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CIGARETTE SMOKING AND HEALTH—UPDATE 1978

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HEARINGS

BEFORE THE

SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS

OF THE

COMMITTEE ON

INTERSTATE AND FOREIGN COMMERCE

HOUSE OF REPRESENTATIVES

NINETY-FIFTH CONGRESS

SECOND SESSION

OCTOBER 5 AND 6, 1978

Serial No. 95-172

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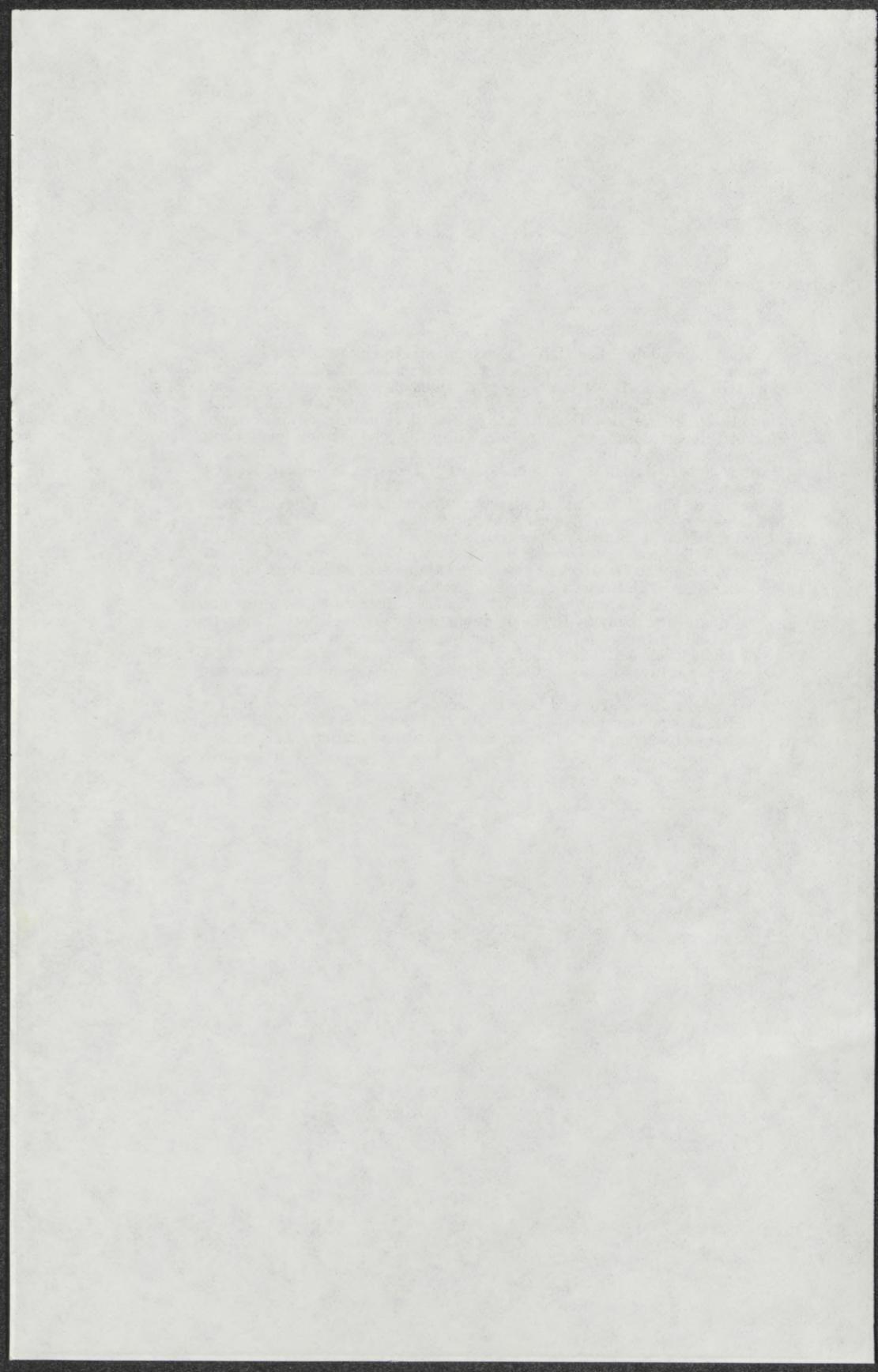
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CIGARETTE SMOKING AND HEALTH—UPDATE

1978

THURSDAY, OCTOBER 5, 1978

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS,
COMMITTEE ON INTERSTATE AND FOREIGN COMMERCE,
Washington, D.C.

The subcommittee met, pursuant to notice, at 10 a.m., in room 2322, Rayburn House Office Building, Hon. Andrew Maguire, presiding. [Hon. John E. Moss, chairman.]

Mr. MAGUIRE. The hearing will be in order.

Good morning ladies and gentlemen.

The Oversight and Investigations Subcommittee will focus today on the relationship between smoking and health.

At the beginning of the 20th century, as health statisticians observed our increasing rate of lung cancer, the medical community began to pay increasing attention to smoking as the possible cause. As more data developed, strengthening the conviction that the link is causal, and that other adverse health effects were probably related to smoking as well, those in Government charged with the responsibility of protecting the public health issued a series of reports containing increasingly severe warnings about the ill effects of smoking. The latest "Smoking and Health," issued by Surgeon General Luther Terry in 1964, concluded:

Cigarette smoking is causally related to lung cancer in man; the magnitude of the effect of cigarette smoking far outweighs all other factors. The risk of developing lung cancer increases with the duration of smoking and the number of cigarettes smoked per day and is diminished by discontinuing smoking.

The report also states:

Cigarette smoking is the most important of the causes of chronic bronchitis in the United States and increases the risk of dying from chronic bronchitis.

It further stated:

A relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal. The smoking of cigarettes is associated with an increased risk of dying from pulmonary emphysema.

This far-reaching report also highlighted suggestions that a relationship also exists between smoking and cardiovascular disease, as well as to laryngeal, esophageal and urinary bladder cancers.

Moreover, the conclusion that smoking is dangerous to your health was supported by an American Medical Association study—financed by \$15 million in tobacco industry money—which stated:

The committee believes that the bulk of research sponsored by this project supports the contention that cigarette smoking plays an important role in the develop-

ment of chronic obstructive pulmonary diseases and constitutes a grave danger to individuals with pre-existing diseases of the coronary arteries.

In the face of this latest evidence of serious risk, there is good news and bad. The good news is that only 39 percent of adult males are now classified as smokers as opposed to 50 percent just 14 years ago. The bad news is that these inroads against smoking are not having an effect upon teenagers among whom smoking has increased dramatically. Recent figures suggest that almost twice as many women between the ages of 13 and 19 now smoke.

Faced with these alarming statistics, Health, Education, and Welfare Secretary Joseph A. Califano recently announced an anti-smoking initiative combining education, regulation, incentives, and research. At a February hearing of the Health and the Environment Subcommittee, Secretary Califano testified that in 1975 the Federal Government spent between \$5 and \$7 billion to help treat smoking-related diseases. Another \$12 to \$18 billion was lost in productivity due to absenteeism. These figures do not, of course, include the great—and as far as I know, unreported—costs to individuals of obtaining treatment.

In the face of this serious and largely preventable drain on our Nation's resources, it is useful to examine the latest data on the health effects of smoking to determine whether or not the public is being adequately safeguarded. Because much of the research into the health effects of smoking has been conducted by researchers who have been funded by the tobacco industry, we have invited witnesses here today to describe the results of this research and compare it with results obtained from other sources.

I believe adults have the right to smoke as long as they do so with adequate knowledge of the risks involved and as long as their smoking does not endanger others. I also believe that the tobacco industry has a responsibility to support and encourage a reasonable and adequate Government policy designed to lower the cost of smoking both in suffering and in dollars, to our Nation. I hope that this morning we will make some progress toward the goal of establishing a common understanding.

Now the first witness that we had called today was Mr. Horace Kornegay, president of the Tobacco Institute. In a letter delivered to Chairman Moss last night, Mr. Kornegay declined to appear. He found that other matters were more pressing in his schedule than to report to this subcommittee and to the Congress.

He did indicate in the letter that the Tobacco Institute was not itself directly involved in research. But it is certainly the feeling of the committee that at a future time we will want to have Mr. Kornegay's testimony one way or the other.

I would now like to call our first witnesses, Mr. Addison Yeaman, and Dr. Robert Hockett of the Council for Tobacco Research.

Gentlemen, will you take your places at the table, please.

Mr. WAXMAN. Mr. Chairman, will we have an opportunity to have Mr. Kornegay appear before us at some other time?

Mr. MAGUIRE. Well, that time and place has not yet been established. But it is certainly my opinion, and I would hope it would be the subcommittee's opinion, that it will be important for us to receive that testimony.

Mr. WAXMAN. I agree with you. I think it would be important for us to hear from the Tobacco Institute. I am disturbed that Mr. Kornegay has refused to appear before us.

I don't know if this refusal has anything in particular to do with the latest information supplied by the American Medical Association, which shows greater health risks from smoking than many of us were ever aware of. This was, incidentally, a study that the tobacco industry helped finance.

I think it is important to hear from him again, in light of that study, and hear his response to this new information. This is particularly important since it has been Mr. Kornegay's past position that there has been no linkage established between adverse health effects and smoking.

Now, it seems to me that with this new study that the industry has helped finance, he ought to appear before us and either acknowledge the relationship or give us some information to refute these new findings.

Mr. MAGUIRE. Well, I certainly agree with the gentleman from California. I might just say that in his letter to Chairman Moss, Mr. Kornegay included the following sentence: "The extent of my information on this subject is contained in my testimony presented before the Subcommittee on Health and the Environment of the House Commerce Committee on February 15, 1978."

Now that, of course, was several months prior to the release of the American Medical Association study to which you have referred.

I would certainly hope that we could have Mr. Kornegay's evaluation of that study and of the ongoing work that the Tobacco Institute has helped to fund at some time soon before this subcommittee. I share the gentleman's sentiments.

Would you rise to be sworn in, please?

You promise to tell the whole truth and nothing but the truth, so help you God?

Dr. HOCKETT. I do.

Mr. MAGUIRE. You are Dr. Hockett?

