HEALTH, WELFARE AND STATE INSTITUTIONS

Minutes of Meeting - April 22, 1975

The twenty-fourth meeting of the Health, Welfare and State Institutions Committee was held on April 22, 1975 at 3:10 p.m. in Room 323.

COMMITTEE MEMBERS PRESENT: Chairman Lee E. Walker
Senator Neal
Senator Gojack
Senator Hilbrecht
Senator Schofield
Senator Young

OTHERS PRESENT: See Exhibit A

Senator Walker advised that we had previously passed A.B. 108 by deleting the section relating to guardianship. The Welfare Division has come up with a new Section 13 which says "In carrying out the objectives of this Chapter, the juvenile court may utilize the services of the Welfare Division". The committee agreed that this shall be added to the bill.

S.B. 98 - Clarifies rights of handicapped persons.

Senator Hilbrecht moved "Do Pass" and amend to include the Division's definition of "handicapped" and Senator Young's recommended restrictive language; seconded by Senator Gojack; Senator Neal voted no on the recommended restrictions; motion carried.

S.B. 459 - Provides for regulation of retail sale of convenience drugs.

Senator Walker advised that a compromise has been reached by the proponents and opponents of the bill.

Mr. George Bennett, Secretary State Board of Pharmacy, advised that they agree with the wording that Mr. Bill Bailey has proposed (see Exhibit B for proposed amendments).

Mr. Bill Bailey, Attorney for the Proprietary Assn., advised that the Retailers Assn. had hoped to be present, but since they were unable to do so, they requested Mr. Bailey to advise the committee that they support the bill with the amendments presented.

Senator Young referred to page two of these amendments and asked if an employee who obtains a retail dealer's permit would be able to sell drugs. Mr. Bennett agreed that this language could be amended to read "unless he or his employer has first obtained...." (see line 6 of amendment #7).

Senator Walker asked Mr. Bennett how long it would take them to issue all of these permits; Senator Hilbrecht suggested that this should not be made effective until January 1, 1975. Mr. Bennett felt that with the addition of another inspector in Las Vegas, they could accomplish this by July 1, 1975.

Senator Neal moved "Do Pass" as amended; seconded by Senator Hilbrecht; motion carried.

Mr. George Hawes, Assistant to Lou Paley - AFL-CIO, advised that they are in opposition to the bill because they feel the drugs should be available in grocery stores, etc. Mr. Hawes was concerned that service stations, small chain stores, etc. would have to have permits to sell drugs. Mr. Hawes further commented that the Pharmacy Board is building itself up into a bureaucracy. Mr. Hawes further advised that he was threatened by Mr. Bailey who said "If the bills fail to pass, Lou will be in serious trouble". Mr. Hawes advised that this is the first time he has been threatened by another lobbyist. Mr. Bailey responded to Mr. Hawes remark by stating that he had met with Mr. Paley and Mr. Bennett to review the amendments with them. Apparently there was a breakdown in communication. Mr. Bailey further stated that "since Lou was not here, I felt he would have concern about the bill not being attached to the amendments".

Senator Walker asked if there was a motion to reconsider the action taken on this bill; being no such motion, the action on this bill remained "amend and Do Pass".

S.B. 460 - Makes various changes relating to pharmacists and pharmacy.

Senator Walker advised the committee that he will obtain language regarding the commitment problem that was discussed at previous meetings. Senator Walker stated that this problem was that it gives the Board the right to suspend someone's license as a pharmacist if he has been declared incompetent.

Phyllis Hansen, Nevada Nurses Association, advised that they are opposed to the deletion of Section 5, line 24, regarding the rules and regulations adopted by the Board. Senator Walker advised Ms. Hansen that the committee had agreed to put that back in the bill and change the "shall" to "may".

S.B. 316 - Amends provisions regulating marriage and family counselors.

Senator Walker advised that it is his understanding that an agreement has been reached on page 2 (the psychologists felt that line 4 would authorize marriage counselors to conduct psychological testing). Senators Young and Gojack agreed that the possibility of an interim

study should be considered on this field as well as related fields.

Senator Hilbrecht moved that an interim study committee be appointed to study health related bills; seconded by Senator Young; motion carried.

Senator Walker commented that they had a interim study last session on mental health which they did not do much with because the Rand committee was authorized by the Fleischmann Foundation to study this. The committee should be authorized to review the Rand study to determine whether we want to adopt anything from the Rand study. Senator Hilbrecht so moved; seconded by Senator Young: motion carried.

S.B. 346 - Creates board of social examiners; provides for licensing of social workers.

Senator Young moved "Do Hold"; seconded by Senator Neal; Senator Hilbrecht voted "No"; motion carried.

S.B. 394 - Prohibits certain uses of special parking permits or plates for physically handicapped.

Fred Little, Department of Motor Vehicles, advised that they support this bill. Mr. Little advised that the problem has been that individuals who are not handicapped were driving vehciles that had handicapped plates; they were utilizing the handicapped parking spaces over and above the allotted time.

Blaine Rose commented that special parking permits and plates were issued to handicapped persons and to persons who were driving the handicapped. Some of these people were abusing the permit when they were not transporting handicapped persons. This bill adds language which states that the permits are not to be used in certain instances. (See Exhibit B-1).

Senator Young asked if these permits were revoked if they were being used improperly; Mrs. Rose replied that they had not done this in the past; however, they are now able to do this if it brought to the attention of DMV.

Senator Hilbrecht suggested that this be made a misdemeanor and give the Department the authority to revoke permits.

Senator Young moved "Do Pass" and amend to include misdemeanor section; seconded by Senator Hilbrecht ; motion carried.

A.B. 17 - Protects public health by imposing certain restrictions on smoking in public places.

Assemblyman Vergiels, in being the sponsor of this bill, advised the committee that so as not to hurt the economy of Nevada, he has gone along with amendments and stated that the casinos have been very cooperative with the amendments. Mr. Vergiels advised the committee that they were welcome to letters and informative data that he has received with respect to this bill.

Senator Young referred to line 12, page 1, of the bill and asked if this refers to elevators, etc. that are used by the public; Mr. Vergiels answered in the affirmative. Senator Young suggested that the word "public" be added at the beginning of line 12. Senator Hilbrecht suggested that on page 1, line 12, the word "art" be deleted.

Mr. Vergiels referred to page 1, line 16, and suggested that the wording "Hallway" be deleted so that a person could go out in the hall and smoke. Senator Hilbrecht felt it should be left the way it is.

Senator Young referred to page 2 and asked if the question of constitutionality of the healing arts being included in this had arisen. Mr. Vergiels stated that no one opposed this.

Mr. Frank Fahrenkopf, representing the Tobacco Institute of America, advised that they are opposed to this bill. (See Exhibit C for copy of testimony). Mr. Fahrenkopf presented the committee with copies of a letter from the American Cancer Society (Exhibit D) and an article from the Las Vegas Review Journal (Exhibit E).

Elaine Cooney, Mari Meyer and Sandra Sterrett of the Hug High Smoking Program spoke in favor of the bill. Elaine Cooney stated that this program was started 3 years ago with the intent that high school students would advise elementary students of the dangers and effects of smoking. The girls presented the committee with exhibits of cross-sections of the human lung in its various stages of that of a smoker. The girls invited the committee to attend one of their presentations.

Dr. Stephen D. Dow, Chairman of the Nevada Heart Fitness Inst., spoke in favor of the bill. Dr. Dow feels that smoking does have an adverse health effect on the non-smoker. Dr. Dow presented the committee with various articles relating to heart disease, smoking, etc. (see Exhibits F-1 through F-6). Dr. Dow advised that it is difficult to obtain data comparing the smoker to the non-smoker -- you would have to have two groups, one smokers and the other non-smokers, and then compare one to the other. Dr. Dow stated that it is very difficult to structure this type of study.

Mr. Dallis Pierson, Nevada Lung Association, advised that the Heart Association, Heart Fitness Institute, Nevada Heart Association and the Nevada Lung Association all support this bill. They feel that this bill would be a vote for the State of Nevada. Mr. Pierson presented the committee with a copy of an article published by the Tobacco Institute (see Exhibit G), and a copy of the 1972 Surgeon General's Report (see Exhibit H).

Senator Hilbrecht asked if he thought this legislation would be enforceable; Mr. Pierson replied that this is social legislation and feels that most people will not smoke in an area where it is prohibited.

Mr. Oliver Hansen, Sparks, spoke in favor of the bill. He is 65 years old and has always had good health. He has never smoked but has been bothered by the smoke from other people. Mr. Hansen feels that second-hand smoke is harmful to him. Mr. Hansen is in favor of conservative legislation but not in favor of legislation which stiffles free enterprise.

Senator Hilbrecht moved "Do Pass" and amend; seconded by Senator Schofield, motion carried.

Being no further business at this time, the meeting was adjourned at 5:15 p.m.

Respectfully submitted,

Sharon W. Maher

Sharon W. Maher, Secretary

APPROVED:

Lee E. Walker

Lee E. Walker, Chairman

With respect to A.B. 17, Senator Hilbrecht furnished a copy of letter from Dr. Gary Symonds which is attached hereto and marked as Exhibit I.

SENATE *Health, Welfare* COMMITTEE

ROOM # *323*

DAY *Tuesday* DATE *4-22-75*

NAME	ORGANIZATION	ADDRESS
Stephen D. Dow MD.	Nevada Heart Fitness Inst.	555 N Arlington, Reno
Roy Pussinelli	Southworth Tob. Co	233 East 5th Reno Nev
Bill Harrison	Reno-Sparks Convention Authority	P.O. 837 - Reno
Frank Fahrenkopf	Tobacco Inst. Washoe Cty	PO Box 1249 Reno
Valerie Voelker	Hug High School Smoking & Health program	630 Hoge Rd Reno
Tammy Garate	Hug High School's Smoking & Health program	77 Rhode Island Reno
Susan Parkhurst	Hug High School's Smoking & Health Program	210 Bartlett St Reno
Vicki Carpenter	Hug High Smoking Program	1950 Reed St. Reno
Dallis Pierson	Nevada Lung Assn.	877 Aitken St Reno
John Vergiels	Assembly	
Debbie Figoni	Hug High Smoking Program	8025 Channel Way Reno
Kathy Schammon	Hug High Smoking Program	10873 Osage
Mike Vesper	Sea & Ski Corp.	Reno, Nev.
Robert A. Groves	Atty Gen'l Office	
Phyllis Hansen	Nev. Nurses' Assoc.	1450 E. 2nd St.
Sandra Sterrett	Hug High Smoking Program	645 Magnolia Way Reno
Mari Meyer	Hug High Smoking Program	7680 Yorkshire Dr.
Elaine Cooney	Student to Student Program on Smoking and Health	13723 Mt Whitney Reno
Faye V. Hazen Sr	Invited by Nevada Lung association	2006 Prater Way, Sparks
Olive F. Hansen	" " " " "	739 16th St Sparks Nev
Joe Braswell	Inter-Tribal Council of Nevada	Capital Plaza Rm 115 1100 E. William - Carson City
Elaine Rose	Rehabilitation Serv.	C.C.
[illegible] Topazian	Welfare	CC
[illegible] Cervantes	C.C. Juvenile Court	Las Vegas
Shay Porter	intern	

EXHIBIT A

PROPOSED AMENDMENTS TO:
NEVADA S.B. 459

PROPOSED AMENDMENTS

(In the material below dashes (---) indicate those words to be deleted from the bill. Underscoring indicates language to be added to the bill.)

1) Delete Section 2(1), (2) and (3) on page 1, lines 1 through 25 in their entirety.

2) Amend Section 3(1) on page 2, lines 1 through 5 as follows:

"~~Sec. 3 (1)~~ Sec. 2(1) Any person desiring to engage in the retail sale of ~~convenience drugs~~ non-narcotic, non-prescription drugs which are prepackaged, fully prepared by the manufacturer and labeled in accordance with federal law and the law of this state shall obtain a retail dealer permit from the board. The application shall be accompanied by the fee fixed by the board. Drugs covered by this permit shall not include:

(a) Any controlled substance
(b) Any drug, the label of which is required to bear a statement substantially reading 'Caution: Federal law prohibits dispensing without a prescription.'
(c) Any drug intended for human use by hypodermic injection."

3) Amend Section 3(2) on page 2, lines 5 through 7 to read as follows:

"~~Sec. 3 (2)~~ Sec. 2(2) The retail dealer permit authorizes the holder to stock, display, offer for sale and sell at retail ~~convenience drugs in their original unopened packages, subject to such reasonable regulations as the board may adopt,~~ such drugs that are provided in Sec. 2(1) of this chapter."

4) On line 10, page 2, renumber Sec. 4 as Sec. 3.

5) On line 19, page 2, renumber Sec. 5 as Sec. 4.

6) On line 32, page 2, renumber Sec. 6 as Sec. 5.

EXHIBIT B

7) Amend Section 6(3) on page 2, lines 45 through 48 as follows:

"~~Sec. 6(3)~~ Sec. 5(3) A person selling ~~convenience drugs at retail in their original unopened packages~~ such drugs as provided in Section 2(1) of this chapter need not be a registered pharmacists under the provisions of this chapter, but no person may sell ~~convenience drugs~~ such drugs unless he has first obtained a retail dealer permit from the board."

8) On line 13, page 3, renumber Sec. 7 as Sec. 6.

9) Then amend what is now Section 7, page 3, line 31 to read as follows:

"For issuance of ~~retailer's convenience drug~~ retail dealer permit . . .25"

10) Amend NRS 639.073 by adding the following:

"1. If the public interest would best be served, the board may adopt regulations restricting the sale of drugs to sale by or under the direct supervision of a registered pharmacist; provided, however, that nothing shall prevent the retail sale by the holder of a retail dealer permit issued by the board of drugs as provided in Sec. 2(1) of this chapter."

April 15, 1975

SECTION 1. NRS 482.384 is hereby amended to read as follows:

482.384 1. The department may issue [a special parking permit or] special plates:

(a) To any person [holding a valid driver's license issued pursuant to chapter 483 of NRS] who owns a motor vehicle, other than a commercial vehicle, who has a permanent physical handicap which impairs his mobility when not in a motor vehicle.

2. The department may issue a special parking permit:

[(b)] (a) To any person who:

[(1) Does not hold a valid driver's license ; or]

[(2)] (1) Owns or does not own a motor vehicle; and

[(3)] (2) Has a permanent physical handicap which impairs his driving ability and impairs his mobility when not in a motor vehicle; and

[(4)] (3) Has need to be driven by another person to a destination in a motor vehicle.

3. The department may make such rules and regulations as are necessary to ascertain eligibility for such special parking permits and special plates.

4. Applications for special parking permits or special plates for physically handicapped persons shall be made to the department on forms provided by the department which shall require information necessary to determine the applicant's eligibility for a permit or special plates for physically handicapped persons and shall be accompanied by a certificate from a licensed physician describing the character and extent of the the applicant's disability.

5. Physically handicapped persons shall pay the regular motor vehicle registration fee as prescribed by this chapter. No additional fee

may be charged for special parking permits or special plates.

6. Only one special parking permit or one set of special plates for physically handicapped persons may be issued to any eligible applicant in any one registration period.

7. Each set of special plates for physically handicapped persons issued pursuant to this section shall expire at the end of the last registration month of the registration period for which it was issued.

8. Permits or special plates shall not:

[illegible] Authorize parking in any area on a highway where parking is prohibited by law.

9. Special plates issued pursuant to this section shall be of a design determined by the department.

10. No person, other than the physically handicapped person or person actually transporting a handicapped person shall be entitled to use the handicapped plates or permits.

SEC. 2. NRS 484.407 is hereby amended to read as follows:

484.407 1. Except as provided in subsection 2, owners of motor vehicles displaying a special parking permit or special plates for physically handicapped persons issued pursuant to NRS 482.384 may park such motor vehicles for not more than 4 hours at any one time in parking zones restricted as to the length of time parking is permitted, without penalty, removal or impoundment of such vehicle if such parking is otherwise consistent with public safety[.] and being used by a physically handicapped person or used by a person transporting a physical handicapped person.

2. This section does not authorize the parking of a motor vehicle in any privately or municipally owned facility for off-highway parking without paying the required fee for the time during which such vehicle is so parked.

