other reproductions of certificates or reports in the [Office of Vital Statistics]. Such reproductions when certified by the state registrar shall be accepted as the original records. The documents from which permanent reproductions have been made and verified may be disposed of as provided by regulation.

Section 24. [*Disclosure of Information from Vital Records.*]

(a) To protect the integrity of vital records, to ensure their proper use, and to ensure the efficient and proper administration of the system of vital statistics, it shall be unlawful for any person to permit inspection of, or to disclose information contained in vital records, or copy or issue a copy of all or part of any such record except as authorized by this act and by regulation or by order of [court of competent jurisdiction]. Regulations adopted under this section shall provide for adequate standards of security and confidentiality of vital records.

(b) The state agency may authorize by regulation the disclosure of information contained in vital records for research purposes.

(c) Appeals from decisions of custodians of vital records, as designated under authority of Section 6(b), who refuse to disclose information, or to permit inspection or copying of records as prescribed by this section and regulations issued hereunder, shall be made to the state registrar whose decisions shall be binding upon such custodians.

(d) When 100 years have elapsed after the date of birth, or 50 years have elapsed after the date of death, marriage, or [divorce, dissolution of marriage, or annulment], the records of these events in the custody of the state registrar shall become public records and information shall be made available in accordance with regulations which shall provide for the continued safekeeping of the records.

Section 25. [*Copies or Data from the System of Vital Statistics.*] In accordance with Section 24 and the regulations adopted pursuant thereto:

(1) The state registrar [and other custodian(s) of vital records authorized by the state registrar to issue certified copies] shall upon receipt of a written application issue a certified copy of a vital record in his or her custody or a part thereof to any applicant having a direct and tangible interest in the vital record. Each copy issued shall show the date of registration and copies issued from records marked "Delayed" or "Amended" shall be similarly marked and show the effective date. The documentary evidence used to establish a delayed certificate shall be shown on all copies issued. All forms and procedures used in the issuance of certified copies of vital records in the state shall be provided or approved by the state registrar.

(2) A certified copy of a vital record or any part thereof, issued in accordance with Section 25(1), shall be considered for all purposes the same as the original and shall be prima facie evidence of the facts stated

HB

94

STATE OF ALASKA
FISCAL NOTE

Revision Date May 19 , 1983

I. REQUEST

Bill/Resolution No.: CSHB 94 (Judiciary)
Title: "...seizure of items..."
Sponsor: House Judiciary (orig.-Bettisworth)
Requestor: House Finance Committee

II. FISCAL DETAIL

Agency Affected: Department of Law
Program Category Affected: Admin. of Justice
BRU, Program of Subprogram(s) Affected: Prosecution

EXPENDITURES/REVENUES: (Thousands of Dollars)

	FY 83	FY 84	FY 85	FY 86	FY 87	FY 88
OPERATING						
100 PERSONAL SERVICES		118.4	149.8	158.8	168.3	178.4
200 TRAVEL		10.0	12.8	13.6	14.4	15.3
300 CONTRACTUAL		18.0	20.3	21.5	22.8	24.2
400 COMMODITIES		10.0	5.7	6.0	6.4	6.8
500 EQUIPMENT		17.5				
600 LAND & STRUCTURES						
700 GRANTS, CLAIMS, ETC						
TOTAL OPERATING		173.9	188.6	199.9	211.9	224.7
CAPITAL						
REVENUE						

FUNDING: (Thousands of Dollars)

GENERAL FUND		173.9	188.6	199.9	211.9	224.7
FEDERAL FUNDS						
OTHER (Specify Source)						

POSITIONS:

FULL-TIME		3	3	3	3	3
PART-TIME						
TEMPORARY						

III. SOURCE OF FUNDS TO OFFSET FISCAL IMPACT OF BILL:

Not specified by sponsor.

IV. ANALYSIS: Attach a separate page for any Analysis

Prepared By: Richard I. Pegues, Director (Richard L. Pegues) Phone: 465-3672
Division: Administrative Services Division Date: May 19, 1983
Approved by Commissioner: Norman C. Gorsuch, Attorney General (Richard L. Pegues/for) Date: May 19, 1983
Department: Department of Law

Distribution:
Original to Legislative Finance
Copy to Office of Management and Budget (for Legislature introduced bills)
Copy to Department (for Governor introduced bills)
Copy to Sponsor
Copy to Requestor (if different from Sponsor)

CSHB 94
Fiscal Note
Analysis

This bill would have a significant negative impact upon the Department of Law's efforts to prosecute violations of the state fish and game laws. It allows seizure of items used in violations of the law only if the seizure is necessary to preserve items of evidence or to prevent the removal of items from the judicial district in which the violation occurred. A defendant who disagrees with this judgment on the officer's part may be expected to seek repossession, and this would have to be litigated. The bill would require the commitment of additional attorney time to attend the hearings which must be held within seven days after charges are filed in order to extend a seizure. All items that the owner establishes are necessary to the owner's livelihood must be returned to the defendant unless the department proves to the court that these items are needed as evidence or they are likely to be removed from the judicial district. This is a change in criminal procedure which is not found in any other area of criminal law; it would significantly impair our ability to prosecute these cases. Also, because there is no floor limit on the value of items, the disposal of objects of even very low monetary value such as tackle boxes would have to be litigated.