TESTIMONY OF ROBERT CASAD HOCKETT, PH. D., RESEARCH DIRECTOR, THE COUNCIL FOR TOBACCO RESEARCH—U.S.A., INC.

Dr. HOCKETT. Yes.

Mr. MAGUIRE. Where is Mr. Yeaman?

Dr. HOCKETT. I believe Mr. Yeaman is not here.

Mr. MAGUIRE. Mr. Yeaman is not here?

Dr. HOCKETT. No; he is not here.

Mr. MAGUIRE. Had he advised the subcommittee he was not going to appear?

Dr. HOCKETT. Well, I think so. His view was that we had been asked to discuss the research, and he is by profession a lawyer and is the president of our organization, but he didn't feel that he was competent to talk about the research program.

Mr. MAGUIRE. Well, the subcommittee had no information, Dr. Hockett, until this moment that Mr. Yeaman was not going to be present. He had been invited. He has been listed as a witness. To

the knowledge of the members of the subcommittee and staff, until this moment, he was going to be here.

Now, did he just decide to take it upon himself not to appear, after having informed the subcommittee that he was going to be here?

Dr. HOCKETT. Well, the message I had from him was that all he could contribute to this was a little historical sketch of how this organization came into being, which I can do.

Mr. MAGUIRE. Well, it is a very curious situation, because ordinarily the Congress makes decisions about whom it is that it would like to have testify rather than the persons themselves deciding, sometimes with a few hours' notice, as in Mr. Kornegay's case, and sometimes with no notice whatsoever, as in Mr. Yeaman's case, that they have nothing to contribute to the discussion.

I find it rather astonishing.

Dr. HOCKETT. That is all I know about the whole thing.

Mr. MAGUIRE. I find it rather appalling, as a matter of fact. You are going to give us the little historical sketch that Mr. Yeaman would have given us had he been here?

Dr. HOCKETT. Right.

Mr. MAGUIRE. Which, of course, begs the question as to whether the committee might have had some ideas of its own or some questions of its own about what it would like to ask Mr. Yeaman.

Dr. HOCKETT. I will try to answer them, if there are such questions.

Mr. MAGUIRE. Well, let us proceed, and we hope you will be able to answer them.

Dr. Hockett.

Dr. HOCKETT. I am Robert Casad Hockett, research director of the Council for Tobacco Research—U.S.A., Inc. I have been employed by the council and its predecessor, the Tobacco Industry Research Committee, since 1954, first as associate scientific director, then as acting scientific director, and in my present capacity.

Because of this long-term connection, I have been asked by my associates to appear in response to your letter of September 27, 1978, addressed to Dr. William U. Gardner, scientific director, and received by him on Monday, October 2. I am happy to respond to this request. My curriculum vitae is attached to this statement [see p. 9].

In January 1954, there was formed an unincorporated organization called the Tobacco Industry Research Committee, later changed to incorporated form as the Council for Tobacco Research—U.S.A., Inc. The membership includes segments of virtually the entire tobacco industry from the growers to the manufacturers. The charter of the council states the purpose for which it was formed, which was "to aid and assist research into tobacco use and health and to make available to the public factual information on this subject." That statement of purpose has been the governing policy from 1954 to the present time.

The scientific leadership of the council is in a scientific director, assisted by myself as research director and several qualified scientific staff members. The scientific director is presently Dr. William U. Gardner, formerly head of the Department of Anatomy of the

Yale University School of Medicine and formerly president of the International Union Against Cancer.

The responsibility for entertaining and evaluating applications for research grants or proposals for contracts rests in a scientific advisory board constituted, from the inception of the Tobacco Industry Research Committee to the present time, of highly distinguished scientists in the disciplines relevant to the investigation of the possible relationship between smoking and human health.

I should say at this point that the Council does not own or operate any research facility. Grants-in-aid for research are made to independent scientists who are assured complete scientific freedom in conducting their research, in the conclusions they reach, and in the publication or nonpublication of their work.

The membership of the scientific advisory board is presently as follows:

Sheldon C. Sommers, M.D., chairman, director of laboratories, Lenox Hill Hospital, clinical professor of pathology, College of Physicians and Surgeons of Columbia University, New York, N.Y.;

Richard J. Bing, M.D., director of cardiology and intramural medicine, Huntington Memorial Hospital, Pasadena, Calif., professor of medicine, University of Southern California School of Medicine, Los Angeles, Calif.;

Joseph D. Feldman, M.D., head, Department of Immunopathology, Scripps Clinic and Research Foundation, La Jolla, Calif.;

William U. Gardner, Ph. D., scientific director, the Council for Tobacco Research—U.S.A., Inc., E. K. Hunt professor of anatomy (emeritus), Yale University School of Medicine, New Haven, Conn.;

Robert J. Huebner, M.D., Chief, Laboratory of RNA Tumor Viruses, National Cancer Institute, Bethesda, Md.;

Leon O. Jacobson, M.D., former director, the Franklin McLean Memorial Research Institute, Joseph Regenstein professor of biological sciences, University of Chicago, Chicago, Ill., and just recently he retired from the deanship of the university's medical school;

Henry T. Lynch, M.D., professor and chairman, Department of Preventive Medicine and Public Health, Creighton University School of Medicine, Omaha, Nebr.;

Hans Meier, D.V.M., D.M.V., M.R.S.H., senior staff scientist, the Jackson Laboratory, Bar Harbor, Maine;

Lee W. Wattenberg, M.D., professor of pathology, Department of Laboratory Medicine and Pathology, University of Minnesota Medical School, Minneapolis, Minn.; and

John P. Wyatt, M.D., director, Tobacco and Health Research Institute, University of Kentucky, Lexington, Ky., and is and was before receiving that appointment a distinguished and well-known student of pulmonary diseases.

This board meets at least twice a year to consider and evaluate applications for grants and proposals for contracts. The criteria applied are scientific merit and relevance to the question of the relation of smoking to health. Subject only to the Council's commitment to engage in no activity affecting the business conduct of its members, the scientific advisory board and the scientific director enjoy full freedom and discretion in respect to the research for which grants should be made.

All funds for this research are provided by the member companies.

By the end of December 1977 the Council and its predecessor, the Tobacco Industry Research Committee, had approved projects for 365 principal investigators in 243 medical schools, hospitals, and research institutes. These awards by January 1, 1978, had totaled more than \$46 million and had produced more than 1,500 reports published in scientific journals. The research budget for 1978 is approximately \$6.25 million.

The Council's successive annual reports contain abstracts of all published papers prepared by the respective grantees. As I said, all grantees enjoy complete freedom in respect to their research and, indeed, the full decision as to publication. We have previously furnished the subcommittee the Council's annual reports for 1975 and 1976. I now offer for the subcommittee's use a copy of the annual report for the year 1977.

At one of the early meetings of the scientific advisory board it was agreed that major emphasis should be placed upon study of the major diseases reported by epidemiological studies to be associated statistically with tobacco use. These included cancer, especially carcinoma of the lung, heart and artery diseases, and somewhat later, chronic lung ailments such as emphysema and bronchitis. Though numerous other diseases of less importance have received some attention, these priorities have been maintained.

The disorders enumerated are all constitutional diseases. They generally develop slowly, are associated with aging and influenced by genetically based predispositions. Strong emphasis was to be placed upon elucidating the pathogenesis of these diseases; that is, the steps and stages by which progression from a normal condition to a disease condition occurs.

At present these steps are still quite obscure and this obscurity remains as a serious block to early recognition, rational treatment, identification of highly susceptible individuals, and determining whether, how, to what extent, under what circumstances, and in whom any particular external influence, whether a food, food ingredient, atmospheric contaminant, medicine, dye, cosmetic, tobacco smoke or other influence, could contribute significantly to the process.

These guidelines are still in force. Their applications to studies of respiratory function and chronic disease conditions have been discussed in the Council's annual report for 1975. Applications to studies related to cardiovascular function and diseases are discussed similarly in our 1976 annual report. Copies of these have already been furnished. For that reason, I will not go into detail on those at the moment.

I would like, however, to comment a little further on cancer research and the problem of carcinogens and carcinogenesis. Our scientific advisory board has consistently questioned the relevance of mouse-skin painting with tobacco smoke condensates, usually called tars, to the problem of human lung cancer.

The Board's conviction is that the goal of producing an animal model, involving long-term inhalation of fresh whole smoke under highly controlled and standardized conditions could be made far more relevant than such skin painting. We have devoted millions

of dollars and a great deal of time and attention to the creation of inhalation equipment and related technology, including means of measuring dosage. Developments toward this goal have been substantial and work is continuing.

We now know that carcinogenesis is a process rather than a single event. A whole series of sequential steps must occur in a normal body cell to bring about its conversion to a cancer cell and make its unrestrained multiplication possible. At each step the body has defenses for reversing or blocking these changes toward cancer as we know it clinically. A great deal of progress has been made at the level of biochemistry in describing what happens in some of these steps and stages toward cancer and in describing the defense mechanisms. But there is still a long way to go.

In recent years it has become quite apparent that there are scores of substances in the environment that possess only one or a few of the biochemical activities concerned in this process of cancer production and that many of these activities are very weak. To define the activities of such feeble and partial possible contributors to the carcinogenesis process and to evaluate their significance in real life situations is difficult. Nevertheless, it must be attempted.

Tobacco smoke must be studied in the context of these concepts if the applications that have been drawn from epidemiology are to be tested realistically.

Pioneer studies supported by the Council are being conducted with highly inbred mouse strains that can be defined biochemically in terms of certain susceptibility and resistance systems. These are described in annual reports.

The nearest human equivalent to litter mates in inbred mouse strains occurs in identical twins, since such twins have an identical genetic makeup. For that reason, the Council is assisting some studies aimed at getting a better picture of the relative importance of heredity and environmental influences in the origins of cancer and in other diseases, too. Also under study are familiar groups—kindreds—in which cancer is prevalent, in contrast to other kindreds in which it is virtually absent.

Cardiovascular diseases are also recognized as having a complex pathogenesis comprising a series of stages strongly influenced by genetic predispositions but influenced as well by environmental factors. As described in our 1976 report, identification of the first essential lesion that initiates the process of arterial wall thickening and degeneration is a particular goal. Elucidation of the biochemical processes involved in these progressive changes and the influences that promote or defend against them is another. Study of identical twins is also underway to evaluate the relative importance of heredity and environmental factors in generating or promoting these conditions. Tobacco use is, of course, included in these investigations. Our 1976 annual report describes this work.