SMOKING AND THE NONSMOKER

My purpose in appearing before you today is to provide you with some perspective about the "rights" of nonsmokers and smokers. I am not a doctor or a scientist but I have had an opportunity to become acquainted with the dispute. I have gained a fairly good idea of just what evidence there is -- and more importantly -- what there is not.

I will briefly present some of the facts in this controversy. You may be assured that they are completely and accurately documented. I'll also try to answer whatever questions you may have. You may well come up with some tough ones that I can't give you a definitive answer to off the top of my head. If that happens, I'll give you the best information I have and then check with persons who are experts and get you the rest of the information as soon as possible.

I don't think that it's really necessary, anyway, that one be a scientist or a doctor to understand what's involved in this sort of controversy. What we're faced with is a situation in which one group of persons, without any good scientific evidence to support their position, is trying to make illegal a widespread and long standing social practice of another group of people that they find annoying. Their position is nominally based

upon the argument that smoking in public places is actually hazardous to the health of other persons, of nonsmokers, and that therefore smoking in public should be banned. But this is only their stated reason -- medical and scientific evidence does not warrant the conclusion that cigarette smoking under normal conditions is hazardous to the health of nonsmokers. Their real motivation is simply that they don't like smoking -- it annoys them. Furthermore, a lot of them would like to see smokers so mini-prohibited they would quit -- the "I know what's good for you" approach.

It might be helpful to briefly review some of the history of this dispute so that you can see how recent it is and how little support there is for any claim of medical hazards to nonsmokers.

The whole smoking and health issue as it relates to the active smoker -- the one who smokes himself -- really first became subject to general, public controversy in 1964 when an advisory committee composed of scientists issued its famous report to the U.S. Surgeon General.[1] This controversy continues. Since the initial 1964 Report (the anti-smoking propaganda arm of the Public Health Service) -- the National Clearinghouse for Smoking and Health, prepared six more reports -- these came out in 1967, 1968, 1969, 1971, 1972, and 1973. Not until 1972 was any mention made about smoking being a possible hazard to the health of nonsmokers.[2]

All the others made no such claims whatsoever. And the 1973 Report was also silent on the subject.[3]

In Great Britain, the Royal College of Physicians has issued two reports on smoking and health. The first of these came out in 1962 [4] and the second in 1971 [5]. Neither of the two reports treated cigarette smoke as a health hazard to nonsmokers.

It is interesting that the claims made in the 1972 Surgeon General's Report also contradict statements of other U.S. Government agencies. I would like to quote for you from a publication put out by the U.S. Department of Health, Education and Welfare: "Smoking, Health & You"

> "Can it harm you to breathe the smoke from other people's cigarettes?
>
> "No. It may make your eyes tear or make you cough a bit; but it cannot harm you." [6]

Even the U.S. Surgeon General admitted after the 1972 report was issued that he could not "say with certainty that exposure to tobacco smoke is causing serious illness in nonsmokers". He continued by saying that "the long term research necessary for such a finding has not yet been done."[7] Now Jesse Steinfeld, who was the Surgeon General who made that statement in 1972, certainly was no friend of cigarette smoking; yet even he had to admit a lack of certainty on this question.

Let's look for a minute at some of the so-called "evidence" used by the persons who want to prohibit other people from smoking in public. They throw out figures about astronomical amounts of tobacco being burned annually and call that "a major pollutant in our environment".[8] They complain about the carbon monoxide in tobacco smoke as harming the nonsmoker.[9] What they don't mention is that a study published in 1970 by the New York Academy of Sciences found that cigarette smoke contributed a "negligible" portion of the carbon monoxide found in the air we breathe.[10] Let me put it another way -- the study determined that cigarette smoke contributed less than one ten thousandth of the carbon monoxide in our air. Motor vehicles caused more than 5,900 times as much carbon monoxide as cigarettes, and even forest fires produced more than 700 times as much.

The kind of extreme experiment that some opponents of cigarette smoking like to cite is one in which a group of people is put into a cramped, unventilated space while they smoke as many cigarettes as fast as they possibly can. Let me give you an example of an unrealistic study which has been used to support the claim that smoking in automobiles is hazardous to non-smoking passengers. In 1967, a (Czechoslovakian) scientist reported that he had put four people inside a small European car with its doors and windows closed inside an enclosed garage.[11] Not even the wind was allowed to hit the car. The two smokers each smoked five cigarettes in sixty-two minutes, smoking them to an extremely

small butt length -- one fifth of an inch. Only under these exaggerated conditions was an elevated carbon monoxide level reported. In such an airtight space, I'm sure everyone was uncomfortable, smoker and nonsmoker alike. Their normal reactions would have been to roll down the windows, or stop smoking, or both. I don't think we really need a law telling people that if there are four people in their Volkswagon it's unlawful for them to drive it into a garage, roll up the windows, shut the garage doors, and sit there for an hour while smoking a half a pack of cigarettes.

I won't belabor this point. I do think it is important to realize, however, that the question you face is not completely unique. Several government agencies, both federal and state, have decided precisely this question based on extensive expert evidence by doctors and scientists. Let me read you the conclusion of an 85-page study of cigarette smoking in aircraft conducted jointly by the Federal Aviation Administration, the Department of Health, Education and Welfare, and the Department of Transportation. The report, which was issued in December 1971, states as follows:

> ". . . it is concluded that inhalation of the by-products from tobacco smoke generated as a result of passenger smoking aboard commercial aircraft does not represent a significant health hazard to nonsmoking passengers."[12]

The Federal Interstate Commerce Commission also conducted an extensive study in 1971 of smoking on buses. The Commission's

conclusion is as follows:

> "We agree with the examiner's conclusions that petitioner has failed adequately to demonstrate the deleterious effects of second-hand smoke upon the health of motor bus passengers."[13]

The California Public Utilities Commission has also studied the problem of smoking on buses. This is the conclusion of THE Commission:

> "It is traditional that an individual's freedom of choice should be preserved, where no serious problem is created for others. The smoke[r] is usually less of a bother than the alcoholic, one who chews tobacco or garlic, or the compulsive talker. . . .
>
> "The nonsmoker will suffer some discomfort when exposed to concentrated cigarette smoke in an enclosed area, but there is no proof that his health is impaired thereby."[14]

These findings by government agencies that have considered all the evidence are not surprising. They are based on solid scientific evidence provided by scientists from all over the world -- studies for example by Yaglou (an American)[15]; Eckardt and MacFarland (an American and a Canadian)[16]; Bridge and Corn (Americans)[17]; Harke (a German)[18]; and Anderson and Dalhamn (Swedes)[19]. The American study by Bridge and Corn concluded this way:

> ". . . our results suggest that concentrations of CO [carbon monoxide] from cigarette and cigar smoking do not present an inhalation hazard to nonsmokers."[20]

And a recent review of the literature by another scientist (Schievelbein) has concluded that:

> "No proof of a threat to the health of nonsmokers through 'passive smoking' can be found in studies available to date."[21]

To add a little more perspective on this matter, it is interesting to note that even some of the most outspoken anti-tobacco critics, such as the British organization, Action on Smoking and Health, have admitted that "[t]here is no evidence that other people's smoke is dangerous to healthy non-smokers. . . ."[22]

One of the easiest ways of showing how extremely unlikely it is that so-called "passive smoking" is harmful is to consider the pipe smoker. Not only is the pipe smoker an active smoker, but we also know from experience that he is one of the greatest "passive" smokers around -- he is constantly enveloped in a wreath of pipe smoke; and pipe smoke -- the Surgeon General's Committee told us in 1964 -- has almost ten times the benzypyrene content of cigarette smoke.[23] Yet, according to the 1964 Report to the Surgeon General, the mortality rates for pipe smokers are "little if at all higher than for non-smokers, even with men smoking ten or more pipefuls per day and with men who had smoked pipes for more than thirty years."[24] The 1964 Report further makes clear that this is true even among pipe smokers who inhale.[25]

So, the claims that tobacco smoking is hazardous to the non-smoker are not justified by the scientific evidence. These claims are merely a facade disguising what is an attempt by one group of persons to write their personal prejudices into law. Granted that tobacco smoke may be annoying to some people -- this does not make it a proper subject for legislation. The answer, it seems to me, is that both smokers and nonsmokers should be sensitive to the rights and wishes of each other. This is the way the problem has been handled in the past and, overall, this approach has been pretty successful.. Unfortunately, we're now in a situation in which some nonsmokers have abandoned any attempt to understand or respect the wishes of smokers. They are now trying to attach a criminal label to behavior which does not conform with their own personal desires. But, as the government's top physician, Assistant HEW Secretary Merlin K. DuVal, said to a Congressional Committee not long ago when asked about government restrictions on smoking:

> "I would submit that at this time this is an area of individual rights . . . . It would seem to me that there is no way in which there could be a proper governmental intrusion . . . ."[26]

In conclusion, I can do no better than to read you what Dr. Paul B. McCleave, the Director of the Department of Medicine and Religion of the American Medical Association, has said about the dangers of this kind of activity:

"As is always the case in any group that becomes anti of any situation or circumstance, there are always loud voices and much flag waving. So it is in the anti-smoking group. Public travel is public and not a private individual's right. What my seatmate may do, and my reaction to his acts, I must accept as one who is in public transportation.

". . . smoking may be offensive to certain people but so is an alcoholic breath, a sweating body, an unkempt figure, a crying baby, or an undisciplined child on an airplane. May I ask, as one who travels over 100,000 miles a year on planes, that if you ban smoking then will you ban these other annoyances and inconveniences to one who travels?"[27]

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24. Ibid., p. 36.

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26. DuVal, M. Testimony before Consumer Subcommittee of U.S. Senate Committee on Commerce, February 1, 1972. Page 28.

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February 28, 1975

Frank J. Fahrenkopf, Jr., Esq.
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Reno, Nevada 89504

Dear Frank:

RE: Nevada Division, American Cancer Society stand on legislation concerning smoking in public places.

It must first be understood that the American Cancer Society is absolutely opposed to smoking in any form because it may be harmful to one's health.

The American Cancer Society encourages establishments to set aside no smoking areas in public places, businesses, etc.

The Board did not feel it could approve the bill in its present form because it would be essentially uninforcible. The American Cancer Society board would support a joint legislative resolution encouraging establishments in Nevada to set aside no smoking areas.

This action was taken in the Executive Committee Meeting, February 6, 1975, in Las Vegas.

Sincerely,

Gary W. Davis
Executive Vice President

EXHIBIT D

R-J viewpoint

Public smoking ban proposal costly issue?

The banning of smoking in certain public places being considered by the State Assembly is a dangerous proposal which could have a serious detrimental effect on the state economy.

Surely the legislators in their own smoke filled rooms must have forgotten the indulgences which keep our economy thriving when they came up with the measure to tell people when and where they could and could not smoke.

The most restrictive of two bills authored by Assemblyman John Vergiels and others would prohibit the smoking of tobacco in any form in any "elevator, indoor theater, library, art museum, lecture or concert hall, department store, restaurant or bus which is used by or open to the public."

Smoking would further be prohibited in any "room in a public building while a meeting open to the general public is in progress." Doctors offices would also be off limits to smokers.

The prohibition of smoking would work just about as effectively as the prohibition of drinking did a generation ago. The law would prove unenforceable unless Vergiels and his colleagues intend to establish a whole new vice squad to run about extinguishing the outlawed cigarettes of knowing or unknowing offenders.

Visitors to our town, who came in search of a little enjoyment, would have to be told as they entered restaurants and convention sessions, that they would instead be faced with a little discomfort by foregoing the pleasure of smoking.

The law would cause more than a small annoyance for the tourists who would not be accustomed to such restrictions in their own communities. Many would leave with an unpleasant irritation which might keep them from coming back for another visit.

We agree with the Las Vegas Convention and Visitors Authority when they stated, "Prohibiting smoking in public areas and partitioning smokers from non-smokers would both destroy our image as a sun and fun resort and severely cripple our ability to solicit conventions."

The law also would cause numerous inconveniences for our own residents and for the many businesses which would have to comply with the restrictions.

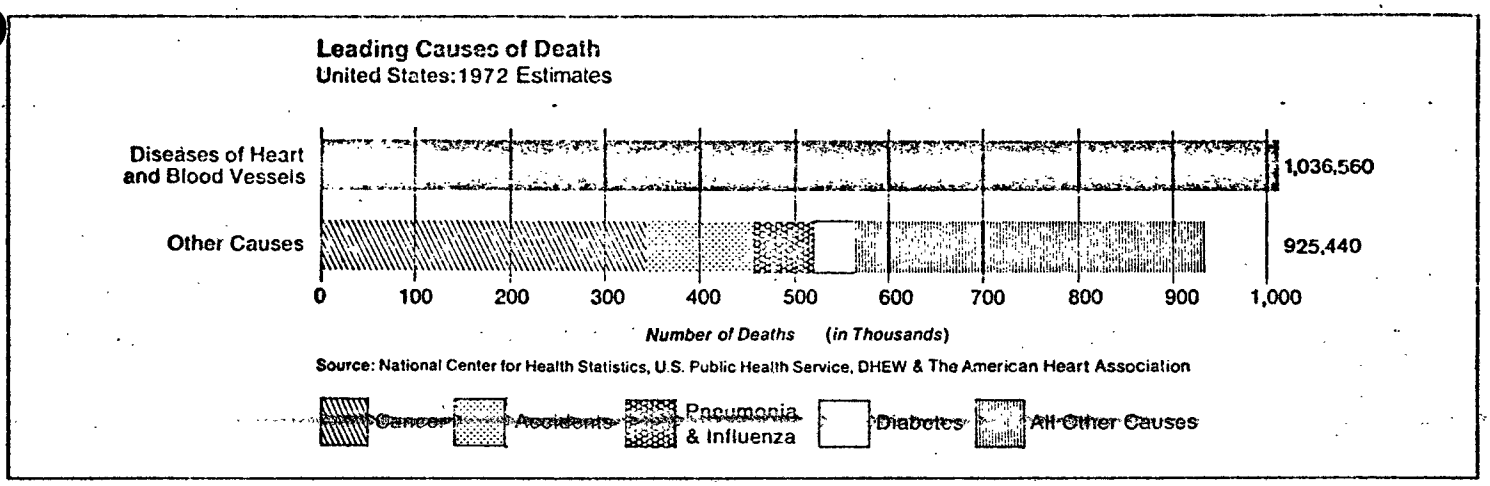
The proposed legislation allows for separate smoking areas "where it is possible to confine the smoke to such areas."

Proponents of the bill argue that provision allows for the accommodation of smokers. What they overlook is the costly remodeling it would require of restaurants and convention centers. Many establishments would not be able to provide separate smoking and non-smoking sections without severely limiting their available seating space or destroying their present decor.

The restrictive proposals now under discussion by the Assembly Health and Welfare Committee are ones which should be allowed to go up in smoke for the welfare of the whole community.

EXHIBIT E

STILL NUMBER ONE



You can help us be number two, or number three, or number four, or r

THE COST OF LIVES:

More than one million persons in the U.S. died from heart and blood vessel diseases in 1972. This equals 53% of all deaths in the nation—more than cancer, accidents and all other causes combined. Of these, heart attack accounted for more than 683,000 deaths, and stroke for more than 210,000 deaths.

Twenty-eight million Americans have one or more heart and blood vessel diseases. Chief among these are high blood pressure, heart attack, stroke, rheumatic heart disease and congenital heart defects.