The Department of Law is opposed to the bill in its present form. Under current law, items which are used in the violation of fish and game laws may be seized. This seizure is not merely to preserve the items as evidence, but is also a sanction for the commission of the offense, the prevention of the movement of items such as boats and airplanes out of the jurisdiction, or their transfer to an innocent purchaser. In the past, persons who are under investigation for, or who have been charged with offenses have deliberately transferred their interest in items to prevent seizure by the state.

Of significant concern is the provision requiring the return of seized items within seven days after the charges are filed. The state should continue to retain possession of seized items until the conclusion of the criminal case as the court may order forfeiture as part of the criminal sentence. A seven day period is much too short to enable the department to adequately prepare for court hearings in these cases. Many items used in fish and game violations are seized in extremely remote parts of the state; there may be no representative of the Department of Law in the area on a regular basis. Local enforcement officers have the authority to file charges and seize evidence or items in connection with these charges, but will have no attorney available to attend a court hearing within seven days.

This bill would significantly impair the Department of

Law's ability to successfully investigate and prosecute fish and game violations. In light of the tremendous value to the State of Alaska of our natural resources and wildlife population, it would be inadvisable to decrease the protection of resources by the liberalization of the laws designed to deter violations of the fish and game laws and to facilitate the apprehension of offenders.

FISCAL ANALYSIS CSHB 94 (Judiciary)

The impact of CSHB 94 (Judiciary) is expected to result in the addition of two Attorney III positions (SR22) and one Legal Secretary I position (SR10) at Anchorage.

The first year of the analysis will be FY 84 and costs have been calculated on a 10 month basis to account for the time required to establish new positions and the time it takes to get a new program underway. The costs beyond FY 84 are over a 12 month basis and include a 6% annual inflation factor.

1st Year (10 months)

	Atty III	Atty III	Leg Sec I	Total
Personal Services	47.5	47.5	23.4	118.4
Travel	5.0	5.0	-0-	10.0
Contractual	8.0	8.0	2.0	18.0
Commod. - ongoing	1.5	1.5	1.5	4.5
Commod. - single time	2.0	2.0	1.5	5.5
Equipment - single time	1.5	1.5	14.5	17.5
				173.9

2nd Year (12 months + 6% annual inflation)

Personal Services	60.0	60.0	29.8	149.8
Travel	6.4	6.4	-0-	12.8
Contractual	9.1	9.1	2.1	20.3
Commodities	1.9	1.9	1.9	5.7
				188.6

1.	POSITION TITLE Attorney III			RANGE/STEP 22A	BARG. UNIT X	FORM 12 PAGE/LINE	COV.	APPROV.	DISAPP.
2.	TYPE OF POSITION PFT	STAFF MONTHS 10	RP NUMBER	PCN NUMBER	BRU PRIORITY	LOCATION Anchorage	ELECTION DISTRICT	LEG.		

3.	CONTINUATION LEVEL		ADDITION	
4.	TYPE OF EXPENDITURE			AMOUNT
	1		2	3
	PERSONAL SERVICES			
5.	Salary 3,714/month		37,140	
6.	Benefits		5,727	
7.	Supplemental Benefits		2,240	
8.	Fixed Benefits		2,400	
9.	TOTAL PERSONAL SERVICES	01		47,507
10.	Travel	02		5,000
11.	Contractual	03		8,000
12.	Commodities	04		3,500
13.	Equipment	05		1,500
14.	Other			
15.	TOTAL COST			65,507

	RECEIPT CODE	FUNDING SOURCE		
16.		Federal Receipts	1002	
17.		G.F. Match	1003	
18.		General Funds	1004	65,507
19.		I-A Receipts	1005	
20.		Program Receipts	1028	
21.		Other		

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4A KEY NUMBER __ __ - __ __ - __ __ - __ __ - __ __

JUSTIFICATION

This is the first of two attorney positions required by the Department of Law in order to absorb the significant increase in workload which will result from new legislation authorizing the return, within seven days, of items seized during arrests for fish and wildlife violations. Significant amounts of attorney time will be needed to extend seizures to protect evidence and to protect items that would otherwise be removed from the judicial district where the violation occurred.

13 REQUEST FOR NEW POSITION

AGENCY DEPARTMENT OF LAW

PROGRAM ADMINISTRATION OF JUSTICE

BRU PROSECUTION

COMPONENT THIRD JUDICIAL DISTRICT

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Revised Date

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