It is appropriate to mention here the contributions of the tobacco industry to a number of independent, scientific studies of smoking and health besides that of the Council for Tobacco Research. The Council has, of course, been interested in these programs, has been kept informed of their progress, and visits have been made by members of the Council staff. But we have had no responsibility for their conduct.

The chairman's letter to Dr. Gardner specifically referred to the report of the American Medical Association Education and Research Foundation. As that report stated:

Throughout its life, the committee maintained liaison with the National Institutes of Health and the Council for Tobacco Research, U.S.A., keeping these organizations apprised of current trends in the committee's work and to minimize duplication of effort.

The report, not itself a research study, includes a useful compilation of abstracts of papers published as a consequence of AMA-ERF support. Nearly all the original papers were published in the scientific literature prior to 1975. The research publications from the AMA-ERF program deserve careful consideration. The AMA-ERF committee is entitled to summarize its impressions of the program's overall implications.

I notice, however, that it did not attempt any judgments as to whether smoking contributes to the initiation or development of atherosclerosis, the condition widely thought to underlie or augment most manifestations of heart and artery diseases.

Scientists are well aware that while specific facts and observations accumulate at a prodigious rate, their integration into plausible integrative hypotheses is slow. Broad perspectives must prove their durability by surviving the tests of time and experience.

[Dr. Hockett's curriculum vitae referred to follows:]

CURRICULUM VITAE OF DR. ROBERT C. HOCKETT

Dr. Robert C. Hockett, Research Director of The Council for Tobacco Research - U.S.A., Inc., 110 East 59th Street, New York, New York 10022, was born in Fayette, Missouri on July 1, 1906.

A graduate of the Ohio State University, Dr. Hockett pursued graduate study in the same institution and received the Ph.D. degree in chemistry there in 1929. As a National Research Council Fellow in Chemistry, he was a guest scientist at the National Institutes of Health, U. S. Public Health Service from 1929 to 1931 and then Associate Chemist on the Institute staff from 1931 to 1935.

In 1935 he joined the faculty of the Massachusetts Institute of Technology as Assistant Professor of Chemistry, becoming Associate Professor in 1941.

In 1943 he was granted a leave of absence from M.I.T. to serve as Scientific Director of the Sugar Research Foundation, Inc., which position he occupied until 1952. He has also been Visiting Professor at the Universities of Illinois and North Carolina.

From 1952 until 1954 he served as a consultant to industrial firms on problems relating to foods, nutrition, pharmaceuticals, fermentations and sponsored research.

In 1954 he joined the present Council for Tobacco Research's predecessor organization - the Tobacco Industry Research Committee - as an Associate Scientific director.

Dr. Hockett is a Fellow of the American Academy of Arts and Sciences, the New York Academy of Sciences, the American Public Health Association, the Royal Society of Arts, and the American Institute of Chemists. He holds membership in the American Chemical Society, in which he has served as an Alternate Councilor, member of the Nomenclature Committee, Vice-Chairman of the Division of Carbohydrate Chemistry in 1944, Chairman in 1945 and 1946, and Secretary-Treasurer from 1956 to 1960. He also holds membership in the American Society of Biological Chemists, American Association for the advancement of Science, Friends of the World Health Organization, Royal Society of Health, and the Phi Beta Kappa Associates.

He has served as a Lecturer in Switzerland for the American-Swiss Foundation for Scientific Exchange, member of the Food Industries' Advisory Committee to the Nutrition Foundation, Inc., Collaborator to the United States Department of Agriculture, member of the Advisory Committee for Advances in Carbohydrate Chemistry, as Associate to the State Department's Committee for Interamerican Scientific Publication and member of the Sugar Advisory Committee to the Administrator of the Production and Marketing Act, U.S. Department of Agriculture.

He is author of numerous research papers on chemistry of the carbohydrates, of articles and lectures on nutrition and public health, of many reviews and summaries on tobacco and health research and contributor to a book, Beet Sugar Economics.

Mr. MAGUIRE. I am afraid we are going to have to respond to a call to the floor of the House. We will be back very shortly, if you would kindly not disappear while we are gone.

[Brief recess.]

Mr. MAGUIRE. Doctor, I wonder if you would just tell us, by way of summary, do you agree that there is a statistical association between smoking and lung cancer?

Dr. HOCKETT. I am glad you asked that because that brings up a point that I think is very important.

I would say yes, there is a statistical association, but that is only the beginning of the question.

Mr. MAGUIRE. Is there a statistical relationship between smoking and chronic bronchitis?

Dr. HOCKETT. Apparently there is.

Mr. MAGUIRE. Do you believe there is?

Dr. HOCKETT. I believe there is.

Mr. MAGUIRE. Do you believe there is a statistical association between smoking and emphysema?

Dr. HOCKETT. Yes.

Mr. MAGUIRE. The 1976 publication of "Health Consequences of Smoking" indicates, on page 627, that carbon monoxide concentrations in cigarette smoke are in the range of 5,240 to 21,400 micrograms per cigarette and that nicotine concentrations are in the range 200 to 2,400 micrograms per cigarette. Nicotine is the substance that we think of much more frequently as being present in cigarettes and being harmful. I want to ask you whether you are aware that carbon monoxide has qualities which are deadly to the human beings?

Dr. HOCKETT. I would like to comment further on that matter of the concentration, if I may.

Mr. MAGUIRE. Surely.

Dr. HOCKETT. When a man takes a puff from a cigarette, that is usually about 35 milliliters. This is regarded as a "standard" puff. He takes one of those perhaps every minute or so for 10 minutes.

If a man smokes three whole packs of cigarettes in that manner, and inhales 10 puffs from every cigarette—which is unusual—and you figure out how much the total smoke he has inhaled, it is a very, very small percentage of the total air that he inhales during the course of 24 hours. It figures out to about fifteen one-hundredths of 1 percent.

Hence, it is not important what the concentration of the carbon monoxide is in that relatively small amount of smoke that he inhales. You have to compare the total carbon monoxide he could get by smoking with what he will acquire by breathing room air containing much lower concentrations but which are considered acceptable if breathed continuously for many hours.

There are tolerated allowances for industrial exposures on the basis of an 8-hour day.

Now, you see the quantity of smoke inhaled, even with heavy smoking, is very, very small as compared with the amount of ambient air we breathe during the course of a day. So, you have to consider the relative concentrations in air and smoke in terms of the relative volumes that are inhaled.

It isn't concentration that counts, but it is how much you get altogether in the course of an hour or a day. That is a question poorly understood by many commentators.

Mr. MAGUIRE. But there is carbon monoxide in the cigarette and in the cigarette smoke.

Dr. HOCKETT. Of course.

Mr. MAGUIRE. And carbon monoxide is a deadly substance to human beings.

Dr. HOCKETT. Well, that depends, now. That depends. You have got to qualify that. It is not a poison in the same sense that lead is a poison, that accumulates and destroys tissue in the liver.

Mr. MAGUIRE. But if you put yourself in a car and lock yourself in and let the carbon run into the car, you die, right?

Dr. HOCKETT. You can be killed by that. It is just like drowning. Is water poisonous? Is it deadly? It is deadly in your lungs—

Mr. MAGUIRE. That is a very unscientific statement, Doctor, as I am sure you recognize.

Dr. HOCKETT. It is not. That is a very good analogy. Carbon monoxide can, under certain conditions, be deadly. But you cannot just make a categorical statement and say it is deadly.

What it does is to join with the red blood corpuscles and get in the way so that the oxygen cannot get into that particular spot. It crowds out some of the oxygen. If you wait a bit, it will come back out again and the blood corpuscle is as good as new.

So, it is not a deadly poison in the sense it is destructive. It just gets in the way for a brief period. You have got to consider the nature of any poison and how it works in order to reach any judgments.

So, I cannot assent to any sweeping statements. I think water is a good analogy. Water in the wrong place in the body is deadly. Yet, it is indispensable. Carbon monoxide is not indispensable.

Mr. MAGUIRE. The information that we have on cigarettes by brand, indicates that there are enormous differences in the amount of carbon monoxide that is present in the different brands. For example, King Sano Menthols have 13.6. Carlton has 2.6. Is that difference something we should be concerned about?

Dr. HOCKETT. Well, I don't know anything about the brands, and I don't know how sound these figures are. I have understood that the output of carbon monoxide in current brands has been decreasing steadily over the years. That is just hearsay. We have not studied this.

Mr. MAGUIRE. You mean you are in charge of studying these questions for the Council and you don't know anything about that information?

Dr. HOCKETT. We don't study brands, individual brands.

Mr. MAGUIRE. But it is individual brands and individual cigarettes that people actually smoke.

Dr. HOCKETT. That is right, but we have to lay down the basic principles and facts, and then the industry has to apply those to its manufacturing practices as they see fit.

We are not allowed under antitrust laws to work on individual brands or even to consider them. We work on basic science that is relevant to their problems.

Mr. MAGUIRE. But basic science would also, I am sure, be interested in whether there was a different impact on the individual smoker smoking one cigarette which had more than 10 times the amount of carbon monoxide in it than another one had.

Dr. HOCKETT. I will repeat again, we can only study the effects of various concentrations of carbon monoxide in the air over various periods of time on the lungs and the bodies of animals and human subjects. But we would not concern ourselves in terms of brands at all.

Mr. MAGUIRE. We have hydrogen cyanide, don't we, as one of the constituents of cigarettes?

Dr. HOCKETT. It is known to be present in very small traces probably more or less comparable to the amounts that are present in various liqueurs, such as Kirschwasser. We know that in very small concentrations hydrogen cyanide is a respiratory stimulant.

Mr. MAGUIRE. It helps to make you breathe better, is that it?

Dr. HOCKETT. In tiny, tiny quantities. So, here again this is one of those things you can only evaluate in terms of the quantities that are involved. This has been known for a long, long time.

I don't think anybody has ever really shown that it has the slightest bit of deleterious effect on the body. It is rapidly converted by metabolism to thiocyanate which increases slightly the amount of thiocyanate that is ordinarily present in the saliva already.

Mr. MAGUIRE. But again, cyanides in too great a quantity kill people.