THE COST IN DOLLARS:

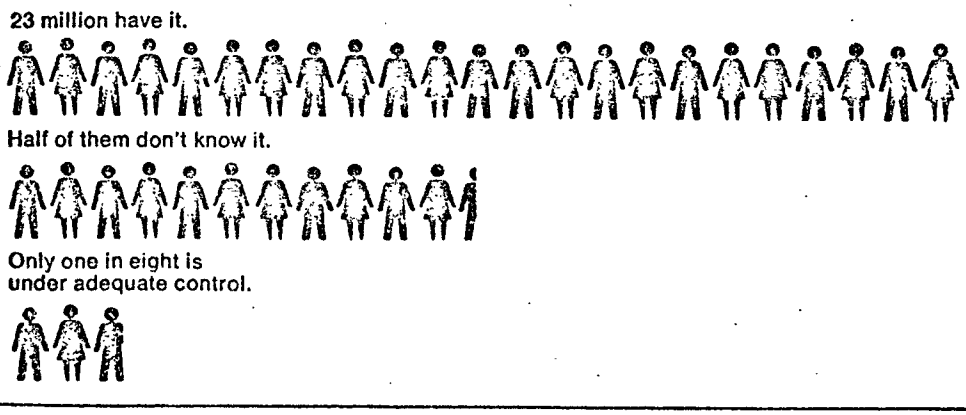
Heart and blood vessel diseases cost the nation an estimated $20 billion a year in lost income and payments for medical care. In addition, industry and the nation lose 52 million man-days of production each year. Especially costly to industry are deaths in the 45-64 age group, within which the loss of management skills, production "know how" and the cost of training replacement personnel can be critically important. Heart attack is the leading killer of people aged 45-64 and stroke ranks third. Together they account for 39% of all deaths in this key employee group.

WHAT WE'RE DOING ABOUT IT:

- We're investing millions of dollars in research each year to find the causes of heart disease and to save lives through advances in diagnosis, treatment, surgery and prevention.

- We're carrying out a nationwide campaign to teach the early warning signs of heart attack and stroke and to help people reduce their risk of these two killers.

- We're working to make every American aware that high blood pressure is a silent killer—that it *can* and *must* be detected and controlled.



Some 23 million Americans have high blood pressure. Half of them don't know it because it has no symptoms. Of those who have it, only one in eight has the disease under adequate control. Left untreated, high blood pressure can result in stroke, heart and kidney failure or heart attack. High blood pressure is easily detected by a simple test and can usually be controlled. To be sure, have your blood pressure checked and follow your doctor's advice.

Continuing research and programs aimed at prevention will help save more lives.

This is the goal of

YOUR HEART ASSOCIA

EXHIBIT F-1

04-5520 350M PHE

Report of Inter-Society Commission for Heart Disease Resources *

PLEASE ADDRESS CORRESPONDENCE TO:

Irving S. Wright, M.D., *National Chairman*

or

Donald T. Fredrickson, M.D., *Project Director*

Inter-Society Commission for Heart Disease Resources
301 East 64th Street—Suite 6B
New York, New York 10021

* *organization created to implement a contract between Regional Medical Programs Service and the American Association to help fulfill the requirements of Section 907 of Public Law 89-239 which established the Regional Medical Programs in 1965. The purpose of the contract and the Commission is to establish guidelines for medical facilities in the prevention, treatment, and rehabilitation of patients with cardiovascular diseases.*

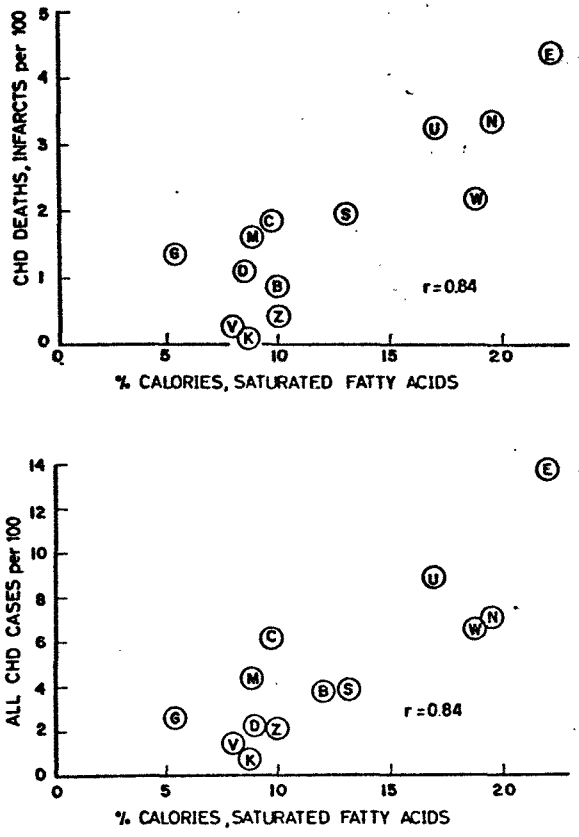


Figure 7

International cooperative study on Epidemiology of Cardiovascular Disease; men originally age 40-59 in seven countries; age-standardized 5-year incidence rates for fatal CHD plus non-fatal MI (upper figure) and for all CHD (lower figure) among men CHD-free at entry, plotted against percentage of total calories provided by saturated fatty acids in the diet; for identification of the cohorts, see legend for Figure 5 (40).

of hyperlipidemia. This conclusion is supported by impressive clinical and experimental data as well as by prospective epidemiologic findings demonstrating significant correlations between blood pressure and the subsequent development of CHD (figs. 9, 10).[6, 6a-k] This relationship between blood pressure and CHD risk is continuous. At each higher step of the blood pressure scale risk is increased. Hypertension has also been established as a major risk factor for cerebrovascular disease, including atherothrombotic cerebral infarction and cerebral hemorrhage (fig. 9).[6, 6a-k]

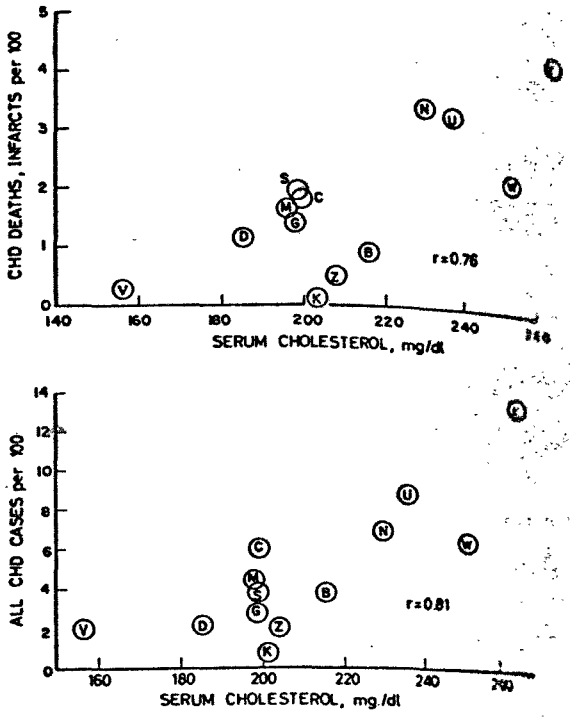
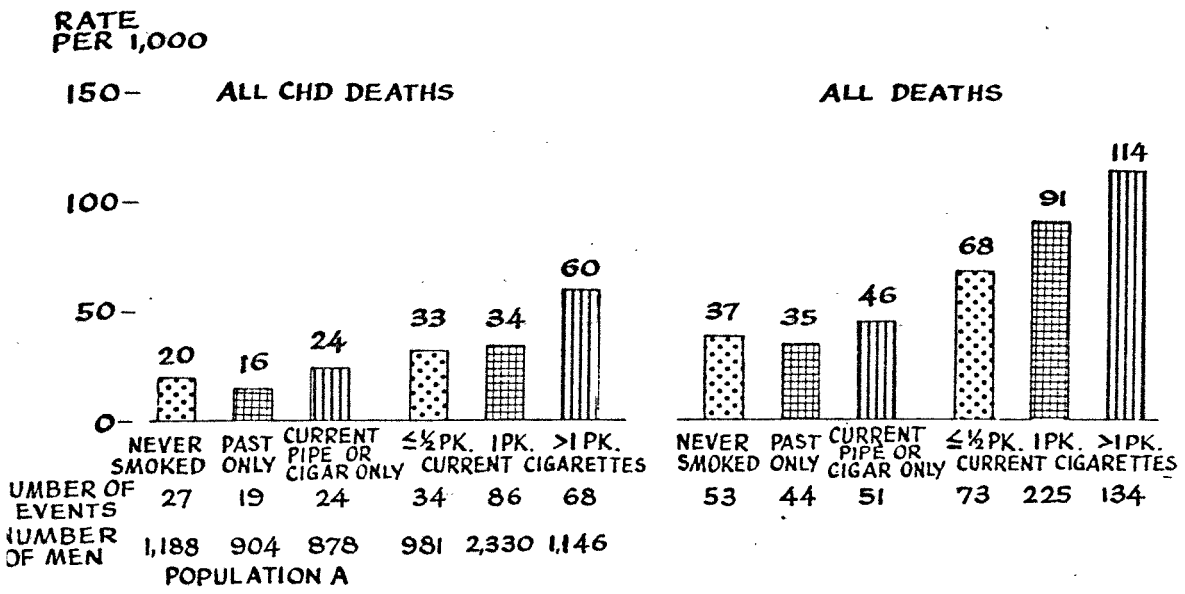


Figure 8

International cooperative study on Epidemiology of Cardiovascular Disease; men originally age 40-59 in seven countries; age-standardized 5-year incidence rates for fatal CHD plus non-fatal MI (upper figure) and for all CHD (lower figure) among men CHD-free at entry, plotted against median serum cholesterol concentrations; for identification of the cohorts, see legend for Figure 5 (40).

Cigarette Smoking

The 1964 Surgeon General's report on cigarette smoking established that on the average cigarette smokers in the United States have a 70 per cent greater chance of developing CHD than non-smokers.[42] Recent data from several prospective studies in this country have extended and strengthened knowledge on the association between smoking and atherosclerotic diseases.[6, 6a-k, 43-45] The largest of these has involved one million men and women originally age 40 to 84. Data are available after three and six years of follow-up (tables 6, 7).[46, 47] They show that for each sex and age group CHD mortality increased with increased intensity of cigarette smoking. The youngest men smoking two or more packs of cigarettes a day were at highest risk. Risk of cerebrovascular disease was also greater for



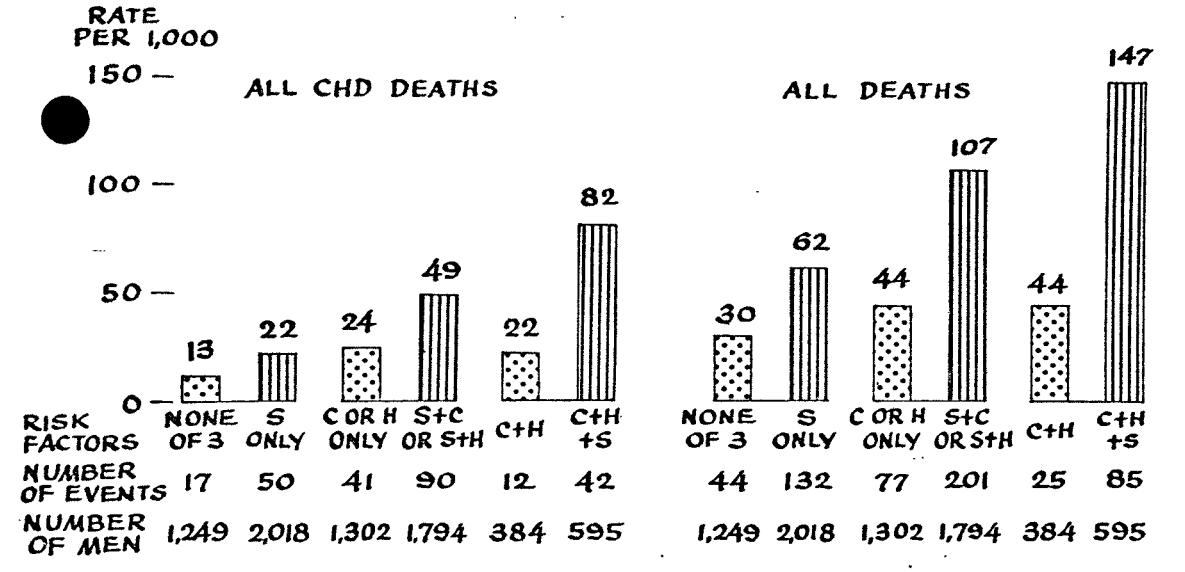


Figure 10 (continued)

ıe age group, the higher the relative sk associated with cigarette smoking (table).[6, 6a–k, 47, 48] It has also been demonstrated ıat the association between cigarette smok-ıg and CHD risk is independent of such ther risk factors as serum cholesterol level nd blood pressure (fig. 10).[6, 6a–k, 49, 50] In ddition, three studies have recently shown ıat erosclerosis of aorta and/or coronary rter is more severe at autopsy in persons ho been habitual cigarette smokers prior to death compared to those who had never smoked (fig. 11).[51–54]

Finally, as the annual supplements to the Surgeon General's report have noted, research progress has been recorded in elucidating possible mechanisms whereby smoking may exert its deleterious effect on the atherogenic process.[43–45]

Combinations of Major Risk Factors

Morbidity and mortality rates from CHD among Americans living in the same commu-

Table 6

Coronary Heart Disease Mortality Ratios among Current Cigarette Smokers Only, by Amount Smoked Daily—American Cancer Society Study of One Million Men and Women (46)

Age and sex	Non-smokers	Cigarettes smoked daily			
		Under 10	10-19	20-39	40+
Men:					
45 to 54	1.0	2.4	3.1	3.1	3.4
55 to 64	1.0	1.5	1.9	2.0	2.1
65 to 74	1.0	1.3	1.6	1.6	*
75 to 84	1.0	1.2	1.4	1.1	—
Women:					
45 to 54	1.0	0.9	2.0	2.7	—
55 to 64	1.0	1.3	1.6	2.0	—
65 to 74	1.0	1.1	1.4	1.9	—
75 to 84	1.0	—	—	—	—

*Expected deaths were less than 10.

nities differ markedly particularly when classified with respect to all three of the foregoing risk factors—*serum cholesterol, blood pressure and cigarette smoking*—considered simultaneously. Detailed data are now available from the pooled findings of six major U. S. prospective studies dealing with several thousand middle-aged American men free of clinical CHD at initial examination (fig. 12).[6, 6a-k] Those free of the three risk factors—hypercholesterolemia, cigarette smoking and hypertension—experienced much lower CHD morbidity and mortality rates over a ten-year period than did the groups of men with any two or all three of these traits. CHD mortality rate was one-third to one-sixth as high and the sudden death rate was one-fourth to one-sixth as high. As a result of the low mortality rate from atherosclerotic diseases, the men free of these major risk factors had less than one-third the ten-year mortality rate from all causes than the men with two of these traits, and about one-fifth the total mortality rate of those with all three risk factors.

These impressive findings indicate that these three risk factors—hypercholesterolemia, hypertension and cigarette smoking—are properly designated major risk factors for premature atherosclerotic disease, especially coronary heart disease. This designation is

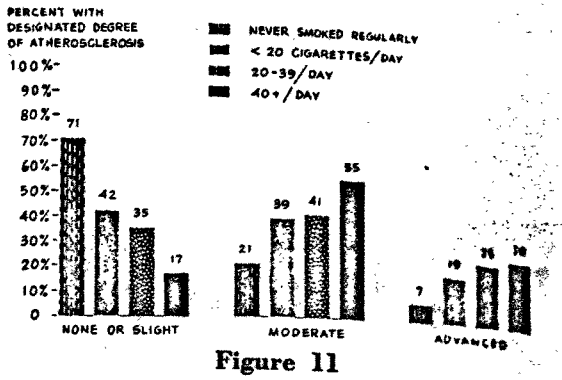


Figure 11

East Orange, New Jersey Veterans Administration Hospital study; degree of coronary atherosclerosis at autopsy, men age 45-59 at death; men who had never smoked regularly and those who had been current cigarette smokers prior to death (51, 54).