Dr. HOCKETT. You see now we are getting into the quantitative aspect, which is crucial here. We can detect its presence in the smoke by the modern analytical methods at lower and lower levels down to picograms, millionths of a millionth of a gram.

So, we know these things, but we have to evaluate their effects in terms of how much you could possibly get of them.

Mr. MAGUIRE. Would you recommend, then, that everybody have a little hydrogen cyanide each day?

Dr. HOCKETT. I don't think it would do any harm if they do. I am sure we do, most of us.

Mr. MAGUIRE. Have a little carbon monoxide?

Dr. HOCKETT. Well, yes, we can tolerate a lot of that. It has been shown that in the natives of New Guinea, there is a high percentage, from the levels of carbon monoxide in their huts. They breathe this. The cavemen had it, too.

Carbon monoxide is formed in the swamps, in the marshes, and is present in the ambient air. A certain amount of it is formed in our own bodies by the degradation of the blood pigments.

So, it is something we live with, but of course—and the amounts, the quantities that are put out by vehicles in the street are very, very much larger than the levels that you can get even by heavy smoking.

Mr. MAGUIRE. Maybe we should all have a little bit of tar and a little bit of nicotine each day.

Dr. HOCKETT. Well, it isn't that. But you must realize we all live in a sea of poisonous substances and our bodies are equipped by adaptation through evolution to choose the things we need and to discard the things we don't.

The environment is never ideal for human life. The body has to choose and select the things it wants. There are all kinds of things that are not desired by the body that are cast off. Now, the thing is we must try not to overtax the system, you see.

Mr. MAGUIRE. You referred to the poisonous environment. I take it you are saying that cigarettes contribute to that poisonous environment?

Dr. HOCKETT. Well, so do a great many other things.

Mr. MAGUIRE. But cigarettes are among them?

Dr. HOCKETT. I guess we have to say they are among those things.

Mr. MAGUIRE. They are among the poisonous substances in our environment.

Dr. HOCKETT. Well, now, I am not going to put it that way.

Mr. MAGUIRE. Well, you just did. I am just trying to tack it down.

Dr. HOCKETT. You are tacking it down. I was speaking not only of "poisonous" substances in the environment but of substances not needed by the body and therefore excluded or discarded by it. The term "poisonous" as usually understood would not apply to most of these.

Mr. MAGUIRE. Well, fine, we will look at how it comes out in the record.

Dr. HOCKETT. For that matter, there are poisons in cabbage, in tomatoes, in potatoes. These things are thrown off by the bodies of normal people.

Mr. MAGUIRE. All right. You know carbon monoxide increases the heart rate; in other words, it makes the heart work harder than normal.

Dr. HOCKETT. Right.

Mr. MAGUIRE. It decreases the oxygen which is available to the heart and to the blood vessels. It slowly and consistently, over many years, adversely affects the heart. In that sense, it is what you might call a slow poison.

This, together with other factors in cigarette smoke, is thought to be related to the over 160,000 cardiovascular disease casualties that we have in this country. People who are just plain dead as a result of cardiovascular disease.

Dr. HOCKETT. I think you are linking two things that may not be related at all. As a matter of fact, carbon monoxide has a very minimum of chronic effects. It comes and it goes.

Mr. MAGUIRE. Well, once the heart is damaged, it is awfully hard for the heart to come back, isn't it?

Dr. HOCKETT. But there is very little evidence, if any, that carbon monoxide damages the heart.

Mr. MAGUIRE. Well, there is some evidence that it does.

Dr. HOCKETT. I don't really know of any cogent evidence on that subject. I don't like to make a sweeping statement because somebody might dig up an obscure little note somewhere which might contradict it.

Mr. MAGUIRE. Which might be accurate.

Dr. HOCKETT. I don't think carbon monoxide is regarded in that sense as having any damaging effects. All it does do really is to cut down somewhat the amount of available oxygen that is circulating in the blood. And—

Mr. MAGUIRE. That is certainly not a good thing, is it?

Dr. HOCKETT. Well, this happens all the time. A reasonably healthy person has a great tolerance, he has a great deal of reserve in the capacity of his lungs.

If you are a little bit anemic, that cuts down the amount of oxygen being delivered to your organs and so on. But this carbon monoxide has a very temporary effect unless you are breathing it constantly, all the time.

Mr. MAGUIRE. All I can tell you, Doctor, is that——

Dr. HOCKETT. I think this can be misjudged very easily, its significance.

Mr. MAGUIRE. I do a little jogging from time to time. I sometimes jog with other people. They tell me that once they have started jogging they simply cannot continue to smoke because they cannot breathe. The body cannot do the work that it is supposed to be able to do.

Dr. HOCKETT. That is a matter of what you might call training. Training is just the capacity, the efficiency, of the body in transporting oxygen to the tissues. Now, you tax it when you run. So this builds up tremendously on experience and training. That is what training is, repetitive experience.

There was a study on that involving smokers and nonsmokers, and the smokers increased their tolerance to exercise at the same rate as the nonsmokers——

Mr. MAGUIRE. OK. Let me turn to——

Dr. HOCKETT [continuing]. With exercise.

Mr. MAGUIRE. Let me turn to another issue here. When an individual is making up his or her mind about whether or not to smoke, and there are these dangers, which you have described——

Dr. HOCKETT. Have I described some dangers?

Mr. MAGUIRE [continuing]. Which are well known.

Dr. HOCKETT. What dangers are those? I don't remember describing them.

Mr. MAGUIRE. Do you think that a person ought to have all of the scientific information that we have at their disposal? For example, that carbon monoxide, in certain concentrations, is a poison? That cyanide, in certain concentrations, is poisonous, et cetera? Would you have any objection to having all of these items listed on the package and displayed in the stores so that people can know exactly what they are smoking?

Dr. HOCKETT. I might have some objections to that because the ordinary citizen would not be able to judge the significance of these things in the amounts in which they are present. I don't think it would be really helpful. I don't see any reason why he should be told how much cyanide there is.

Mr. MAGUIRE. You would rather have them ignorant of the facts?

Dr. HOCKETT. I would rather have—if there are warnings, I think it would be perfectly proper to tell people about any risks that are known, as long as that is done strictly objectively and scientifically, and they are left to make their own decisions as to whether they can balance the benefits against the so-called hazards, and find a benefit.

There are benefits, too, that have been described. I am not insisting that they are true.

Mr. MAGUIRE. Name one.

Dr. HOCKETT. Well, one is that smoking relieves anxiety and certain tension and so on. Some people consider this very important. There are others that testify it assists in digestion. It has been shown definitely it does stimulate peristalsis in the intestinal tract, which may be an important thing.

There are reports, too, there are many reports—this may not be very important, but actually, there is less Parkinson's disease among smokers than there is among nonsmokers. They have even tried using cigarettes as a therapeutic measure for people with Parkinsonism.

Then there are such things as—I had another one in mind. Aphthous ulcers. That is not a very important disease, but people who smoke have fewer aphthous ulcers. These are not important things, really, many of them, but these are well-documented effects of smoking.

There are a great many people who really believe it is of tremendous help to them emotionally and from the standpoint of tension. Included among these people was Sigmund Freud. In fact, he could not work effectively without using tobacco.

Mr. WAXMAN. Would the gentleman yield.

Sigmund Freud died of cancer of the mouth because of smoking didn't he?

Dr. HOCKETT. That is right. He didn't smoke cigarettes.

Mr. WAXMAN. What did he smoke?

Dr. HOCKETT. Cigars.

Mr. WAXMAN. What would be the scientific difference?

Dr. HOCKETT. There is quite a lot of difference between cigarette and cigar smoking.

Mr. WAXMAN. Could we have for the record some of the studies you have which show the differences?

Dr. HOCKETT. We have not really done much on that. The work on the composition of smoke has been done mostly by experienced and skillful tobacco chemists.

Mr. WAXMAN. I would like a citation—I would like the record held open for a citation from you of the scientific studies—

Dr. HOCKETT. You want citations on this? Well, a lot of this work on cigars has been done by your own Department of Agriculture.

Mr. WAXMAN. You are a scientist and you are asserting with a great deal of positiveness that cigar smoke is hazardous to health and may cause—

Dr. HOCKETT. I didn't say it was hazardous to the health.

Mr. WAXMAN. You are saying it is different, and it is hazardous, and cigarette smoking is not.

Dr. HOCKETT. I haven't said it is hazardous to the health. I am saying it is different. There is more nicotine in most cigar smoke.

Mr. WAXMAN. Do you think there is a relationship between nicotine and adverse health effects?

Dr. HOCKETT. No, I don't see—I haven't been able to see any relationship. Bear in mind—

Mr. WAXMAN. I am taking the gentleman's time. I will have an opportunity to question you later. But I do want this information from you. It seems to me that you have stated so cautiously that

we don't know for sure about the dangers of cigarette smoking, but you said with assurance you know there are dangers with cigars.

Dr. HOCKETT. No; I am not saying that. I am saying they are different. We haven't nearly as much data on the composition of cigar smoke. We don't know as much about it. It is harder to analyze this.

There is not the interest in cigar smoking because epidemiologically, for example, it doesn't seem to show up by statistical associations, which I don't admit are causal, it does not show up as nearly dangerous as cigarette smoke.

Yet, there is as much more or many things in that smoke, including nicotine. That problem has never been really cleared up. It is something we are striving to do.

If you will define what you would like to have, I will do my best to supply it.

Mr. WAXMAN. I will send you a letter based on your statement, and I will ask for more information. But you stated with such assurance that Sigmund Freud was raised as a person who benefited from smoking. I point out the fact that he died of cancer of the mouth, which most everyone acknowledges was from smoking.

Dr. HOCKETT. I don't know whether it was from smoking or not.

Mr. WAXMAN. You think it was from pine nuts?

Dr. HOCKETT. No, I don't suppose I could. Nobody would know. However, the point I was making was that he found this to be an important stimulus to his mental faculty. He felt it was. I think there is some significance in that. That was my only point.

Now, what killed him, and whether he would have got cancer of the mouth anyhow, I don't know. At any rate, cancer of the mouth is not even significantly connected with cigarette smoking. I think there is a very small statistical relationship.

I hope I might have a chance to speak a little later about statistical associations because that is a very important subject.

Mr. MAGUIRE. Well, perhaps it is also an aphrodisiac. Perhaps it also reduces the tendency towards aging. I mean, there may be all sorts of magical things that result from smoking.

If it in fact helps the digestive process, as you suggested, with peristalsis, and if it in fact is useful in treating Parkinson's disease or relieving some of the symptoms, would you recommend that it be treated then as a drug and regulated as a drug for those purposes?

Dr. HOCKETT. No; not at all.

Mr. MAGUIRE. Why not?

Dr. HOCKETT. Well, we have other drugs that will do these same things if you want to use drugs for that purpose.

Mr. MAGUIRE. So we could do the same things with other substances which did not have all of the dangerous, hazardous, killing side effects that cigarettes have?

Dr. HOCKETT. Well, I haven't said that they have any. The point is that part of this is probably the circumstances under which a man smokes a cigarette. He relaxes while he is doing it, after breakfast, and the combination of things, his enjoyment of it, that all helps his digestion.

Mr. MAGUIRE. Recently, Secretary Califano announced that it has been found that asbestos workers, for example, as they relaxed

after breakfast, increased the likelihood that they would die of cancer some 30 times over as a result of that smoking. Are you familiar with that statistic and do you agree with that?

Dr. HOCKETT. That again is epidemiological evidence on which I would like to make further comments if the opportunity arises.

Mr. MAGUIRE. And you are aware that there have been, in the last couple of years, some very interesting findings with respect to nitrosamines, one of the most potent human carcinogens, and that nitrosamines are found in cigarette smoke?

Dr. HOCKETT. We have been at this now for many years. They are found, but they are just barely identifiable in cigarette smoke at extraordinarily low concentration.

We could not possibly have identified them a few years ago until these extraordinarily sensitive analytical methods had been developed.

Furthermore, it is well known that the analysis of cigarette smoke for nitrosamines is a very touchy thing because they can form in the smoke after it is concentrated and condensed in a condenser vessel. Nitrosamines may form that were not present in the fresh smoke as it goes into the lung. So, this is a very difficult subject.

Mr. MAGUIRE. Well, you raise a very—

Dr. HOCKETT. It is barely detectable. We have other studies on the biochemical level, to find out how much it takes to have any effect on a mammalian creature. That, of course, is the other part of the problem.

As I say, as I warned before, we can find in smoke all kinds of things at extremely low levels. We have to learn to distinguish which ones are of any consequence in that combination.

Mr. MAGUIRE. Well, perhaps we will be able to make the necessary, refined distinctions after hundreds of thousands more Americans have died.

Dr. HOCKETT. That is the only way we are ever going to handle the question of environmental cancer. We are up against it. You know that OSHA and all the rest of these organizations are very concerned about how do we determine how dangerous this, that and the other substance is.

As I mentioned, many of these are very weak and feeble. But, they may add a straw somewhere to the load. It is a tough problem. We are all in it together.

It just happened that tobacco is one of the first things that got the spotlight. But now we have it all over the map—perfumes, hair sprays, you name it.

You are aware of the confusion that exists and the battles that are going on as to how much is tolerable. We cannot escape these things. We might as well realize we cannot escape them. So, we have to try to—

Mr. MAGUIRE. Should we just throw up our hands and say we are not going to worry about these problems and that it doesn't help to minimize our exposures?

Dr. HOCKETT. Well, we have to try to develop quantitative methods so that we can evaluate their relative effects, and we have got to balance the hazards against the benefits, and that is tough. I said that to the American College of Angiology 25 years ago, and it

is still true. That is—the problem it can't be solved quickly. We might as well face it.

Mr. MAGUIRE. Does it bother you at all that people are dying in the meantime?

Dr. HOCKETT. It bothers me all the time that people are dying. I have lost many friends and relatives. But I have not been convinced in any case that tobacco use had anything to do with it.

Mr. MAGUIRE. You really are not convinced that tobacco use has anything to do with people dying?

Dr. HOCKETT. With the people that I am speaking of. Or if you mention a particular disease, no, a particular disease, I think the causal relationship is very greatly clouded, for the reasons that I stated here.

We have not worked out what the steps and stages of development of these diseases are, and so we have no way to test these things.

Mr. MAGUIRE. Everybody, except the Tobacco Institute and the Council on Tobacco Research, has accepted the causal relationships.

Dr. HOCKETT. I don't think so. I know a great many scientists who do not.

Mr. MAGUIRE. That is the evidence that the committee has before it.

You raised an interesting point about nitrosamines when you said that nitrosamines are not present in tremendously large amounts in the smoke that is inhaled initially, nor are they formed in the body. However, we have no threshold of safety for nitrosamines, since they are such a potent carcinogen. Moreover, scientists, Hoffmann and Hecht specifically, have found that side stream smoke, which is exhaled by the smoker and may then be inhaled by other nonsmokers, has 20 to 50 times the concentration of nitrosamines originally contained in the smoke that the smoker took in. What do you think the implications of that are for the public health?

Dr. HOCKETT. What do you mean by 20 to 50 times? You mean the total amount that came from the cigarette or the per cubic centimeter or cubic foot of air in the room?

Now, the report I read alleged that a person, bystander in the room where somebody was smoking, might pick up in a certain number of hours the amount of nitrosamines equivalent to that which the smoker got.

That might be true. But the point is nobody has shown that the smoker picks up a quantity that has any consequence at all health-wise. Remember, now, we are getting down into the picograms. A few years ago we could not even have discovered that there was any there.

Does that increase the danger? We have to judge these things in another way, not so much what is there, but what the effect of the total smoke is on a suitable experimental animal, and in very carefully controlled conditions.

We have to consider the effect of the total smoke. That has not been shown in any animal to produce lung cancer.

Mr. MAGUIRE. The findings are that nitrosamines are present in cigarette smoke. Nitrosamines are very dangerous, cancer-causing substances—

Dr. HOCKETT. They are present in beer, too, and bacon.

Mr. MAGUIRE. Would you wait a moment, please?

Dr. HOCKETT. I am sorry.

Mr. MAGUIRE [continuing]. Which are present in higher concentrations in the smoke exhaled by the smoker than in the smoke that is inhaled by the smoker.

These are the scientific findings. Do you, Doctor, have any findings to the contrary? Have you done any research which suggests that either of those statements is not true?

Dr. HOCKETT. No. What I have to say is that I—

Mr. MAGUIRE. The answer is yes or no. Have you done any research which shows that those things are not true?

Dr. HOCKETT. We haven't done any research—we have done research on the formation and mechanisms of formation of nitrosamines in smoke, and the duration of their life in smoke.

We know, too, that nitrosamines are formed in the body. The ingredients for forming nitrosamines are present in beer, in bacon and so on, and even saliva normally contains nitrates.

So, if you eat anything that includes amines, nitrosamines can be formed in the body. If you drink beer and eat fish you probably have the materials for making nitrosamines.

Mr. MAGUIRE. But they are present also in the tar, are they not?

Dr. HOCKETT. If the tar is allowed to become stale, if it is kept. That is one reason that we are skeptical of the use of tar in experiments to try to evaluate what tobacco smoke can do to animals.

Tar is not smoke. It has changed chemically. One of the changes that can take place is the formation of nitrosamines while it is stored and kept around. You have to deal with fresh smoke that is in the condition that it is in when a human smoker inhales it, which is within a fraction of a second from the time it formed in the cigarette.

This has been totally ignored in all the work that is done with so-called tar painted on the skins of mice.

As I said in my presentation, we have no faith in that work. We have gone to great expense to try to develop suitable equipment for bringing about the inhalation of absolutely fresh, whole and normal cigarette smoke by very carefully selected animal strains, about whose biochemistry we know something. I think that conclusions have got to wait for this.

Mr. MAGUIRE. Until a mouse is a human being, you won't believe any of the test results?

Dr. HOCKETT. No. As I indicated before, by studying human beings we are trying to find the biochemical resemblances and differences between humans and mice, so that we can hope eventually to bridge that gap.

Mr. MAGUIRE. After hundreds of thousands of more people have died as a result of poisonous substances you acknowledge are in the cigarette.

Dr. HOCKETT. I don't feel they are dying as a result of this. Smoking is—

Mr. MAGUIRE. You don't think a single person has died as a result of smoking a cigarette?

Dr. HOCKETT. I don't say that. I just don't think we have any quantitative picture as of now of what the effect of this is. The reason for that is that statistical associations do not demonstrate a casual relation.

Mr. MAGUIRE. You said earlier they did.

Dr. HOCKETT. No, I didn't.

Mr. MAGUIRE. You said that there were statistical associations.

Dr. HOCKETT. I said there are associations. I admitted the existence of associations. But we started out our program here, on our advisory board—we had Edwin B. Wilson, who was probably the most outstanding statistician in the country at this time. He was on the board of the American Cancer Society.

From the beginning he said we must remember the statistical association does not prove cause. It only proves these two things are paralleling one another. You have to eliminate all other possible causes.

Mr. MAGUIRE. That would have been fine, Doctor, 20 or 30 years ago before all of the scientific evidence had been put in place and before the Surgeon General and all other reputable scientists had concluded there were causal relationships.

Dr. HOCKETT. They did not—

Mr. MAGUIRE. Let me go on, please. I am not prepared to continue to listen to an unscientific statement on this subject.

Mr. Kornegay told the Health and the Environment Subcommittee last February that this industry does not want children to smoke. Now, would you agree that the industry does not want children to smoke and that children perhaps shouldn't smoke?

Dr. HOCKETT. I saw that statement. I believe he was sincere in making it. I can only guess what his basis is. There have been studies, particularly with rats, that show when a child or a rat, a young rat is maturing, is growing fast, it is using a lot of aminoacids for building a lot of protein, muscle, and so on.

It was shown in studies with rats that nicotine presumably and perhaps other ingredients in smoke reduced somewhat the efficiency of the production of muscle and other body tissues from these amino acids.

But as soon as the animal matured, then its requirement for protein was reduced and the nicotine had no effect. So, that kind of thing I suppose is the basis of his statement.

It is better with young children who are rapidly building muscle and other tissue not to have the influence of smoke superimposed upon that. But it no longer seems to hold once maturity has been reached. I think there is good evidence for that.

Mr. MAGUIRE. The gentleman from California.

Mr. WAXMAN. Thank you, Mr. Chairman.

I feel a real sense of frustration in questioning you, Dr. Hockett. We are debating a subject over whether cigarette smoking is hazardous to health.