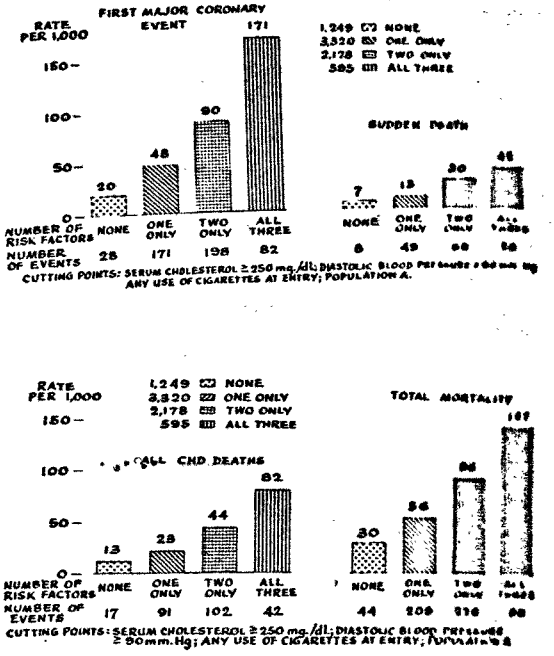


Figure 12

National cooperative Pooling Project; hypercholesterolemia, hypertension, cigarette smoking and 10-year age-adjusted rates per 1,000 men for: any major coronary event, sudden death (upper graph), any coronary death, death from all causes (lower graph); any major coronary event includes nonfatal MI, fatal MI, sudden death due to CHD; U. S. white males, age 30-59 at entry; all rates age-adjusted by 10-year age groups to the U. S. white male population, 1960 (6, 6a-k).

appropriate, first because of the impact of these factors on risk, particularly when present

—e.g., college and university home economics departments, hospital dietitian instruction programs, schools of medicine, dentistry and nursing, and teachers' colleges. These sources should develop educational programs based on modern concepts of sound nutrition. Succeeding generations should have the advantage of this knowledge beginning in elementary school.

Food manufacturers have an excellent opportunity to provide public education through advertising. They should be encouraged to call attention in their advertising to the type and amount of fat and the cholesterol content of their products.

There is a great need for extensive and continuous dissemination by the news media of information on diet, as well as other risk factors. Public service communications in this area should be substantially strengthened and broadened.

With proper education, information and the availability of fat modified foods, it will be possible for most Americans to make desirable changes in their diets without major dislocation of personal eating habits.

Americans should be encouraged to modify habits with regard to all five major sources of fat in the U. S. diet—meats, dairy products, baked goods, eggs, table and cooking fats. Specifically a superior pattern of nutrient intake can be achieved by altering habits along the following lines:

. . . Use lean cuts of beef, lamb, pork and veal, cooked to dispose of saturated fat and eaten in moderate portion sizes;
. . . Use lean meat of poultry and fish;
. . . Use fat-modified,* processed meat products (frankfurters, sausage, salami, etc.);
. . . Use organ meats (e.g., liver) and shellfish in moderation since they are higher in cholesterol than muscle of red meat, chicken and fish;
. . . Avoid fat cuts of meat, addition of saturated fat in cooking meat, large meat portions and processed meats high in saturated fat;
. . . Use low fat and fat modified dairy products;
. . . Avoid high saturated fat dairy products;
. . . Use fat modified baked goods (pies, cookies, cakes, sweet rolls, doughnuts, crullers);
. . . Avoid baked goods high in saturated fat and cholesterol;
. . . Use salad and cooking oils, new soft margarines and shortenings low in saturated fat;
. . . Avoid butter, margarine and shortenings high in saturated fat;
. . . Avoid candies high in saturated fat;
. . . Avoid egg yolk, bacon, lard, suet;
. . . Use grains, fruits, vegetables, legumes.

Elimination of Cigarette Smoking

D. *The Commission recommends that high priority be given to the elimination of cigarette smoking as a national habit.*

Advertising and Sales

1. *Efforts should be made to reduce smoking among young people by strict restraints on advertising and the sale of cigarettes.* All advertising of tobacco in the mass media (including television, radio, newspapers and magazines) should be discontinued. Short of this all advertising should carry an honest, frank, highly visible warning for potential consumers.

Mass Media Education

2. *The mass media education program emphasizing the health hazards of smoking should be continued indefinitely to redress the imbalance created by decades of cigarette advertising.*

School Education

3. *Education programs on the risks of smoking should be strengthened and extended throughout the school system beginning with the early primary grades.* Parents, teachers, health professionals and other adults in

*Throughout this set of guidelines *fat modified* refers to products made with reduced saturated fat and cholesterol content.

positions of responsibility (e.g., television entertainers and sports personalities) should be made aware of the serious adverse influence of their own smoking habit as a poor example for children who may become lifelong cigarette smokers. It is noteworthy that physicians have been particularly successful in giving up cigarette smoking and are in a unique position to exert great influence in helping their patients stop smoking.

Vending Machines

4. *Cigarette vending machines should be removed from all medical facilities and public buildings or, preferably, banned altogether.*

Public Facilities

5. *The prohibition against smoking in large meetings and mass transit facilities should be vigorously enforced.*

Use of Tax Funds

6. *Revenues from progressive increases in taxes on tobacco should be earmarked for smoking control programs and the care of patients with diseases associated with smoking.*

Subsidies

7. *Current large subsidies by government for growing and exporting tobacco should be critically reviewed with the objective of making economic supports for agriculture consonant with national health goals.*

Phase Out of Cigarette Industry

8. *Planning by appropriate social science experts should go forward for the orderly phase out of the cigarette industry without major economic dislocation of those whose livelihood is involved.*

Detection and Control of Hypertension

E. *The Commission recommends a major national effort to detect and control hypertension.* Recent studies have shown that the prevalence of elevated blood pressure is generally high in the United States, especially in the black population (table 17).[99] Many hypertensives have not been identified; many others known to have the disease are not receiving adequate therapy. Programs are urgently needed to identify hypertensives in the community and assure their subsequent treatment. The recently published positive results from the Veterans Administration field trial of drug therapy for so-called "mild" hypertension underscore the potential significance of such programs.[78]

Community Programs

F. *The Commission recommends that community programs be developed and expanded for the detection and treatment of persons of all ages who are very susceptible to premature atherosclerotic diseases due to combinations of the major risk factors.*

This recommendation is premised on extensive experience demonstrating that effective community programs for prevention of disease generally combine measures addressed to the entire population with concerted efforts for the detection and care of high risk individuals. All available evidence indicates that this well-established principle applies to the prevention of the atherosclerotic diseases.

On the basis of recent experience, detection programs are likely to identify a very large proportion of the population—e.g., about 20 or 30 per cent of middle-aged adults—as being at unusually high risk. For such individuals, community services should be provided to assist their physicians in long-term management.* Such programs will require the training and use of large numbers of allied health personnel, as well as physicians.

Drug Treatment of Hyperlipidemia; Exercise Programs

G. *The Commission presents the following observations on drug treatment of hyperlipidemia and on exercise programs, and their possible role in the preventive effort.*

Drugs for the treatment of hyperlipidemia have been developed in recent years. For example, several years of experience with cholestyramine, clofibrate, dextrothyroxine and nicotinic acid have demonstrated that

*Detailed proposals concerning these community services will be presented in subsequent reports of the Commission.

THE HEALTH CONSEQUENCES OF SMOKING

JANUARY 1974

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service

SUMMARY OF RECENT FINDINGS ON THE RELATIONSHIP OF SMOKING AND CANCER

1. Recent epidemiologic evidence confirms the finding that cigarette smoking is the major cause of lung cancer for both men and women.
2. Current evidence suggests that, even in the presence of a possible genetic susceptibility to the development of lung cancer, cigarette smoking remains the major cause of lung cancer.
3. Results from several studies demonstrated a dose-response relationship between smoking and oat cell carcinoma; a major prospective study demonstrated such a relationship for well-differentiated squamous cell carcinoma, oat cell carcinoma, and adenocarcinoma.
4. The current epidemiologic data suggest that the incidence of lung cancer in women continues to rise. The rising incidence of lung cancer in women correlates well with the increasing trends in smoking among women.
5. Present data are conflicting with regard to dose-response relationships for cigar and pipe smokers and the development of lung cancer; the data are consistent for the fact that light cigar smokers are at a low risk of developing lung cancer.
6. Recent data confirm the synergistic effect of asbestos and smoking exposure on the risk of developing lung cancer in both men and women.
7. Results from experimental studies in hamsters continue to demonstrate that exposure to benzo(a)pyrene results in the production of respiratory tract malignancies, especially squamous cell carcinomas.
8. Data from experimental studies in animals suggest that chronic respiratory infections may enhance the carcinogenicity of components of cigarette smoke, as may alterations in the immune system.
9. Current evidence suggests that components of cigarette smoke induce AHH activity in pulmonary macrophages in humans and in pulmonary parenchymal tissue and embryo cells in animals. The role of AHH in tumorigenesis and/or as a host defense mechanism against potential carcinogens is presently unclear.
10. Recent epidemiologic data strongly indicate that cigarette smoking plays an independent role in the development of oral cancer.
11. Recent epidemiologic data confirm the association between smoking and pancreatic cancer.

SUMMARY OF RECENT CARDIOVASCULAR FINDINGS

1. Data from recent epidemiologic studies suggest that cigarett smoking acts independently of and in conjunction with cer tain cardiac arrhythmias to increase the risk of mortalit from coronary heart disease in men. Smokers also have greater probability of dying from CHD at an earlier age tha nonsmokers.
2. New epidemiologic data suggest that women who smoke cigar ettes have a greater risk of sudden death from CHD than d nonsmoking women.
3. The results of experimental studies demonstrate that the ele vated levels of carboxyhemoglobin frequently seen in smoker may result in significantly decreased cardiac work perform ance and precipitation of ischemic electrocardiographi changes and arrhythmias in patients with clinical and sub clinical CHD. Carboxyhemoglobin levels may be of value i determining a person's risk of developing arteriosclerotic car diovascular disease.
4. Findings from experimental studies confirm that nicotine act indirectly to cause elevations of plasma FFAs. The relative role of sympathetic versus adrenocortical stimulation of the rise in FFAs remains to be determined.
5. Epidemiologic data reveal strong associations between cigarette smoking and development of peripheral vascular disease.
6. Data from epidemiologic studies support a strong association between atherosclerotic brain infarction and cigarette smoking in premenopausal women and in men of all ages. No association between ABI and smoking has yet been demonstrated in menopausal women.

SUMMARY OF RECENT NON-NEOPLASTIC BRONCHOPULMONARY FINDINGS

1. Results from epidemiologic studies on elderly populations demonstrate an increased prevalence of respiratory symptoms and impairment of pulmonary function among smokers of both sexes compared to nonsmokers.
2. Data from several recent studies indicate that standard pulmonary function tests and physical work capacity are impaired in apparently healthy smokers compared to nonsmokers.
3. Recent epidemiologic data suggest that smokers who retain their cigarettes in their mouths continuously while smoking ("droopers") have a higher prevalence of chronic bronchitis than those smokers who remove the cigarette from their mouths between puffs.
4. A recent epidemiologic study confirms the observation that cigarette smoke and air pollution act synergistically in the development of symptoms of respiratory disease.
5. Results from several recent studies indicate that cigarette smokers have a higher prevalence of functional abnormalities of the small airways than do nonsmokers.
6. Results from a recent study suggest that although a history of lower respiratory disease as an infant is related to the prevalence of cough at age 20, cigarette smoking is a far more important factor in the development of cough in young adulthood.
7. Data from a major retrospective study indicate that cigarette smoking is related to the development of bullous disease of the lung.
8. Experimental studies in animals have shown that exposure to nitrogen dioxide, a constituent of the vapor phase of cigarette smoke, results in emphysema-like changes in the pulmonary parenchyma, diminished mucociliary clearance, and impairment of bactericidal activity of alveolar macrophages.
9. Data from experimental studies have demonstrated that the filtered gas phase of tobacco smoke may effect changes in pulmonary alveolar macrophage metabolism through inhibition of the glycolytic pathway; cigarette smoke may also impair oxygen consumption and pinocytic activity of pulmonary alveolar macrophages.

Contents

LIST OF TABLES

PUBLIC EXPOSURE TO AIR POLLUTION FROM TOBACCO SMOKE

The purpose of this chapter is to summarize the present state of evidence concerning the effects of exposure to an atmosphere containing either tobacco smoke or its constituents. Since the identification of cigarette smoking as a serious health hazard to the smoker was based on clinical and epidemiological observations that nonsmokers have much lower mortality and morbidity rates from a number of conditions, it is obvious that cigarette smoking is normally a greater hazard to the smoker than is the typical level of exposure to air pollutants produced by the smoking of cigarettes which many nonsmokers experience. This would be consistent with the voluminous data which show a dose-response relationship between the level of exposure to smoke and the magnitude of its effect.

The research so far reported on the nature and effects of exposure to smoke-pollutants in the atmosphere has not been as extensive and well-controlled as that done on the health effects of smoking on the smoker himself. Knowledge on this subject can be separated into four major areas of concern:

1. The extent to which the components of cigarette smoke contaminate the atmosphere and are absorbed by the nonsmoker.
2. The effects of low levels of carbon monoxide on human health.
3. Allergic, adverse, and irritative reactions to cigarette smoke among nonsmokers.
4. The known harmful effects of the passive inhalation of cigarette smoke in animals.

THE EXTENT TO WHICH THE COMPONENTS OF CIGARETTE SMOKE CONTAMINATE THE ATMOSPHERE AND ARE ABSORBED BY THE NONSMOKER

Theoretical models of this contamination have been constructed. Owens and Rossano (*44*) have noted that most popular cigarettes release into the atmosphere approximately 70 mg. of dry particulate matter (about 60 mg. in the sidestream and slightly over 20 mg. in the mainstream, about one-half of the latter being absorbed by the smoker and one-half expelled into the ambient air) and 23 mg. car-

bon monoxide per cigarette. This material adds to the cleaning problem of the air of any enclosed space and contributes to residual odors. In a recent study of particulate matter filtration in domestic premises (*35*), the authors observed that the smoking of one cigar completely overcame the effect of an electrostatic filtration device for one hour.

Atmospheric pollutants caused by smoking are derived from two major sources: mainstream and sidestream smoke. Mainstream smoke emerges from the tobacco product through the mouthpiece during puffing, whereas sidestream smoke comes from the burning cone and from the mouthpiece during puff intermissions (*60*). The tobacco smoke released into the atmosphere consists of all the sidestream smoke as well as that part of the mainstream smoke which has been either held in the smoker's mouth or taken into his lungs and then expelled. The actual amount of material to which individuals are exposed in the presence of smokers depends upon the amount of smoke produced, the depth of inhalation on the part of the smoker, the ventilation available for the removal or dispersion of the smoke, and the proximity of the individual to the smoker. The length of time of exposure to those pollutants is extremely important in determining how much is absorbed into the body. The pattern of smoking influences the amount produced by altering the content of the exhaled smoke. As shown by Dalhamn, et al. (*10, 11*), mouth absorption removes approximately 60 percent of the water-soluble volatile components (e.g., acetaldehyde), 20 percent of the nonwater-soluble volatile components (e.g., isoprene), 16 percent of the particulate matter, and only three percent of the carbon monoxide. Thus, the smoker who does not inhale "filters" a portion of the smoke components in his mouth before expelling them into the ambient air. On the other hand, the lungs retain from 86 to 99 percent of the volatile and particulate substances and approximately 54 percent of the carbon monoxide inhaled. Hence, the inhaling smoker "filters" the mainstream smoke rather effectively before expelling it into the ambient air. A factor which has apparently not been investigated is the difference in the smokers' "filtration" of mainstream smoke when the smoke is exhaled through the nose instead of the mouth.

Thus, the nonsmoker breathes smoke-containing air composed of sidestream smoke and mainstream smoke exhaled by smokers. The inhaling smoker receives nearly the full amount of mainstream smoke as well as a portion of sidestream smoke and smoke exhaled by himself and other smokers. The smoker who does not inhale receives those compounds which are absorbed from the mainstream smoke in his mouth, as well as absorbing the sidestream smoke and the smoke exhaled by himself and other smokers contained in the air he breathes.

Since pipe and cigar smokers inhale less commonly than do cigarette smokers, their contribution to the substances in the air breathed in exposure to smoke pollutants consists of a composite of sidestream smoke and relatively unfiltered mainstream smoke which has been held in the mouth and then expelled.

The actual effluents in the mainstream and sidestream cigarette smoke have been considered by Pascasio, et al. (*45*) and Scassellati Sforzolini and colleagues (*50, 51*). These authors stated that "tar" and nicotine levels in sidestream smoke may be significantly higher than those of mainstream smoke and may be harmful to the nonsmoker. Actual volume measurements were not reported, however.