If you are sincere in your belief that cigarette smoking is not hazardous to health, I think you are probably one of the few people in the country that believes that. I think most smokers believe cigarettes are hazardous to their health.

I think the evidence is becoming so overwhelming that while we have not had conclusive proof, because there is no such thing as conclusive proof, the evidence is so overwhelming that people cannot ignore the evidence.

Mr. Chairman, my frustration is that rather than debate the question of whether the 1964 Surgeon General's report first said cigarette smoking is hazardous, I would like to have talked to Mr. Kornegay from the Tobacco Institute to discuss why an increasing number of teenage girls are smoking, why young people seem to be picking up this habit, and what the tobacco industry is doing to promote this trend.

It seems to me the question we ought to be dealing with is not whether cigarette smoking is hazardous to health. I just cannot believe in my own mind that anybody can sincerely say to us that there is no evidence that cigarette smoking is hazardous to your health. I think that issue has been resolved.

What I would have wanted to discuss with the tobacco people is why their advertising seems to be directed in a way to show that vigorous, exciting and happy people are smoking cigarettes; why the image is being portrayed in a way that indicates it is a badge of maturity; and whether this advertising is in fact directed at increasing smoking among teenagers. It is the younger age group that is increasing smoking while cigarette usage among older age groups is decreasing.

I have some advertisements. It is not anything new. I am sure everybody has seen them. These are just from one issue of Newsweek magazine. A woman and a man smoking a cigarette, bicycling, smiling, looking very delighted.

A man with a lasso on a horse—what could be more vigorous than that—with a cigarette dangling out of his mouth. A woman smiling—I don't think she could look any happier. A man, who certainly is one of the most virile looking men around, with a cigarette. A woman with a little badge or sign under her eye that she would rather smoke cigarettes than switch.

I would like to find out why this advertising campaign is going on, despite evidence to the contrary that cigarette smoking is not going to produce happy, healthy people.

But you are not the one to answer these questions. You are a researcher.

Dr. HOCKETT. That is right.

Mr. WAXMAN. You are a scientist. You are really, I must say this with all due respect, the flak-catcher for the tobacco industry, sent before this congressional committee so nobody else from the Institute would come to answer our questions.

We are sitting here debating with you whether cigarette smoking is hazardous to health. You are telling us there is some evidence, but it is not conclusive. And we are arguing about nitrosamines and carbon monoxide. I think it is the most absurd argument I have ever heard.

Your council has just funded a study by the American Medical Association, and the American Medical Association came out with a statement.

Dr. HOCKETT. No, we didn't, that wasn't in our hands at all. I commented on that before. The industry made grants to the Ameri-

can Medical Association, and they set up an organization somewhat like ours and a committee and had a program.

Mr. WAXMAN. But the tobacco industry sent money to the AMA and said study this question. The AMA says, "The committee believes that the bulk of research sponsored by this project supports the contention that cigarette smoking plays an important role in the development of chronic obstructive pulmonary diseases and constitutes a grave danger to individuals with pre-existing diseases of the coronary arteries."

The AMA is acknowledging the health risks. The scientists on that study overlap with the scientists that are in your research group.

Dr. HOCKETT. There was one common member on the board.

Mr. WAXMAN. Did you agree with that statement?

Dr. HOCKETT. You notice that they say supports the contention. They are entitled to their opinion, that it supported a contention that cigarette smoking involves that, but I don't—

Mr. WAXMAN. Aren't we really being semantically cute? "Supports the contention" means that the belief most people have that cigarette smoking causes health problems, is substantiated by some evidence. You are saying that it hasn't been proved and, therefore, we don't know for sure.

Dr. HOCKETT. Well, I would put it this way. Of course, it is not my job to draw conclusions. What we try to do is to provide an atmosphere where scientists can do constructive work, and I do believe that science progresses by having people challenge one another and point out exceptions and problems and things that don't agree.

That is the way science progresses. We have got to have free discussion, comparing notes, challenge, defense, and so on. It moves slowly. I think we forget how slow it is.

Mr. WAXMAN. I express my frustration now because I really don't want to debate the subject with you. If you believe cigarette smoking is not hazardous to your health, you are entitled to your belief. But I think you are one of the few people still believing that.

I think most cigarette smokers would disagree with you. People smoke cigarettes because it is addictive. Do you agree with that statement?

Dr. HOCKETT. I think it is perhaps, well, people get—I don't know what addictive means. But I think that you somehow adjust to smoking. Remember, it used to be when a person smoked the first time he got nauseated. After a while that didn't happen anymore.

Whatever that reflex is, it changed. So, in that sense I think there is an adjustment.

Mr. WAXMAN. Do you believe there is a dependency?

Dr. HOCKETT. Well, there is a dependency on a lot of things. I depend on stewed apricots pretty strongly.

Mr. WAXMAN. Do you find your dependence on stewed apricots is scientifically equivalent to most peoples' dependence on cigarettes?

Dr. HOCKETT. I just don't know. People miss things they are accustomed to using. I think that is a very tough question. I don't know really what addiction is.

Mr. WAXMAN. You are a scientist. Maybe you haven't done the research. Don't you really have doubts that your desire for stewed

apricots is greater than the dependency most people seem to place upon smoking?

Dr. HOCKETT. I don't know. I just cannot answer that.

Mr. WAXMAN. Mr. Chairman, I really don't want to ask any more questions of this witness. I wanted to ask Mr. Kornegay why the industry advertises cigarette smoking as desirable for young people.

I want to see the tobacco industry's market studies. They evidently are looking to sell cigarettes and they are finding an increasing market with young people.

I want to talk to Mr. Kornegay about the lobbying of the tobacco industry. The Federal Trade Commission could not even get an authorization bill through the Congress because there was such heavy lobbying against it, and the Federal Trade Commission is looking into some of these same questions.

I want to find out about the relationship between smoking and children and what kind of programs can be developed to combat this. This witness is not a witness that I can ask any of those questions.

I feel that I have been used. I feel that the tobacco industry has thumbed its nose at the U.S. Congress and I quite frankly think that Mr. Kornegay ought to be subpoenaed and brought before this committee to answer some of these questions, and not just to be excused by sending a flak-catcher who will deny any knowledge of the important questions that we want to discuss.

If this committee can stand up to members of the President's Cabinet because we believe that Congress has a right to know certain facts, then I think we ought to stand up to the tobacco industry and demand that they come before us and talk about how much money they are spending on advertising, whether it is directed to increasing children's smoking and whether they are lobbying Members of Congress and policymakers to protect their financial interests.

These are questions that I think we as Members of Congress have a right to know and the American people have a right to have answered in a public forum.

Mr. MAGUIRE. Thank you, Congressman Waxman.

The committee would like to know if there is a representative of the Tobacco Institute present in the room. Is there? If there is, would that person please identify himself? No one?

Mr. WAXMAN. We obviously know no one is coming. Mr. Kornegay has canceled out on us. We only had three witnesses and they have all canceled out on us, except this one gentleman who is telling us all he does is research and his research hasn't finally established the fact that cigarette smoking is hazardous to your health.

Well, that is just a wonderful debate. I just think it is a useless waste of our time and his. I gather you are being paid for it and I guess we are, too. I don't see that any of us are serving any useful purposes, at least for us, anyway, at taxpayers' expense.

I suppose you are serving a useful purpose for your employer coming here today. But I don't think that should excuse the tobacco industry from having a representative to answer the important

questions that we have to ask rather than this discussion of whether cigarette smoking is hazardous.

Mr. MAGUIRE. Well, as the gentleman well knows, sometimes interest groups monitor proceedings, although they have declined to participate in them.

Mr. WAXMAN. I wouldn't doubt half the audience is here from the tobacco industry.

Mr. MAGUIRE. Mr. Segal, committee staff, has some questions.

Mr. SEGAL. Dr. Hockett, I would first ask for elaboration of the statement you made in response to the chairman's question before about children not smoking. Your answer was with respect to the proposition that probably children's muscular functions were not quite mature at that point.

Could you elaborate on what you mean by that?

Dr. HOCKETT. I don't really think I can elaborate very much further. I just pointed out that during the period when new muscle tissue and other organs containing protein, when these are forming, an organism, whether it is a human or an animal, is much more sensitive to many outside extraneous influences.

It is just good sense to let the maturity come about before various things are done.

Mr. MAGUIRE. Various insults to the body?

Dr. HOCKETT. I don't know whether you would call them insults or not. We drink beer. We think that is not harmful. In Europe children drink beer. But here we feel they ought to be mature before they drink beer.

Mr. SEGAL. But cellular changes are what you are getting at. There are in fact cellular changes by cigarette smoke on muscular cells.

Dr. HOCKETT. I just put it this way. It has been shown with rats, anyway, that young growing rats need more protein than ones that have reached maturity.

It seems as if, if they are exposed to smoke, they require still a little more protein than the ones that are not exposed to smoke. If you give them enough protein, there is no difference between the smoking and unsmoking rats in size, growth, or maturation is concerned. They need a little more protein.

Mr. SEGAL. Would you suggest, then, that one of the things that might be part of the educational program that the Tobacco Institute has agreed ought to be done for children would include that those children who smoke ought to be given "a little more protein"?

Dr. HOCKETT. Well, I think—I don't know what the Institute ought to do. But I feel that it is very important that children in general should get adequate protein.

Mr. SEGAL. But a little more. You would recommend that children who smoke get a little more protein than children who don't smoke.

Dr. HOCKETT. All children should get a quantity of protein that is really ample and a little extra. A great many children are on the margin. It may be, quite well be, that some of the damages that are reputed to be due to smoking are just due to the fact that they are not adequately nourished with respect to protein. Otherwise, it might not have any effect on them.

I don't know the answer to that. I have not seen a study that would show whether that is the case or not.

Mr. SEGAL. OK. One further followup question to Mr. Waxman's. He read you the conclusions from the AMA's ERF study that talked about coronary artery diseases, and he also read the conclusion about chronic obstructive pulmonary disease.

Do you agree with the conclusions that the AMA group reached?

Dr. HOCKETT. As I remember it, they said that they believed that people with severely damaged cardiovascular systems, for them smoking would be hazardous. There probably are people like that, but I don't know how bad it would have to be to be a hazard.