Actual measurements of the contamination due to cigarette smoking have been carried out by a number of research groups. A recent, well-controlled study by Harke (*24*) involved the smoking of 42 cigarettes in 16 to 18 minutes using German blend cigarettes of 85 mm. length, 18 mm. filter, and smoked to a 25 mm. butt length in a room with a volume of 57 cubic meters (approximately the equivalent of a room with a 10-foot ceiling and dimensions of 12 by 14 feet). The author observed that in the absence of ventilation the atmosphere contained up to 50 p.p.m. carbon monoxide and .57 mg./m.^{3} nicotine. With substantial ventilation, these levels fell significantly (to approximately 10 p.p.m. carbon monoxide and .10 mg./m.^{3} nicotine). He also found that cigar smoke (9 cigars of Clear Sumatra tobacco smoked in 30 to 35 minutes) produced similar amounts of contamination while pipe smoke (3 grams of Navy type medium cut tobacco smoked as eight pipefuls in 35 to 40 minutes) produced much less. Other authors have made similar measurements. Galuskinova (*20*) found that 3,4-benzpyrene levels in a smoky restaurant were from 2.82 to 14.4 mg./100 m.^{3} as compared to outside atmospheric levels of 0.28 to 0.46 mg./100 m.^{3}, although burning of food particles may have contributed to the presence of 3,4-benzpyrene in this setting. Kotin and Falk (*33*) have shown that sidestream cigarette smoke condensate may contain more than three times as much benzo(a)pyrene as mainstream smoke. Srch (*55*) observed that the smoking of 10 cigarettes to a 5 mm. butt length in an enclosed car of 2.09 m.^{3} volume produced carbon monoxide levels up to 90 p.p.m. Lawther and Commins (*34*), working with a ventilated chamber, found levels of up to 20 p.p.m. of carbon monoxide after seven cigarettes were smoked in one hour; however, peaks of up to 90 p.p.m. were recorded at the seat next to the smoker. Coburn, et al. (*9*) recorded levels of 20 p.p.m. of carbon monoxide in a small conference room after 10 cigarettes were "burned." Harmsen and Effenberger (*25*) reported up to 80 p.p.m. of carbon monoxide in an enclosed 98 m.^{3} room (approximately the equivalent of a room with a 10-foot ceiling and dimensions of 18 by 20 feet) in which 62 cigarettes had been smoked in two hours.

TABLE 1.—*Percent of COHb during and following exposure to 50 p.p.m. of CO.*

Time during exposure	Mean	Range	Number of subjects
Preexposure	0.7	0.4–1.5	11
30 minutes	1.3	1.3	3
1 hour	2.1	1.9–2.7	11
3 hours	3.8	3.6–4.2	10
6 hours	5.1	4.9–5.5	5
8 hours	5.9	5.4–6.2	5
12 hours	7.0	6.5–7.9	3
15 ½ hours	7.6	7.2–8.2	3
22 hours	8.5	8.1–8.7	3
24 hours	7.9	7.6–8.2	3
Time without exposure after 1 hour of exposure			
30 minutes	1.8	1.8	3
1 hour	1.7	1.6–1.8	3
2 hours	1.5	1.4–1.5	3
5 hours	1.1	1.0–1.1	2
Time without exposure after 3 hours of exposure			
30 minutes	3.7	3.4–3.9	3
1 hour	3.3	2.7–3.8	3
2 hours	2.7	2.3–3.0	3
Time without exposure after 8 hours of exposure			
30 minutes	5.6	5.1–5.9	3
1 hour	5.1	4.8–5.4	3
1 ¾ hours	4.0	—	—
11 hours	1.5	1.4–1.7	3
Time without exposure after 24 hours of exposure			
30 minutes	7.5	7.2–7.8	3
1 hour	6.7	6.4–7.1	3
2 hours	5.8	5.6–6.2	3

SOURCE: Stewart, et al. (56).

Another set of contaminants probably present in a tobacco smoke-polluted atmosphere are the oxides of nitrogen. These, specificially NO and NO_2, have been shown to be present in tobacco smoke although the type most likely to be present in the atmosphere is NO_2. No measurements have been reported of the amount of NO_2 in smoke-filled rooms. The importance of obtaining and evaluating this information is stressed by the results of Freeman and Haydon and

their colleagues (*17, 18, 19, 27, 28*) and of Blair, et al. (*5*) who observed bronchial and pulmonary parenchymal lesions in rodents continuously exposed to low levels of NO_2.

Other experimenters have measured carboxyhemoglobin (COHb) levels in nonsmokers exposed to cigarette smoke pollutants. Srch (*55*) observed that the COHb level in two nonsmokers rose from 2 to 5 percent (that of smokers from 5 to 10 percent) when seated in the cigarette-smoke contaminated car mentioned above (exposure to 90 p.p.m.). Harke (*24*) reported that when seven nonsmokers were exposed for approximately 90 minutes to a "smoked" room containing 30 p.p.m. of CO there was a rise in COHb from a mean of 0.9 percent to 2.0 percent. In 11 smokers subjected to the same conditions, COHb rose from a mean of 3.3 percent to 7.5 percent. With improved ventilation of the experimental room, the COHb level decreased significantly.

The CO exposures and COHb levels reported above closely approximate the results obtained following experimental chamber exposure of humans to various levels of CO. The uptake of CO by the person depends on, among other parameters: CO concentration, previous COHb level, the level of activity, and the person's state of health. Equilibrium between CO concentration in the lung and in the blood requires over 12 hours exposure. However, as may be noted in table 1, reproduced from Stewart, et al. (*56*) and derived from measures of COHb in young sedentary males who were not smoking, over half of the equilibrium COHb level is reached within three to four hours of the onset of exposure. The equilibrium value associated with 100 p.p.m. is approximately 14 to 15 percent COHb. Exposure to 100 p.p.m. in the nonsmoker can lead to 3.0 percent of COHb within 60 minutes and 6.0 percent in two hours (*16*). Of equal significance is that COHb has a half-life of at least three to four hours in the body. As shown in table 1, the COHb level fell only to 2.7 percent in the two hours following cessation of exposure to 50 p.p.m. from the end exposure level of 3.7 percent. This lengthy half-life extends the period of effect of exposure to CO and provides for a buildup of COHb concentration from fresh exposures.

THE EFFECTS OF LOW LEVELS OF CARBON MONOXIDE ON HUMAN HEALTH

The data on the effect of low levels of carbon monoxide on human psychological and physiological function have been summarized in two recent publications (*8, 58*).

There is presently much discussion as to the physiologic and psychophysiologic effects of exposure to levels of CO approximating 50 to 100 p.p.m. Beard and Grandstaff (*4*) observed that exposure to 50 p.p.m. of CO for from 27 to 90 minutes altered auditory dis-

crimination, visual acuity, and the ability to distinguish relative brightness. McFarland (*40*) observed that COHb levels of 4 to 5 percent caused visual threshold impairment. Ray and Rockwell (*48*), reporting on a study of the driving ability of three subjects under varying CO exposure, observed that the presence of 10 percent COHb was associated with increased response time for taillight discrimination and increased variance in distance estimation. Schulte (*52*) observed that increased errors in cognitive and choice discrimination tests were manifest at levels of COHb as low as 3 percent. Chevalier, et al. (*7*) have also observed that levels of 4 percent COHb in nonsmokers are associated with an increase in oxygen debt formation with exercise similar to that seen in smokers.

On the other hand, other investigators utilizing complex psychomotor tasks in men and monkeys have observed no decrement in function upon exposures to CO at 50 to 250 p.p.m. (*2, 3, 23, 41, 56*).

Animals exposed to low levels of CO (50 to 100 p.p.m.) continuously for weeks have shown varying degrees of cardiac and cerebral damage similar to that produced by hypoxia (*21, 47, 57*).

Finally, the possible effects of exposure to 50–100 p.p.m. CO on patients with coronary heart disease (CHD) were investigated by Ayres, et al. (*1*) who observed a decrease in arterial and mixed venous oxygen tensions with COHb saturations of 5 percent. Certain patients with CHD developed altered lactate and pyruvate metabolism with COHb levels of 5 to 10 percent suggesting myocardial hypoxia.

The evidence concerning the effect of low levels of carbon monoxide has recently been reviewed and evaluated by the National Air Quality Criteria Committee of the National Air Pollution Control Administration (*58*). The following is taken from the published conclusions of the Advisory Committee (also see table 2):

> "Experimental exposure of nonsmokers to 58 mg/m³ (50 ppm) for 90 minutes has been associated with impairment in time-interval discrimination.... This exposure will produce an increase of about 2 percent COHb in the blood. This same increase in blood COHb will occur with continuous exposure to 12 to 17 mg/m³ (10 to 15 ppm) for 8 or more hours....
>
> "Experimental exposure to CO concentrations sufficient to produce blood COHb levels of about 5 percent (a level producible by exposure to about 35 mg/m³ for 8 or more hours) has provided in some instances evidence of impaired performance on certain other psychomotor tests, and an impairment in visual discrimination....
>
> "Experimental exposure to CO concentrations sufficient to produce blood COHb levels above 5 percent (a level producible

TABLE 2.—*Effects of carbon monoxide.*

Environmental conditions	Effect	Comment
58 mg./m.³ (50 p.p.m.) for 90 minutes	Impairment of time-interval discrimination in non-smokers.	Blood COHb levels not available, but anticipated to be about 2.5 percent. Similar blood COHb levels expected from exposure to 10 to 17 mg./m.³ (10 to 15 p.p.m.) for 8 or more hours.
115 mg./m.³ (100 p.p.m.) intermittently through a facial mask	Impairment in performance of some psychomotor tests at a COHb level of 5 percent.	Similar results may have been observed at lower COHb levels, but blood measurements were not accurate.
High concentrations of CO were administered for 30 to 120 seconds, and then 10 minutes was allowed for washout of alveolar CO before blood COHb was measured.	Exposure sufficient to produce blood COHb levels above 5 percent has been shown to place a physiologic stress on patients with heart disease.	Data rely on COHb levels produced rapidly after short exposure to high levels of CO; this is not necessarily comparable to exposure over a longer time period or under equilibrium conditions.

SOURCE: Adapted from U.S. Public Health Service, Air Quality Criteria for Carbon Monoxide. Washington, D.C., U.S. Department of Health, Education, and Welfare (58).

by exposure to 35 mg/m³ or more for 8 or more hours) has provided evidence of physiologic stress in patients with heart disease. . . ."

The levels of carbon monoxide found to be present in "smoked" rooms (20 to 80 p.p.m.) are similar to the levels (30 to 50 p.p.m.) which the Advisory Committee has concluded are associated with adverse health effects:

> "An exposure of 8 or more hours to a carbon monoxide concentration of 12 to 17 mg/m³ (10 to 15 ppm) will produce a blood carboxyhemoglobin level of 2.0 to 2.5 percent in nonsmokers. This level of blood carboxyhemoglobin has been associated with adverse health effects as manifested by impaired time interval discrimination. Evidence also indicates that an exposure of 8 or more hours to a CO concentration of 35 mg/m³ (30 ppm) will produce blood carboxyhemoglobin levels of about 5 percent in nonsmokers. Adverse health effects as manifested by impaired performance on certain other psychomotor

tests have been associated with this blood carboxyhemoglobin level, and above this level there is evidence of physiologic stress in patients with heart disease."

These levels of CO are also similar to that set as the time-weighted occupational Threshold Limit Value of 50 p.p.m. for a 40-hour week (five 8-hour days) which has been in effect in the United States for the past several years (*13*). A further reduction in this limit to 25 p.p.m. is now under consideration. These levels of CO exceed those recently set by the Environmental Protection Agency as the national primary and secondary ambient air quality standards for CO (*14*). These standards are:

(a) 10 milligrams per cubic meter (9 p.p.m.)—maximum 8-hours concentration not to be exceeded more than once per year.

(b) 40 milligrams per cubic meter (35 p.p.m.)—maximum 1-hour concentration not to be exceeded more than once per year.

ALLERGIC AND IRRITATIVE REACTIONS TO CIGARETTE SMOKE AMONG NONSMOKERS

(A more detailed discussion of this subject is presented in the Allergy chapter of this report.)

Several investigators have reported on the discomfort and symptoms experienced by both allergic and nonallergic individuals upon exposure to tobacco smoke. Johansson and Ronge (*31, 32*) in 1965 and 1966 have observed that the acute irritation experienced by nonsmokers in the presence of tobacco smoke is maximal in warm, dry air and that nonsmokers experience more nasal irritation than ocular irritation as compared with smokers exposed to similar amounts of smoke in the atmosphere. Speer (*54*) studied the reactions of 441 nonsmokers divided into two groups, one composed of individuals with a history of allergic reactions and the other of individuals without such a history. The allergic group underwent skin testing for the presence of sensitivity to tobacco extract while the "nonallergic" group was determined solely by questionnaire concerning subjective allergic responses. Approximately 70 percent of both groups experienced eye irritation while other symptoms differed in their frequency from group to group (nasal symptoms: allergic 67 percent, "nonallergic" 29 percent; headache: allergic 46 percent, "nonallergic" 31 percent; cough: allergic 46 percent, "nonallergic" 25 percent; and wheezing: allergic 22 percent, "nonallergic" 4 percent). Thus, a significant proportion of nonsmoking individuals report discomfort and respiratory symptoms on exposure to tobacco smoke.

Other authors have attempted to separate out those patients who may have specific allergies to smoke. Zussman (*61*) found that in a random series of 200 atopic patients 16 percent were clinically sensitive to tobacco smoke, and that a majority of these were aided by desensitization therapy. In an earlier study, Pipes (*46*) observed that 13 percent of 229 patients with respiratory allergy showed positive skin tests to tobacco smoke. Savel (*49*) has recently reported on eight nonsmokers observed to be clinically hypersensitive to tobacco smoke. After *in vitro* incubation of their lymphocytes with cigarette smoke, increased incorporation of tritiated thymidine was recorded; similar exposure of the lymphocytes of those not sensitive resulted in depression of tritiated thymidine uptake.

Luquette, et al. (*39*) have recently reported on the immediate effects of exposure to cigarette smoke in school-age children. They observed that heart rate and blood pressure rose with such exposure, although questions remain about the adequacy of their controls and the manner in which the experimental situation may have excited the subjects. Finally, Cameron, et al. (*6*) observed that acute respiratory illnesses were more frequent among children from homes in which the parents smoked than among children of nonsmoking parents. The meaning of these results is uncertain since smoking by the children was not considered and the level of exposure to cigarette smoke in their homes was not measured. Shy, et al. (*53*) in a study of second grade Chattanooga school children failed to demonstrate a relationship between parental smoking habits and the respiratory illness rates of their children.

THE KNOWN HARMFUL EFFECTS OF THE PASSIVE INHALATION OF CIGARETTE SMOKE IN ANIMALS

A number of investigators have studied the effects of the passive inhalation of high concentrations of cigarette smoke on the pulmonary parenchyma and tracheobronchial tree of animals. The results of these investigations are listed in detail in the recent report to Congress, "The Health Consequences of Smoking," (*59*) in table 9 of the Bronchopulmonary chapter, and table 16 of the Cancer chapter.

The pathologic changes observed in the respiratory tract of the animals included parenchymal disruption, bronchitis, tracheobronchial epithelial dysplasia and metaplasia, and pulmonary adenomatous tumor formation. Leuchtenberger, et al. (*36*) exposed 151 mice to the smoke of from 25 to 1,526 cigarettes over a period of 1 to 23 months and observed that 20 percent of the animals developed severe bronchitis with atypism. Working with 30 control rabbits exposed to up to 20 cigarettes per day for two to five years, Holland, et al. (*30*) observed increased focal and generalized hyperplasia of

the bronchial epithelium and generalized emphysema in the exposed rabbits. Hernandez, et al. (*29*) observed significantly more pulmonary parenchymal disruption in adult greyhound dogs exposed to cigarette smoke 10 times per week for approximately one year than in nonexposed control animals.

Lorenz, et al. (*38*) observed no increase in respiratory tract tumor formation above that seen in controls in 97 Strain A mice exposed to cigarette smoke for up to 693 hours. Essenberg (*15*), however, exposed Strain A mice to cigarette smoke for 12 hours a day for up to one year and observed significantly more papillary adenocarcinomas in the exposed than in the control group. An increased percentage of hybrid mice were found by Mühlbock (*42*) to have alveolar carcinomas among the experimental group exposed to smoke for two hours a day for up to 684 days when compared with a nonexposed group. Similarly, Guerin (*22*) observed that 5,1 percent of rats exposed to cigarette smoke for 45 minutes a day for two to six months showed pulmonary tumors compared to 2.4 percent of the control mice.

Leuchtenberger, et al. (*37*), working with 400 female CF_1 mice, observed only a slight increase in the presence of pulmonary adenomatous tumors among those exposed to cigarette smoke compared with those in the control group. The authors commented that the presence of tumors showed an age relationship independent of smoking exposure. Otto (*43*) found that 11 percent of a group of albino mice exposed to 12 cigarettes a day for up to 24 months showed pulmonary adenomas as compared with five percent of the control non-exposed group. Dontenwill and Wiebecke (*12*) found that increasing the exposure of golden hamsters to up to four cigarettes a day for up to two years was associated with an increasing percentage of animals showing desquamative metaplasia and bronchial papillary metaplasia. Harris and Negroni (*26*) exposed 200 C57BL mice to cigarette smoke for 20 minutes a day every other day for life and found eight adenocarcinomas as compared to none in the control group.

Because the damage observed in these experiments was seen after prolonged exposure to high concentrations of cigarette smoke, and because the comparability of animal exposure to smoke with that of human exposure in smoke-filled rooms is unknown, it is presently impossible to be certain from animal experimentation about the extent of the damage that may occur during long-term intermittent exposure to lower concentrations.

SUMMARY

1. An atmosphere contaminated with tobacco smoke can contribute to the discomfort of many individuals.

2. The level of carbon monoxide attained in experiments using rooms filled with tobacco smoke has been shown to equal, and at times to exceed, the legal limits for maximum air pollution permitted for ambient air quality in several localities and can also exceed the occupational Threshold Limit Value for a normal work period presently in effect for the United States as a whole. The presence of such levels indicates that the effect of exposure to carbon monoxide may on occasion, depending upon the length of exposure, be sufficient to be harmful to the health of an exposed person. This would be particularly significant for people who are already suffering from chronic bronchopulmonary disease and coronary heart disease.

3. Other components of tobacco smoke, such as particulate matter and the oxides of nitrogen, have been shown in various concentrations to adversely affect animal pulmonary and cardiac structure and function. The extent of the contributions of these substances to illness in humans exposed to the concentrations present in an atmosphere contaminated with tobacco smoke is not presently known.

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SMOKING AND NONSMOKERS — WHAT IS THE ISSUE?

WITH tobacco consumption near an all-time high, a fervent interest has developed over the effect of passive smoke inhalation on the nonsmoker. Controversy exists on the rights of the nonsmoker to "clean" air versus the rights of the smoker to enjoy a product consumed by man for centuries. This issue extends far beyond the health professions and is complicated by the divergent interests of many groups, with approximately 160 proposals to restrict or segregate the smoker now pending before legislatures in 39 states. The primary issue is how much tobacco the nonsmoker passively inhales and what the potential health hazards of such exposure are. The number of publications attempting to answer these questions is very large. Until the contribution by Hinds and First in this issue of the *Journal*, reliable data on relevant public passive exposures to tobacco smoke have been sparse.

When cigarettes are consumed, tobacco smoke is generated in part as mainstream smoke, essentially all of which is actively inhaled and 70 to 90 per cent of which is retained by the smoker. Sidestream smoke of the burning cigarette is the primary source of exposure to the nonsmoker, representing about 50 per cent of the tobacco burned and responsible for approximately two thirds of the aerosol particles delivered to the environment. Smoke contains about 2000 or more identifiable components and can be divided into gas and particulate phases. Ambient concentrations of trace components can be measured, and potential amounts of pyrolized products available for inhalation by the nonsmoker determined.

Using nicotine as a tracer of the particulate phase, Hinds and First have indicated that the nonsmoker can potentially inhale only extremely small amounts of tobacco smoke. Nicotine may be an ideal tracer for such studies, since it is basically unique to tobacco. Other investigations have indicated that nicotine may occur in the urine of nonsmokers in concentrations at approximately 5 per cent that of smokers.[1] Contrary to the conclusions of Hinds and First, this figure suggests a passive consumption by the nonsmoker of about one full cigarette over an hour. These results, however, were obtained under experimentally high concentrations intolerable to prolonged human exposure. Carbon monoxide, a gas-phase component, has been used as a popular alternative tobacco tracer, producing environmental concentrations of 10 ppm or so.[2] For comparison, concentrations of 30 ppm are not uncommon in dense urban automobile traffic, and 100 ppm can be reached in polluted cities with temperature inversions.[2] Regardless of how studies are performed, only limited conclusions can be achieved by employment of tracers.

Definitive answers to questions of tobacco and nonsmokers should be obtained from data demonstrating the presence or absence of potentially associated diseases in man. That kind of information is extremely hard to come by. Potential health effects of tobacco on the nonsmoker have recently been reviewed[3,4] and are summarized as follows. Acute effects of short-term exposure to environ-

mental tobacco smoke in population studies have not been evaluated in adequate depth. No chronic studies have been reported for adults. No data are available to demonstrate health effects of physiologic responses to nicotine levels reached in adult nonsmokers, and carbon monoxide concentrations in nonsmokers are far below levels that are of known health hazard. Potential effects of other smoke components on nonsmokers are conjectural. Information is lacking on cumulative effects of prolonged passive exposure to tobacco-smoke products. Potential additive or synergistic effects of tobacco-smoke products with nontobacco environmental contaminants need to be investigated. Interesting data have appeared, however, to suggest an increasing prevalence of acute respiratory disease in young children whose parents smoke.[5,6] These studies have been criticized because the data are sparse and perhaps influenced by socioeconomic and housing conditions, infections in parents, genetic differences and other factors.[7]

Hypersensitivity reactions to tobacco in the nonsmoker deserve more clarification. Commonly, atopic persons complain that tobacco smoke provokes respiratory-tract symptoms. Many agents in tobacco smoke could theoretically act as haptens and produce immunologic responses after binding to suitable carriers, but there is little experimental or epidemiologic evidence that sensitization to tobacco smoke exists.[8] Fletcher et al., in a report to the Royal College of Physicians of London, concluded that "...there is no evidence that other people's smoke is dangerous to healthy nonsmokers, but it can be extremely irritating and cause distressing symptoms, especially in allergic persons or in those already affected by heart or lung disease."[9]

One is left, then, with a perplexing and unsolved dilemma. The data of Hinds and First demonstrate that in public places nonsmokers could potentially consume 1/1000 to 1/100 of one filter cigarette per hour, a level of exposure that has had no known serious association with disease. Why, then, do so many nonsmokers appear to be adversely affected by passive exposure to tobacco? Perhaps acrolein and aldehydes, present in the smoke, act as irritants in extremely low concentrations and especially affect latently or overtly hypersensitive persons. The odor threshold for stale smoke components, which is also very low, may trigger emotional responses not yet well understood.

Clearly, numerous nonsmokers feel impaired in their well-being if exposed to tobacco smoke. The World Health Organization defines health as a condition characterized not only by the absence of diseases and infirmity but also by the presence of full psychologic, mental and social well-being.[10] By this definition, the nonsmoker passively exposed to tobacco may indeed have an adverse health response on a psychogenic basis. The issues remain unanswered beyond that, and, to solve this problem, scientific contributions in this area should be broadened, thereby diminishing speculation. In developing crucially needed research on this issue, one should identify the compounds of tobacco smoke that provoke adverse reactions in nonsmokers, establish if these reactions are dose dependent in nature and, if so, delineate the threshold levels of response. Finally, and perhaps most importantly, potential illness-inducing capacities of smoke on the nonsmoker should be more intensely analyzed with appropriate epidemiologic technics.

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MEDICAL INTELLIGENCE

CONCENTRATIONS OF NICOTINE AND TOBACCO SMOKE IN PUBLIC PLACES

WILLIAM C. HINDS, SC.D.,
AND MELVIN W. FIRST, SC.D.

PUBLIC interest has focused on health effects to the large numbers of nonsmokers exposed to tobacco smoke in public places. Recent regulations in a few cities have banned smoking in public places, or have restricted smoking in the manner of United States commercial aircraft.

Two studies[1,2] indicated that in crowded private rooms concentrations of tobacco smoke often exceed 260 μg per cubic meter, the federal air-quality standard for particulate matter that is not to be exceeded more than one day per year. Hoegg[1] estimated that in residences, meeting rooms, or private automobiles, the nonsmoker inhales in one hour the equivalent of smoking 0.01 to 0.20 cigarettes. Bridge and Corn,[2] by measuring carbon monoxide during party situations involving 50 to 73 people in rooms of 140 and 100 m^3 under controlled ventilation conditions, estimated smoke concentrations to be 2000 to 4000 μg per cubic meter and concluded that these levels are a matter of concern.

Estimation of levels of tobacco smoke in public places was undertaken to evaluate the health implications for nonsmokers. Measurements were limited to the particulate phase of tobacco smoke, although it is known that the gaseous phase also contains substances that may affect health. Since the objective was to measure only tobacco smoke, all methods commonly used to measure total suspended particulate matter were ruled out because of the many other sources of particulate matter in the indoor atmosphere. The use of carbon monoxide as a tracer has similar disadvantages because of the widespread distribution of this common air pollutant. Nicotine was chosen as the tracer for tobacco smoke for the following reasons: it is specific for tobacco smoke (the only other source of nicotine is from agricultural sprays, which are unlikely to be a contaminant of the indoor atmospheres tested); with the exception of water, nicotine is the largest single component of the particulate phase of tobacco smoke; nicotine concentration is unaffected by the moisture content of the smoke; and sensitive gas chromatographic analytical methods are available for measurement of nicotine concentrations.

From the Department of Environmental Health Sciences, Harvard School of Public Health (reprint requests should be addressed to Dr. Hinds at the Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115).

Supported by the Massachusetts Lung Association and its local affiliates.

Because of the wide range of public places evaluated and the small number of samples, the procedures employed and the results should be considered a pilot study having the limited objective of defining the extent of the "passive-smoking" problem in public places.

SAMPLING METHODS

The procedure was to enter a public place as a patron and sample a known volume of air through an AA Millipore filter having a collection efficiency for tobacco smoke greater than 99 per cent. Samples were taken with an inconspicuous battery-powered pump at a rate of 4 liters per minute for a maximum period of 2½ hours. The entire sampling system weighed 1.3 kg and was contained in a phenolic box, 17 by 13 by 6 cm (Fig. 1). To obtain realistic samples, the unit was placed as close to the breathing zone as possible—e.g., on a table in a restaurant, or on a lap in a train.

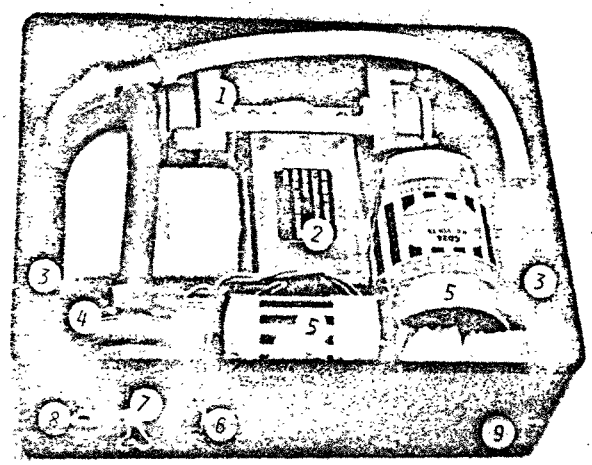

Figure 1. The Sampling System, Showing the Pump (1), Motor (2), Pulsation Damper (3), Filter Holder (4), Rechargeable Batteries (5), On-Off Switch (6), Air Inlet (7), Battery Charging Jack (8), and Case (9).

The material trapped on the filter was extracted with distilled water, concentrated by rotary evaporation, and analyzed for nicotine with a gas chromatographic technic described by Jacin et al.[3] The nicotine content was used to calculate the tobacco-smoke particulate concentration on the basis of an experimentally determined nicotine fraction of 2.6 per cent established by measurement of total particulate mass and nicotine concentration of sidestream smoke in an aerosol chamber. Sidestream smoke is the principal component of indoor tobacco-smoke pollution (i.e., 80 to 90 per cent).[1,2]

Tests were run with filter and nonfilter cigarettes, and current sales figures[4] were used to calculate the weighted average nicotine fraction as 2.6 per cent. No noteworthy concentration effect on nicotine fraction was observed for smoke concentrations ranging from 6000 to 110,000 μg per cubic meter. Our ambient measurements were an order of magnitude smaller than this range.

Twenty-three samples were taken in the Boston area during 1973 and early 1974. Some types of public areas—commuter trains, commuter buses, and bus and airline waiting rooms—were sampled repeatedly, whereas others, such as large, crowded restaurants and lounges, are represented by individual samples. On buses and trains no attempt was made to sample in smoking or nonsmoking sections because these designations are largely ignored by passengers.

RESULTS

Smoke concentration for each category of public place is shown in Table 1 as weight per unit volume of sampled

Table 1. Tobacco-Smoke Concentrations in Indoor Public Places.

Category	No. of Samples	Measured Nicotine Concentration*	Calculated Tobacco-Smoke Concentration*		Equivalent Filter Cigarettes Smoked/Hr
		Average	Range	Average	
Commuter train	6	4.9	20-480	190	0.004
Commuter bus	5	6.3	140-370	240	0.005
Bus waiting room	2	1.0	16-58	40	0.001
Airline waiting room	2	3.1	120	120	0.003
Restaurant	4	5.2	51-450	200	0.004
Cocktail lounge	3	10.3	170-640	400	0.009
Student lounge	1	2.8	110	110	0.002

*μg/m³.

Table 2. Ambient-Air-Quality Standards and Threshold-Limit Values for Suspended Particulate Matter, Nuisance Dust, and Nicotine.

Sample	Concentration μg/m³
Community air-quality standards:	
Suspended particulate matter:	
Annual average	75
Maximum 24-hr concentration (not to be exceeded > once/yr)	260
Occupational standards:	
Nuisance dust:	
Threshold limit value	10,000
Nicotine:	
Threshold limit value	500

air and "equivalent filter cigarettes per hour," the amount of smoke inhaled by a sedentary nonsmoker in one hour divided by the amount inhaled by a person smoking one filter cigarette (16.1 mg).[1,5,6]

The data on tobacco-smoke concentration presented in Table 1 can be compared to bench marks for clean air based on community ambient-air-quality standards and threshold-limit values for occupational exposures shown in Table 2. These community air-quality standards are based on nontoxic dusts, and it is reasonable to assume that tobacco smoke may be considerably more harmful. The concentrations shown in Table 1 are solely the result of tobacco smoke and do not include the background contribution from usual particulate air pollutants.

The smoke concentrations shown in Table 1 are considerably less than those determined by Hoegg[1] and by Bridge and Corn,[2] who did not account for evaporative losses and diffusive losses to surfaces. Furthermore, calculations based on their data give 12 to 22 per cent of persons smoking at a time and room volumes of 10 to 31 m³ per person smoking, whereas spot checks made during the present study gave an average of only 9 per cent of people smoking, and room volumes per person smoking ranged from 28 to 4200 m³. These differences, at least in part, explain why their calculated concentrations of tobacco smoke are higher by a factor of 10 than our measured values.

The data collected during this study suggest that although tobacco-smoke concentrations often exceed the annual average air quality standard for clean air, these levels would not be expected to produce the strong public reaction to tobacco smoke that has developed in the past few years. This observation suggests that annoyance from tobacco smoke is caused by factors other than the average concentration of particulate matter in the indoor atmosphere. For example, annoyance may be a response to peak concentrations of tobacco smoke that are likely to be much greater than the average values given in Table 1.