You are getting there into the area of individuals. We cannot make any judgments about whether you, you, or you is for some special reason, because of your own condition, facing a special hazard from smoking.

That sort of question has to be handled by your individual physician that looks at your condition and your capacities and your responses and your adjustment and advise you. We never get into anything like that. We have to look at the mass and we cannot deal with the individual. That would be my statement about that.

As far as a chronic pulmonary disease is concerned, in our study of that—we are studying this quite intensively—we know that there is a small subfraction of the population that for genetic reasons are highly susceptible to this disease.

It looks as if smoking would be rough on them. But there is a great majority of other people—

Mr. SEGAL. For those people who do have this kind of preconceived whatever it is, genetic or otherwise, would smoking for them represent a grave danger.

Dr. HOCKETT. And we think it is very important to learn how to identify such people.

Mr. SEGAL. But you would agree with that conclusion. For those individuals, who for genetic or other reasons, have preexisting coronary artery diseases, that smoking presents a grave danger to them.

Dr. HOCKETT. I would say there probably are people for whom it is a grave danger. I don't know where you draw the line or how you draw it. But I think it would be rather extreme conditions.

Mr. SEGAL. OK. What about the issue of chronic obstructive pulmonary disease, or lung disease? Do you agree with the conclusion reached by the AMA?

Dr. HOCKETT. I mentioned that I think that most chronic pulmonary, obstructive pulmonary disease is in people who have a congenital predisposition to that disease. It is a question of the balance between protease and antiprotease.

Mr. SEGAL. Fine. We will get into that in a little bit. There was a report issued by the Department of Health, Education, and Welfare in 1976 known as the "Health Consequences of Smoking."

It indicated that clinicians—they reviewed studies and found that clinicians have long observed that the majority of their patients suffering from chronic obstructive pulmonary diseases were cigarette smokers.

Do you have any kind of countervailing studies or evidence to the contrary?

Dr. HOCKETT. Well, we have not concentrated on surveys or population studies of that kind. As I mentioned——

Mr. SEGAL. So, the answer to that would be no?

Dr. HOCKETT. The answer would be no. We are trying to find out the steps and stages by which this disease comes about so we can find out who is under risk and see if they can be helped.

Mr. SEGAL. Could I assume you are familiar with the numerous studies that are also referenced in here as well, that demonstrate cigarette smokers are more likely to suffer from cough and sputum production and dyspnea than nonsmokers?

Dr. HOCKETT. I must say that I have not studied this particular volume thoroughly. I try to follow these annual reports. I have heard such allegations.

Mr. SEGAL. Let me cite one case. You referred in your testimony to twins. On page 231 of this report there was a study by Cederlof, et al., published in 1969, that looked at 4,379 twin pairs, where nonsmoking prevalence of respiratory symptoms was 4.3 as opposed to those who smoked 10 to 30 cigarettes a day, having a prevalence of 15.3, a factor of more than three times as much.

Are you aware of any studies where nonsmokers and smokers have been found to have either the same amount of symptoms of cough and sputum production?

Dr. HOCKETT. Well, you are just talking about some symptoms. Now, those symptoms are not—they don't correspond necessarily to serious pulmonary diseases. These are relatively superficial symptoms you are talking about.

I remember Cederlof's work. We are assisting some of that work.

Mr. SEGAL. Are you a medical clinician?

Dr. HOCKETT. No; I am not.

Mr. SEGAL. Have you been examining people's medical histories and medical records to be able to know whether physicians identify certain items as what you call symptoms, cough, sputum production?

Dr. HOCKETT. Dr. Cederlof tells me he regards these just as symptoms of irritation, that sort of thing. We know there is a certain amount of irritation.

Mr. SEGAL. What about dyspnea?

Dr. HOCKETT. That is shortness of breath. I don't remember that as being involved in that study.

Mr. SEGAL. It was one of the findings. If you would like, I could give you the article to look at and review.

Dr. HOCKETT. Well, I would be agnostic about this anyway.

Mr. SEGAL. As opposed to everything else?

Dr. HOCKETT. Cederlof is glad he found this difference because he was doing this study on identical twins and nonidentical twins. He found that at least there was a difference in cough between, say, the smoking twin and the nonsmoking twin, and that encouraged him because it meant they were probably smoking, he was smoking enough to show up something.

But the important thing, the thing he was after was to see in the identical twins, one of whom smoked and the other didn't, what happened to their cardiovascular system.

Now, there are not too many identical twins——

Mr. SEGAL. We are not talking about cardiovascular. We will get to that. What I would like to pursue—

Dr. HOCKETT. I cannot comment on that. I am sure that cough and sputum are supposed to be connected with smoking. But whether that reflects—

Mr. SEGAL. That is a clinical finding. People can measure that. Physicians can acknowledge a difference, right?

Dr. HOCKETT. Yes.

Mr. SEGAL. OK. Do you agree with the conclusion that the 1976 HEW report came to, which was that cigarette smoking is the most important cause of chronic obstructive pulmonary disease?

Dr. HOCKETT. Well, I would have to be agnostic about that because that is a quantitative value judgment. I am not in a position without a lot of review, research, to make a statement about that.

Mr. SEGAL. You have never done—

Dr. HOCKETT. But an association, I don't think we could deny there is some association.

Mr. SEGAL. You would acknowledge a statistical association, you would acknowledge that it is a cause, but not the most important cause?

Dr. HOCKETT. I don't know whether it is a cause or not.

Mr. SEGAL. OK. Well, the AMA's report said that it, cigarette smoking, played an important role in the development of. Would you go so far as to agree with that statement?

Dr. HOCKETT. I might agree if it said in some individuals. We have no idea how numerous they are or how many there are of them.

Mr. SEGAL. In some individuals. In comparisons between twins, in comparisons between populations.

Dr. HOCKETT. No, just in the population. There are some individuals apparently who have a high susceptibility. We are trying to define that. They are a small minority. But it would be a good idea to identify them and suggest they don't smoke. There are such people.

Mr. SEGAL. The susceptibility—have you identified any of the factors in the susceptibility?

Dr. HOCKETT. We think we are in the process of identifying these.

Mr. SEGAL. What would you categorize—

Dr. HOCKETT. It is a question of the enzymes in the lung. There are enzymes there whose responsibility it is to kill organisms. They have to be in the lung to kill invading organisms.

Yet, those enzymes are so potent that if they escape into the lung they can damage the lung. There are antiproteases that are supposed to stop the action of these enzymes before damage of the lung.

It is like the old question of why doesn't the stomach digest itself. We can eat tripe, we can digest tripe, but the stomach doesn't digest its own wall, which is composed of the same sort of thing as tripe.

The reason for that is that there are antienzymes that are on hand to cut off the process at the right point. This is the picture in the lung.

No, we have concentrated our attention on trying to delineate that picture of what goes on, and where these enzymes are, and

what determines their secretion, and which ones, and where this happens, all that sort of thing. That is described in our annual report for 1975.

Mr. SEGAL. We have read that. You have no problem accepting that generally emerging and prevailing scientific hypothesis of the day about the delicate balance between proteases and antiproteases, is that right?

Dr. HOCKETT. We find that a very good working hypothesis. What we don't know yet is to see whether inhalation of smoke has anything to do with the way these enzymes are secreted or the antielastases are secreted. That is under study now.

Mr. SEGAL. I assume you have reviewed many of the studies that have been carried on in this particular area for you to have had them emerge in your annual report, to the position that you agree that there is this kind of prevailing hypothesis.

Are you aware of what is generally described as the small airways disease?

Dr. HOCKETT. Yes. That is substantially—well, that is similar to emphysema.

Mr. SEGAL. Do you believe there is such an entity known as the small airways disease?

Dr. HOCKETT. Well, I guess as the experts define it, I presume there must be.

Mr. SEGAL. Do you have a different definition?

Dr. HOCKETT. No.

Mr. SEGAL. So you define small airways disease in the same category as—

Dr. HOCKETT. I don't define it. I mean, I follow the definitions.

Mr. SEGAL. You accept the prevailing definitions of small airways disease?

Dr. HOCKETT. Yes.

Mr. SEGAL. Are you aware of the 1968 study by Anthonisen which examined lung volumes for airway closure and also looked at lower lung diseases? He used a marked xenon, I think it was 133, in patients.

Dr. HOCKETT. I don't remember that paper. But there have been many such studies. There has been much discussion about the proper way to measure and detect small airways disease, to measure closing volumes. This has been a very controversial subject, whether that is a good measure or not.

But the thing that I gather from the lung physicians who are engaged in that is that in general lung function in these terms, these physiological terms, tends to deteriorate with age.

Now, to find out the rate at which it gradually slopes off, you have to make measurements successively over about a 3-year period to see what the slope of that relationship is.

Mr. SEGAL. Let's look at the situation not as it applies to the elderly but let's look at children.

Dr. HOCKETT. I am getting to that.

Mr. SEGAL. Could I sharpen your getting to it by asking a specific question? Are you aware of the study in 1971 published by Seely and others in Science where 331 students, with 1 to 5 years of smoking experience, reported to have objective evidence of lung disease?

Dr. HOCKETT. I don't remember that paper. As I have indicated, I would have to scrutinize that very carefully to see how these measurements were made.

Now, they have to be done on a time sequence. Immediately after inhalation of any aerosol, including smoke, there are brief responses that change these measurements. So, you have to have sequential measurements over a period of time in order to see whether there is any lasting effect.

We have such a study going on ourselves. But the methodology is very exacting. Without recalling to mind what the methodology was, in that paper, I could not comment on it.

Mr. MAGUIRE. We will recess while we go to vote.

[Brief recess.]

Mr. MAGUIRE. The committee will come to order. Mr. Segal?

Mr. SEGAL. Dr. Hockett, you were given a copy of the study in order for you to get a chance to check the methodology to see if you think it is scientific.

Dr. HOCKETT. I don't feel I have had an opportunity really to give it enough consideration. I would appreciate the opportunity to take it with me, and if you had some specific questions you want to ask, I would attempt to give you answers.

Mr. SEGAL. The specific question would be, do you have any evidence in your possession by research that you funded that might lead to countervailing conclusions than the ones found here?

Dr. HOCKETT. I noted the question, and I will try to answer it.

[Testimony resumes on p. 40.]

[The following letter and attachment was received for the record:]

THE COUNCIL FOR TOBACCO RESEARCH—U.S.A., INC.