Considerable annoyance from tobacco smoking may also result from gaseous components produced during the tobacco combustion. Gaseous components (not including water vapor) represent approximately 70 per cent of the mass of combustion products in sidestream smoke[7] and include strong irritants and unpleasant odors, such as phenols, aldehydes, and organic acids. Awareness of tobacco smoke is enhanced because its submicrometer particle size produces a highly visible aerosol at low mass concentrations. These factors, taken together, may be a more important cause of the public's adverse reaction to tobacco smoke than the quantity measured in the present study, the average smoke concentration.

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The American Lung Association's

Point-by-Point Examination of Statements

in the Booklet

"True? False? - Tobacco Facts"

published by

The Tobacco Institute

EXHIBIT G

1. TOBACCO SMOKE IS A MAJOR SOURCE OF AIR POLLUTION.

(The Tobacco Institute says this is false.)

The Institute cites studies about carbon monoxide levels in the outside air. No one maintains that tobacco smoke is a major pollutant in the outside air. The concern is about tobacco smoking in enclosed places, where smoke pollutes the air.

The 1972 Surgeon General's Report cites several studies that show increases in the carbon monoxide level in the blood of nonsmokers exposed to tobacco smoke. In one study, where seven cigarettes were smoked in one hour--in a ventilated chamber--the air pollution measurement showed carbon monoxide levels were 90 parts per million at the seat next to the smoker.

Maximum standards allowed in industry cannot average out to more than 50 parts per million; and efforts are now underway to reduce the maximum to 25. Federal Air Quality Standards for the outside air limit concentrations to an average of 9 parts per million. (The Health Consequences of Smoking--A Report of the Surgeon General--1972; pages 123, 128.)

2. IT'S A KNOWN FACT THAT MANY PEOPLE ARE ALLERGIC TO TOBACCO SMOKE.

(The Tobacco Institute says this is false.)

Dr. Jesse Steinfeld, who was Surgeon General in 1972, has this to say about allergies to tobacco smoke: "Over one and a half million people are allergic to tobacco smoke itself and many many millions more are allergic to other substances with the allergy being compounded and aggravated by cigarette smoke." (Speech presented to the Association for Nonsmokers' Rights in Minneapolis, February 23, 1974.)

3. NONSMOKERS IN A SMOKE-FILLED ROOM INHALE NEARLY AS MUCH SMOKE AS SMOKERS.

(The Tobacco Institute says this is false.)

The 1972 Surgeon General's Report cited experiments which measured the carbon monoxide levels in the blood of smokers and of nonsmokers who were exposed to second-hand smoke.

The point is that there is an increase in the carbon monoxide levels in the blood of nonsmokers who are exposed to second-hand smoke. There is also an increase--a slightly greater one--in smokers themselves. In one experiment in a smoke-filled car, for example, the carbon monoxide levels in the blood of nonsmokers rose from 2 to 5 percent. The smokers themselves showed an increase from 5 to 10 percent. (The Health Consequences of Smoking--A Report of the Surgeon General--1972; page 125.)

Federal safety standards for carbon monoxide levels in the blood limit concentrations to 1.5 percent.

The National Air Quality Committee of the National Air Pollution Control Administration says experimental exposure to carbon monoxide concentrations sufficient to produce levels of 5 percent in the blood can impair performance on certain psychmotor tests and visual discrimination. Hours of exposure to these levels also produce physiologic stress in patients with heart disease. (The Health Consequences of Smoking--A Report of the Surgeon General--1972; page 127.)

4. THE SURGEON GENERAL SAYS CARBON MONOXIDE IN EXPERIMENTAL SMOKE-FILLED ROOMS EXCEEDS PERMISSIBLE LEVELS.

   (The Tobacco Institute admits this is true.)

5. THE SURGEON GENERAL UNEQUIVOCALLY SAYS TOBACCO SMOKE IS DANGEROUS FOR NONSMOKERS.

   (The Tobacco Institute says this is false and that the Surgeon General's report did not suggest that the "artifical" levels of carbon monoxide found in experimental smoke-filled rooms are encountered in everyday life.)

   Here is what the Surgeon General said about smoke-filled places:

   "The smoking of most popular cigarettes releases approximately 70 mg. of dry particles and 23 mg. of carbon monoxide into the air. There is four times as much carbon monoxide in side stream smoke (from the burning end of the cigarette) as in mainstream smoke (inhaled by the smoker) and this is true for several of the other harmful constituents in cigarette smoke such as the tars.

   "About the only well-ventilated area in terms of air exchange and removal of contamination is the modern jet airplane which exchanges the air volume several times per minute. Many of the rooms in which we work or in which we hold meetings or conferences and the automobiles in which we drive have a very low rate of exchange of air; and in some instances none at all. This contributes to levels of carbon monoxide which are well above those found to be hazardous for working conditions by the U.S. Environmental Protection Agency." (Dr. Jesse L. Steinfeld, former Surgeon General, speech presented to the Association for Nonsmokers' Rights, Minneapolis, February 23, 1974.)

6. SINCE ANTISMOKING EFFORTS HAVE INCREASED LATELY, THE EVIDENCE AGAINST SMOKING MUST BE INCREASING, TOO.

   (The Tobacco Institute says this is false.)

   The first Surgeon General's Report, which included a vast amount of evidence against smoking, was published in 1964. Its conclusion was this: "Cigarette smoking contributes substantially to mortality from certain specific diseases and to the overall death rate." Reports to Congress have been issued almost every year since then. Each one has confirmed and strengthened the original findings. The evidence that cigarette smoking is a major cause of lung cancer, chronic bronchitis, emphysema, and heart disease is so overwhelming that every major medical and health agency accepts the conclusions.

The Public Health Service estimates that 300,000 Americans die prematurely each year from the effects of cigarette smoking.

7. THOROUGH REVIEWS OF THE WORLD'S SCIENTIFIC LITERATURE INDICATE THAT SMOKE ISN'T A SIGNIFICANT HEALTH HAZARD TO THE NONSMOKER.

(The Tobacco Institute says this is true.)

Evidently the Institute chooses to overlook the 1972 Surgeon General's Report. This report, like every Surgeon General's Report on Smoking, was a review of the world's scientific literature on smoking.

8. YET THE GOVERNMENT HAS SEGREGATED NONSMOKERS ON PUBLIC TRANSPORTATION.

(The Tobacco Institute admits this is true.)

On the basis of health effects of smoke on nonsmokers, the Board of Health of the City of New York has prohibited smoking in certain enclosed public places, including elevators and supermarkets. The Board of Health in the State of Washington has also taken similar action to ban smoking in a wide variety of public places, including reception areas and waiting rooms of any state buildings. Legislation to protect the health of nonsmokers in public places has already been passed in several states and in many cities and counties.

9. WE DON'T NEED LAWS TO PROTECT "NONSMOKERS' RIGHTS."

(The Tobacco Institute says this is true.)

The American Lung Association agrees with the former Surgeon General, Dr. Jesse Steinfeld, who said: "The tobacco companies have nowhere been so upset by the actions of those concerned with the health of the American people or by the Surgeon General in stating that smoking is a health hazard, as they now are when nonsmokers, those two-thirds, the majority, of all Americans, stand up for their rights and demand that smoking be performed only in designated and limited areas." (Speech in Minneapolis, February 23, 1974.)

"Nonsmokers," Dr. Steinfeld says, "have as much right to clean air and wholesome air as smokers have to their so-called right to smoke, which I would redefine as a so-called right to pollute. It is high time to ban smoking from all confined public places such as restaurants, theaters, airplanes, trains, and buses. It is time we interpret the Bill of Rights for the nonsmokers as well as the smoker."

In this brochure, the Tobacco Institute has completely misrepresented the position of the American Lung Association regarding nonsmokers' rights. ALA and its affiliates across the country are actively promoting nonsmokers' rights programs, and in 1975 will increase their efforts to help provide smoke-free environments for those millions of Americans who do not want their world--and lungs--polluted by tobacco smoke.

Contents

LIST OF TABLES

PUBLIC EXPOSURE TO AIR POLLUTION FROM TOBACCO SMOKE

The purpose of this chapter is to summarize the present state of evidence concerning the effects of exposure to an atmosphere containing either tobacco smoke or its constituents. Since the identification of cigarette smoking as a serious health hazard to the smoker was based on clinical and epidemiological observations that nonsmokers have much lower mortality and morbidity rates from a number of conditions, it is obvious that cigarette smoking is normally a greater hazard to the smoker than is the typical level of exposure to air pollutants produced by the smoking of cigarettes which many nonsmokers experience. This would be consistent with the voluminous data which show a dose-response relationship between the level of exposure to smoke and the magnitude of its effect.

The research so far reported on the nature and effects of exposure to smoke-pollutants in the atmosphere has not been as extensive and well-controlled as that done on the health effects of smoking on the smoker himself. Knowledge on this subject can be separated into four major areas of concern:

1. The extent to which the components of cigarette smoke contaminate the atmosphere and are absorbed by the nonsmoker.
2. The effects of low levels of carbon monoxide on human health.
3. Allergic, adverse, and irritative reactions to cigarette smoke among nonsmokers.
4. The known harmful effects of the passive inhalation of cigarette smoke in animals.

THE EXTENT TO WHICH THE COMPONENTS OF CIGARETTE SMOKE CONTAMINATE THE ATMOSPHERE AND ARE ABSORBED BY THE NONSMOKER

Theoretical models of this contamination have been constructed. Owens and Rossano (44) have noted that most popular cigarettes release into the atmosphere approximately 70 mg. of dry particulate matter (about 60 mg. in the sidestream and slightly over 20 mg. in the mainstream, about one-half of the latter being absorbed by the smoker and one-half expelled into the ambient air) and 23 mg. car-

bon monoxide per cigarette. This material adds to the cleaning problem of the air of any enclosed space and contributes to residual odors. In a recent study of particulate matter filtration in domestic premises (*35*), the authors observed that the smoking of one cigar completely overcame the effect of an electrostatic filtration device for one hour.

Atmospheric pollutants caused by smoking are derived from two major sources: mainstream and sidestream smoke. Mainstream smoke emerges from the tobacco product through the mouthpiece during puffing, whereas sidestream smoke comes from the burning cone and from the mouthpiece during puff intermissions (*60*). The tobacco smoke released into the atmosphere consists of all the sidestream smoke as well as that part of the mainstream smoke which has been either held in the smoker's mouth or taken into his lungs and then expelled. The actual amount of material to which individuals are exposed in the presence of smokers depends upon the amount of smoke produced, the depth of inhalation on the part of the smoker, the ventilation available for the removal or dispersion of the smoke, and the proximity of the individual to the smoker. The length of time of exposure to those pollutants is extremely important in determining how much is absorbed into the body. The pattern of smoking influences the amount produced by altering the content of the exhaled smoke. As shown by Dalhamn, et al. (*10, 11*), mouth absorption removes approximately 60 percent of the water-soluble volatile components (e.g., acetaldehyde), 20 percent of the nonwater-soluble volatile components (e.g., isoprene), 16 percent of the particulate matter, and only three percent of the carbon monoxide. Thus, the smoker who does not inhale "filters" a portion of the smoke components in his mouth before expelling them into the ambient air. On the other hand, the lungs retain from 86 to 99 percent of the volatile and particulate substances and approximately 54 percent of the carbon monoxide inhaled. Hence, the inhaling smoker "filters" the mainstream smoke rather effectively before expelling it into the ambient air. A factor which has apparently not been investigated is the difference in the smokers' "filtration" of mainstream smoke when the smoke is exhaled through the nose instead of the mouth.

Thus, the nonsmoker breathes smoke-containing air composed of sidestream smoke and mainstream smoke exhaled by smokers. The inhaling smoker receives nearly the full amount of mainstream smoke as well as a portion of sidestream smoke and smoke exhaled by himself and other smokers. The smoker who does not inhale receives those compounds which are absorbed from the mainstream smoke in his mouth, as well as absorbing the sidestream smoke and the smoke exhaled by himself and other smokers contained in the air he breathes.

Since pipe and cigar smokers inhale less commonly than do cigarette smokers, their contribution to the substances in the air breathed in exposure to smoke pollutants consists of a composite of sidestream smoke and relatively unfiltered mainstream smoke which has been held in the mouth and then expelled.

The actual effluents in the mainstream and sidestream cigarette smoke have been considered by Pascasio, et al. (*45*) and Scassellati Sforzolini and colleagues (*50, 51*). These authors stated that "tar" and nicotine levels in sidestream smoke may be significantly higher than those of mainstream smoke and may be harmful to the nonsmoker. Actual volume measurements were not reported, however.

Actual measurements of the contamination due to cigarette smoking have been carried out by a number of research groups. A recent, well-controlled study by Harke (*24*) involved the smoking of 42 cigarettes in 16 to 18 minutes using German blend cigarettes of 85 mm. length, 18 mm. filter, and smoked to a 25 mm. butt length in a room with a volume of 57 cubic meters (approximately the equivalent of a room with a 10-foot ceiling and dimensions of 12 by 14 feet). The author observed that in the absence of ventilation the atmosphere contained up to 50 p.p.m. carbon monoxide and .57 mg./m.^{3} nicotine. With substantial ventilation, these levels fell significantly (to approximately 10 p.p.m. carbon monoxide and .10 mg./m.^{3} nicotine). He also found that cigar smoke (9 cigars of Clear Sumatra tobacco smoked in 30 to 35 minutes) produced similar amounts of contamination while pipe smoke (3 grams of Navy type medium cut tobacco smoked as eight pipefuls in 35 to 40 minutes) produced much less. Other authors have made similar measurements. Galuskinova (*20*) found that 3,4-benzpyrene levels in a smoky restaurant were from 2.82 to 14.4 mg./100 m.^{3} as compared to outside atmospheric levels of 0.28 to 0.46 mg./100 m.^{3}, although burning of food particles may have contributed to the presence of 3,4-benzpyrene in this setting. Kotin and Falk (*33*) have shown that sidestream cigarette smoke condensate may contain more than three times as much benzo(a)pyrene as mainstream smoke. Srch (*55*) observed that the smoking of 10 cigarettes to a 5 mm. butt length in an enclosed car of 2.09 m.^{3} volume produced carbon monoxide levels up to 90 p.p.m. Lawther and Commins (*34*), working with a ventilated chamber, found levels of up to 20 p.p.m. of carbon monoxide after seven cigarettes were smoked in one hour; however, peaks of up to 90 p.p.m. were recorded at the seat next to the smoker. Coburn, et al. (*9*) recorded levels of 20 p.p.m. of carbon monoxide in a small conference room after 10 cigarettes were "burned." Harmsen and Effenberger (*25*) reported up to 80 p.p.m. of carbon monoxide in an enclosed 98 m.^{3} room (approximately the equivalent of a room with a 10-foot ceiling and dimensions of 18 by 20 feet) in which 62 cigarettes had been smoked in two hours.

TABLE 1.—*Percent of COHb during and following exposure to 50 p.p.m. of CO.*

Time during exposure	Mean	Range	Number of subjects
Preexposure	0.7	0.4–1.5	11
30 minutes	1.3	1.3	3
1 hour	2.1	1.9–2.7	11
3 hours	3.8	3.6–4.2	10
6 hours	5.1	4.9–5.5	5
8 hours	5.9	5.4–6.2	5
12 hours	7.0	6.5–7.9	3
15 ½ hours	7.6	7.2–8.2	3
22 hours	8.5	8.1–8.7	3
24 hours	7.9	7.6–8.2	3
Time without exposure after 1 hour of exposure			
30 minutes	1.8	1.8	3
1 hour	1.7	1.6–1.8	3
2 hours	1.5	1.4–1.5	3
5 hours	1.1	1.0–1.1	2
Time without exposure after 3 hours of exposure			
30 minutes	3.7	3.4–3.9	3
1 hour	3.3	2.7–3.8	3
2 hours	2.7	2.3–3.0	3
Time without exposure after 8 hours of exposure			
30 minutes	5.6	5.1–5.9	3
1 hour	5.1	4.8–5.4	3
1 ¾ hours	4.0	—	—
11 hours	1.5	1.4–1.7	3
Time without exposure after 24 hours of exposure			
30 minutes	7.5	7.2–7.8	3
1 hour	6.7	6.4–7.1	3
2 hours	5.8	5.6–6.2	3

SOURCE: Stewart, et al. (56).

Another set of contaminants probably present in a tobacco smoke-polluted atmosphere are the oxides of nitrogen. These, specificially NO and NO_2, have been shown to be present in tobacco smoke although the type most likely to be present in the atmosphere is NO_2. No measurements have been reported of the amount of NO_2 in smoke-filled rooms. The importance of obtaining and evaluating this information is stressed by the results of Freeman and Haydon and

their colleagues (*17, 18, 19, 27, 28*) and of Blair, et al. (*5*) who observed bronchial and pulmonary parenchymal lesions in rodents continuously exposed to low levels of NO_2.

Other experimenters have measured carboxyhemoglobin (COHb) levels in nonsmokers exposed to cigarette smoke pollutants. Srch (*55*) observed that the COHb level in two nonsmokers rose from 2 to 5 percent (that of smokers from 5 to 10 percent) when seated in the cigarette-smoke contaminated car mentioned above (exposure to 90 p.p.m.). Harke (*24*) reported that when seven nonsmokers were exposed for approximately 90 minutes to a "smoked" room containing 30 p.p.m. of CO there was a rise in COHb from a mean of 0.9 percent to 2.0 percent. In 11 smokers subjected to the same conditions, COHb rose from a mean of 3.3 percent to 7.5 percent. With improved ventilation of the experimental room, the COHb level decreased significantly.

The CO exposures and COHb levels reported above closely approximate the results obtained following experimental chamber exposure of humans to various levels of CO. The uptake of CO by the person depends on, among other parameters: CO concentration, previous COHb level, the level of activity, and the person's state of health. Equilibrium between CO concentration in the lung and in the blood requires over 12 hours exposure. However, as may be noted in table 1, reproduced from Stewart, et al. (*56*) and derived from measures of COHb in young sedentary males who were not smoking, over half of the equilibrium COHb level is reached within three to four hours of the onset of exposure. The equilibrium value associated with 100 p.p.m. is approximately 14 to 15 percent COHb. Exposure to 100 p.p.m. in the nonsmoker can lead to 3.0 percent of COHb within 60 minutes and 6.0 percent in two hours (*16*). Of equal significance is that COHb has a half-life of at least three to four hours in the body. As shown in table 1, the COHb level fell only to 2.7 percent in the two hours following cessation of exposure to 50 p.p.m. from the end exposure level of 3.7 percent. This lengthy half-life extends the period of effect of exposure to CO and provides for a buildup of COHb concentration from fresh exposures.

THE EFFECTS OF LOW LEVELS OF CARBON MONOXIDE ON HUMAN HEALTH

The data on the effect of low levels of carbon monoxide on human psychological and physiological function have been summarized in two recent publications (*8, 58*).

There is presently much discussion as to the physiologic and psychophysiologic effects of exposure to levels of CO approximating 50 to 100 p.p.m. Beard and Grandstaff (*4*) observed that exposure to 50 p.p.m. of CO for from 27 to 90 minutes altered auditory dis-

crimination, visual acuity, and the ability to distinguish relative brightness. McFarland (*40*) observed that COHb levels of 4 to 5 percent caused visual threshold impairment. Ray and Rockwell (*48*), reporting on a study of the driving ability of three subjects under varying CO exposure, observed that the presence of 10 percent COHb was associated with increased response time for taillight discrimination and increased variance in distance estimation. Schulte (*52*) observed that increased errors in cognitive and choice discrimination tests were manifest at levels of COHb as low as 3 percent. Chevalier, et al. (*7*) have also observed that levels of 4 percent COHb in nonsmokers are associated with an increase in oxygen debt formation with exercise similar to that seen in smokers.

On the other hand, other investigators utilizing complex psychomotor tasks in men and monkeys have observed no decrement in function upon exposures to CO at 50 to 250 p.p.m. (*2, 3, 23, 41, 56*).

Animals exposed to low levels of CO (50 to 100 p.p.m.) continuously for weeks have shown varying degrees of cardiac and cerebral damage similar to that produced by hypoxia (*21, 47, 57*).

Finally, the possible effects of exposure to 50–100 p.p.m. CO on patients with coronary heart disease (CHD) were investigated by Ayres, et al. (*1*) who observed a decrease in arterial and mixed venous oxygen tensions with COHb saturations of 5 percent. Certain patients with CHD developed altered lactate and pyruvate metabolism with COHb levels of 5 to 10 percent suggesting myocardial hypoxia.

The evidence concerning the effect of low levels of carbon monoxide has recently been reviewed and evaluated by the National Air Quality Criteria Committee of the National Air Pollution Control Administration (*58*). The following is taken from the published conclusions of the Advisory Committee (also see table 2):

> "Experimental exposure of nonsmokers to 58 mg/m³ (50 ppm) for 90 minutes has been associated with impairment in time-interval discrimination.... This exposure will produce an increase of about 2 percent COHb in the blood. This same increase in blood COHb will occur with continuous exposure to 12 to 17 mg/m³ (10 to 15 ppm) for 8 or more hours....
>
> "Experimental exposure to CO concentrations sufficient to produce blood COHb levels of about 5 percent (a level producible by exposure to about 35 mg/m³ for 8 or more hours) has provided in some instances evidence of impaired performance on certain other psychomotor tests, and an impairment in visual discrimination....
>
> "Experimental exposure to CO concentrations sufficient to produce blood COHb levels above 5 percent (a level producible

TABLE 2.—*Effects of carbon monoxide.*

Environmental conditions	Effect	Comment
58 mg./m.³ (50 p.p.m.) for 90 minutes	Impairment of time-interval discrimination in non-smokers.	Blood COHb levels not available, but anticipated to be about 2.5 percent. Similar blood COHb levels expected from exposure to 10 to 17 mg./m.³ (10 to 15 p.p.m.) for 8 or more hours.
115 mg./m.³ (100 p.p.m.) intermittently through a facial mask	Impairment in performance of some psychomotor tests at a COHb level of 5 percent.	Similar results may have been observed at lower COHb levels, but blood measurements were not accurate.
High concentrations of CO were administered for 30 to 120 seconds, and then 10 minutes was allowed for washout of alveolar CO before blood COHb was measured.	Exposure sufficient to produce blood COHb levels above 5 percent has been shown to place a physiologic stress on patients with heart disease.	Data rely on COHb levels produced rapidly after short exposure to high levels of CO; this is not necessarily comparable to exposure over a longer time period or under equilibrium conditions.

SOURCE: Adapted from U.S. Public Health Service, Air Quality Criteria for Carbon Monoxide. Washington, D.C., U.S. Department of Health, Education, and Welfare (68).

by exposure to 35 mg/m³ or more for 8 or more hours) has provided evidence of physiologic stress in patients with heart disease. . . ."

The levels of carbon monoxide found to be present in "smoked" rooms (20 to 80 p.p.m.) are similar to the levels (30 to 50 p.p.m.) which the Advisory Committee has concluded are associated with adverse health effects:

"An exposure of 8 or more hours to a carbon monoxide concentration of 12 to 17 mg/m³ (10 to 15 ppm) will produce a blood carboxyhemoglobin level of 2.0 to 2.5 percent in nonsmokers. This level of blood carboxyhemoglobin has been associated with adverse health effects as manifested by impaired time interval discrimination. Evidence also indicates that an exposure of 8 or more hours to a CO concentration of 35 mg/m³ (30 ppm) will produce blood carboxyhemoglobin levels of about 5 percent in nonsmokers. Adverse health effects as manifested by impaired performance on certain other psychomotor

tests have been associated with this blood carboxyhemoglobin level, and above this level there is evidence of physiologic stress in patients with heart disease."

These levels of CO are also similar to that set as the time-weighted occupational Threshold Limit Value of 50 p.p.m. for a 40-hour week (five 8-hour days) which has been in effect in the United States for the past several years (*13*). A further reduction in this limit to 25 p.p.m. is now under consideration. These levels of CO exceed those recently set by the Environmental Protection Agency as the national primary and secondary ambient air quality standards for CO (*14*). These standards are:

(a) 10 milligrams per cubic meter (9 p.p.m.)—maximum 8-hours concentration not to be exceeded more than once per year.

(b) 40 milligrams per cubic meter (35 p.p.m.)—maximum 1-hour concentration not to be exceeded more than once per year.

ALLERGIC AND IRRITATIVE REACTIONS TO CIGARETTE SMOKE AMONG NONSMOKERS

(A more detailed discussion of this subject is presented in the Allergy chapter of this report.)

Several investigators have reported on the discomfort and symptoms experienced by both allergic and nonallergic individuals upon exposure to tobacco smoke. Johansson and Ronge (*31, 32*) in 1965 and 1966 have observed that the acute irritation experienced by nonsmokers in the presence of tobacco smoke is maximal in warm, dry air and that nonsmokers experience more nasal irritation than ocular irritation as compared with smokers exposed to similar amounts of smoke in the atmosphere. Speer (*54*) studied the reactions of 441 nonsmokers divided into two groups, one composed of individuals with a history of allergic reactions and the other of individuals without such a history. The allergic group underwent skin testing for the presence of sensitivity to tobacco extract while the "nonallergic" group was determined solely by questionnaire concerning subjective allergic responses. Approximately 70 percent of both groups experienced eye irritation while other symptoms differed in their frequency from group to group (nasal symptoms: allergic 67 percent, "nonallergic" 29 percent; headache: allergic 46 percent, "nonallergic" 31 percent; cough: allergic 46 percent, "nonallergic" 25 percent; and wheezing: allergic 22 percent, "nonallergic" 4 percent). Thus, a significant proportion of nonsmoking individuals report discomfort and respiratory symptoms on exposure to tobacco smoke.

Other authors have attempted to separate out those patients who may have specific allergies to smoke. Zussman (*61*) found that in a random series of 200 atopic patients 16 percent were clinically sensitive to tobacco smoke, and that a majority of these were aided by desensitization therapy. In an earlier study, Pipes (*46*) observed that 13 percent of 229 patients with respiratory allergy showed positive skin tests to tobacco smoke. Savel (*49*) has recently reported on eight nonsmokers observed to be clinically hypersensitive to tobacco smoke. After *in vitro* incubation of their lymphocytes with cigarette smoke, increased incorporation of tritiated thymidine was recorded; similar exposure of the lymphocytes of those not sensitive resulted in depression of tritiated thymidine uptake.

Luquette, et al. (*39*) have recently reported on the immediate effects of exposure to cigarette smoke in school-age children. They observed that heart rate and blood pressure rose with such exposure, although questions remain about the adequacy of their controls and the manner in which the experimental situation may have excited the subjects. Finally, Cameron, et al. (*6*) observed that acute respiratory illnesses were more frequent among children from homes in which the parents smoked than among children of nonsmoking parents. The meaning of these results is uncertain since smoking by the children was not considered and the level of exposure to cigarette smoke in their homes was not measured. Shy, et al. (*53*) in a study of second grade Chattanooga school children failed to demonstrate a relationship between parental smoking habits and the respiratory illness rates of their children.

THE KNOWN HARMFUL EFFECTS OF THE PASSIVE INHALATION OF CIGARETTE SMOKE IN ANIMALS

A number of investigators have studied the effects of the passive inhalation of high concentrations of cigarette smoke on the pulmonary parenchyma and tracheobronchial tree of animals. The results of these investigations are listed in detail in the recent report to Congress, "The Health Consequences of Smoking," (*59*) in table 9 of the Bronchopulmonary chapter, and table 16 of the Cancer chapter.

The pathologic changes observed in the respiratory tract of the animals included parenchymal disruption, bronchitis, tracheobronchial epithelial dysplasia and metaplasia, and pulmonary adenomatous tumor formation. Leuchtenberger, et al. (*36*) exposed 151 mice to the smoke of from 25 to 1,526 cigarettes over a period of 1 to 23 months and observed that 20 percent of the animals developed severe bronchitis with atypism. Working with 30 control rabbits exposed to up to 20 cigarettes per day for two to five years, Holland, et al. (*30*) observed increased focal and generalized hyperplasia of

the bronchial epithelium and generalized emphysema in the exposed rabbits. Hernandez, et al. (*29*) observed significantly more pulmonary parenchymal disruption in adult greyhound dogs exposed to cigarette smoke 10 times per week for approximately one year than in nonexposed control animals.

Lorenz, et al. (*38*) observed no increase in respiratory tract tumor formation above that seen in controls in 97 Strain A mice exposed to cigarette smoke for up to 693 hours. Essenberg (*15*), however, exposed Strain A mice to cigarette smoke for 12 hours a day for up to one year and observed significantly more papillary adenocarcinomas in the exposed than in the control group. An increased percentage of hybrid mice were found by Mühlbock (*42*) to have alveolar carcinomas among the experimental group exposed to smoke for two hours a day for up to 684 days when compared with a nonexposed group. Similarly, Guerin (*22*) observed that 5.1 percent of rats exposed to cigarette smoke for 45 minutes a day for two to six months showed pulmonary tumors compared to 2.4 percent of the control mice.

Leuchtenberger, et al. (*37*), working with 400 female CF, mice, observed only a slight increase in the presence of pulmonary adenomatous tumors among those exposed to cigarette smoke compared with those in the control group. The authors commented that the presence of tumors showed an age relationship independent of smoking exposure. Otto (*43*) found that 11 percent of a group of albino mice exposed to 12 cigarettes a day for up to 24 months showed pulmonary adenomas as compared with five percent of the control non-exposed group. Dontenwill and Wiebecke (*12*) found that increasing the exposure of golden hamsters to up to four cigarettes a day for up to two years was associated with an increasing percentage of animals showing desquamative metaplasia and bronchial papillary metaplasia. Harris and Negroni (*26*) exposed 200 C57BL mice to cigarette smoke for 20 minutes a day every other day for life and found eight adenocarcinomas as compared to none in the control group.

Because the damage observed in these experiments was seen after prolonged exposure to high concentrations of cigarette smoke, and because the comparability of animal exposure to smoke with that of human exposure in smoke-filled rooms is unknown, it is presently impossible to be certain from animal experimentation about the extent of the damage that may occur during long-term intermittent exposure to lower concentrations.

SUMMARY

1. An atmosphere contaminated with tobacco smoke can contribute to the discomfort of many individuals.

2. The level of carbon monoxide attained in experiments using rooms filled with tobacco smoke has been shown to equal, and at times to exceed, the legal limits for maximum air pollution permitted for ambient air quality in several localities and can also exceed the occupational Threshold Limit Value for a normal work period presently in effect for the United States as a whole. The presence of such levels indicates that the effect of exposure to carbon monoxide may on occasion, depending upon the length of exposure, be sufficient to be harmful to the health of an exposed person. This would be particularly significant for people who are already suffering from chronic bronchopulmonary disease and coronary heart disease.

3. Other components of tobacco smoke, such as particulate matter and the oxides of nitrogen, have been shown in various concentrations to adversely affect animal pulmonary and cardiac structure and function. The extent of the contributions of these substances to illness in humans exposed to the concentrations present in an atmosphere contaminated with tobacco smoke is not presently known.

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PRACTICE LIMITED TO CHEST DISEASE

TELEPHONE 702/384-5101

April 21, 1975

The Honorable Norman Ty Hilbrecht
State Senator
Legislative Building
Carson City, Nevada 89701

Dear Senator Hilbrecht:

I would urge you to consider Nonsmokers Rights Legislation, AB-17, favorably. While there is no doubt that smoking is the cause of nearly all cases of pulmonary emphysema and chronic bronchitis, my interest in this bill is concerned more with the growing percentage of the population who do not smoke and who really should not have to tolerate smoking in close proximity in public places. More and more medical meetings, for example, are either outlawing smokers or exiling smokers to a specific area in the meeting hall. I am sure you have had the experience of being packed into an elevator at a time when one of the passengers is smoking a cigar.

I do not feel that this legislation will attack the rights of the smokers but it will defend at least some of the rights of the nonsmokers. I have included copies of two recent articles from the New England Journal of Medicine which I hope will be of some help to you.

Yours sincerely,

Gary Symonds, MD

GS/j

encl.

EXHIBIT I