110 EAST 59TH STREET
NEW YORK, N. Y. 10022

November 3, 1978

The Honorable Andrew Maguire
 Subcommittee on Oversight and Investigations
 Committee on Interstate and Foreign Commerce
 U.S. House of Representatives
 Rayburn House Office Building (Room 2125)
 Washington, D.C. 20515

Re: Testimony of Dr. Robert C. Hockett During
 Hearing on Cigarette Smoking and Health
 Before the Subcommittee on Oversight and
 Investigations of the House Committee on
 Interstate and Foreign Commerce, October 5,
 1978.

Dear Congressman Maguire:

As promised, I am responding to Mr. Segal's question to me during the above-mentioned hearing, with respect to a certain study by Dr. Seely published in 1971. The colloquy concerning this is found at pages 85-87, lines 1583-1599 and 1607-1620 of the typed transcript of the hearing.

Mr. Segal stated, "The specific question would be, do you have any evidence in your possession by research that you funded that might lead to countervailing conclusions than the ones found here?"

The paper referred to by Mr. Segal was entitled, Cigarette Smoking: Objective Evidence for Lung Damage in Teenagers by Janet E. Seely, Eugenija Zuskin and Arend Bouhuys, in Science for May 14, 1971.

While I am unable to find Council-sponsored or other research that specifically countervails the conclusions of this paper, I am also unable to find any research that confirms them. Further, the significance of the conclusions is quite limited. Buist, et al. (1976) reported the results of their study on the effects of smoking cessation on lung function in the American Review of Respiratory Disease. This research, which was funded by the National Institutes of Health, concluded that the lung function of smokers significantly improves upon the cessation of smoking and that the kind of respiratory symptoms reported by Seely, et al. completely disappear.

If I may be permitted, however, I would appreciate the opportunity to add some comments on this paper in the context of my testimony on this subject.

It is noteworthy, I think, that the data on "excessive cough, sputum production, and shortness of breath" are all derived from questionnaires filled out by the teen-agers. While questionnaires have to be used in many clinical studies for want of any alternative, they are recognized to be difficult to

appraise because of their subjective nature. They are therefore regarded as "soft data" to be used with caution and due recognition of the possibility of substantial error. I am sure these reputable investigators are quite cognizant of this situation as the generally cautious tone of their article indicates. It is clear, I trust, that none of these symptoms is specifically diagnostic of any particular disease. While "shortness of breath" does occur in advanced stages of emphysema it is also seen in a great many other situations both serious and trivial.

The inhalation of any finely divided material: dusts, aerosols, etc., including tobacco smoke, will briefly increase "airway resistance" which shows up on instruments that are used to measure physiological functions of the lung. The measurements are also affected by slight colds, other infections, sulfur dioxide in the air, etc., etc. It therefore requires very careful control of conditions to get good replicable quantitative information on the basic physiological competence of the lung. Often the same subject must be measured many times over a short period in order to obtain such data.

Lung function declines with age. Because of the situation mentioned, many clinicians now feel that the same patient must have repetitive measurements over a period of three years at least to get a valid picture of his rate of decline. True emphysema, which is a progressive and irreversible disease, will be reflected in an abnormally steep rate of decline. By screening very large numbers of presumably healthy people, male and female, of different types of body build, tables of "normal" functional data have been developing. Attention has been heretofore placed particularly on older age groups, and males, because so many of the patients have been in this category.

Good reference data on "normal" lung function in children have been lacking. Methodologies for measuring lung function are still in a developmental stage. Suggestions, simplifications, tests, trials, debate, criticism and controversy have been abundant.

The Seely article clearly describes the assumptions that had to be made in setting up standard values for use in conducting and interpreting the instrumental methods used in this case. She did this in a reasonable manner, but obsolescence is rapid in this area.

The question whether smoking affects maturation of the lungs had to be left open by the authors. But, the later research of Buist, et al. suggests that no permanent lung damage occurs.

In addition, I enclose copies of certain pages of the transcript of my testimony, upon which I have corrected typographical errors. The addition to the transcript of my testimony at line 2215 of page 118 is a result of the reporter's transcribing only part of what I was saying when my testimony was interrupted by adjournment of the hearing.

I should appreciate it if you would have the record corrected in accordance with the enclosure and also have this letter included in the record of the hearing.

Yours very truly,

Robert C. Hockett

Robert C. Hockett, Ph.D.
Research Director

The Effect of Smoking Cessation and Modification on Lung Function^{1,2}

A. SONIA BUIST,³ GARY J. SEXTON,⁴ JERROLD M. NAGY, and BENJAMIN B. ROSS

SUMMARY

The purpose of this study was to obtain more information about the effect on lung function of stopping smoking or of modifying the smoking habit and to determine the time course of change. We followed a group of 75 cigarette smokers who attended a smoking cessation clinic in May 1973, using a respiratory symptom questionnaire, spirometry, closing volumes, and the slope of the alveolar plateau of the single-breath nitrogen test. Subjects were tested before stopping smoking and at 1, 3, 6, and 12 months after the initial testing. We found a significant ($P < 0.05$) improvement in closing volume as a percentage of vital capacity and closing capacity as a percentage of total lung capacity at 6 and 12 months and in the slope of the alveolar plateau at 1, 6, and 12 months in those who stopped smoking. There was also a dramatic decrease in respiratory symptoms in those who stopped smoking, a moderate decrease in those who reduced their consumption by at least 25 per cent, and very little change in those who did not appreciably modify their smoking consumption.

Introduction

Little is known about the effect on lung function of stopping smoking or of modifying the smoking habit. Until 5 years ago, few studies had been reported that documented functional changes in smokers who stopped smoking. In one of the earlier studies, Krumholz and co-workers (1) reported a significant decrease in

the functional residual capacity and a significant increase in specific airway conductance and maximal voluntary ventilation in a group of young smokers who stopped smoking. In 1967, Wilhelmssen (2) reported that the 1-sec forced expiratory volume (FEV_1) and maximal expiratory flow at 75 per cent of the vital capacity increased significantly and airway resistance decreased significantly in 16 symptomatic smokers 40 days after stopping smoking. The following year, Peterson and associates (3) reported an improvement in the FEV_1 and mean maximal mid-expiratory flow ($MMEF_{25-75}$) in 12 smokers 18 months after stopping smoking.

The advent of the new tests of peripheral airway function, such as frequency dependence of compliance and closing volumes (CV), brought the hope that it would now be feasible to measure the early functional changes in smokers and the more subtle functional changes that occur after smoking cessation (4). Using these techniques, usually in association with more conventional measurements of lung function, several studies have been carried out in the past 5 years. Ingram and O'Cain (5) reported that in 6 asymptomatic young smokers who demonstrated

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¹ From the Departments of Medicine and Physiology, University of Oregon Health Sciences Center, Portland, Ore. 97201.

² This research is part of the Collaborative Study of Smoking and Airways Obstruction being carried out in Portland, Oregon; Montreal, Quebec; and Winnipeg, Manitoba. Supported by a contract from the Division of Lung Diseases, National Heart and Lung Institute, National Institutes of Health—U. S. Public Health Service.

³ Recipient of a Research Career Development Award from the Division of Lung Diseases, National Heart and Lung Institute.

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July

frequency dependence of compliance (dynamic lung compliance as a percentage of static lung compliance) before stopping smoking, values returned to the normal range within 8 weeks of smoking cessation. They concluded that structural changes in the peripheral airways of young smokers are reversible after cessation of smoking. Shortly thereafter, McFadden and Linden (6) demonstrated that the FEV_1 and MMEF increased, airway resistance decreased, and frequency dependence of compliance returned to the normal range in 3 subjects who stopped smoking. In another study, Martin and co-workers (7) noted that frequency dependence of compliance returned toward the normal range in 8 of 12 smokers 2 months after stopping smoking.

In another recent paper, Dirksen and associates (8) reported a study of heavy smokers and nonsmokers who were all born in Malmö in 1914. Twenty-nine of the smokers succeeded in stopping smoking for 2 months. At the beginning of the study, the smokers were significantly different from the nonsmokers in spirometric tests, CV, and tests of uneven distribution of ventilation (lung clearance index and washout volume). After 2 months of abstinence, the smokers showed no change in spirometric tests or CV but did show a significant improvement in the tests of uneven distribution of ventilation.

In the most detailed and recent study reported or the effect of stopping smoking on peripheral airway function, Bode and co-workers (9) studied 10 subjects between 6 and 14 weeks after stopping smoking and found increases in maximal expiratory flow rates with helium-oxygen at 50 per cent and 25 per cent of vital capacity, reductions in the lung volume at which the maximal expiratory flow rates of air and helium became identical, and decreases in CV. These in-

vestigators found no change in the static pressure-volume curves, slope of the alveolar plateau, or air flow rates after smoking cessation.

Differences between the various reported studies as to which tests show significant change with smoking cessation and as to the time course of change can be attributed largely to differences between the populations in variables, such as age, sex, extent and nature of disease, and frequency of testing. No investigator, to our knowledge, has succeeded in following a large number of subjects for a long period immediately after they have stopped smoking. Conclusions are therefore being drawn in each case from a relatively small number of subjects who may differ appreciably within one study group and from group to group.

The purpose of our study was to obtain further information about the effect of stopping smoking or reducing cigarette consumption on lung function as measured by spirometric tests, CV, and the slope of the alveolar plateau, and to examine the time course of change.

Materials and Methods

The subjects for this study were drawn from among persons attending a Seventh-Day Adventist Five-Day Plan to Stop Smoking held in Portland, Oregon in May 1973. Seventy-five persons of a total enrollment of 112 (67 per cent) elected to participate in the study, which, they were informed, would span a one-year period. Both men and women were included, with an age range of 21 to 63 years. At the end of the study, the subjects were divided into 3 groups: (1) a "Smokers" group, which consisted of persons who did not stop smoking, i.e., who were smoking at every test occasion; (2) an "Intermittent" group, which consisted of persons who stopped smoking initially for at least one month but started smoking again one or more times during the year; and (3) a "Quitters" group, which consisted of those who stopped smoking for the entire year. For the

TABLE 1
DESCRIPTION OF GROUPS BY SEX, AGE, AND AMOUNT SMOKED

Group	Men (no.)	Women (no.)	Age* (years)	Smoking Habit* (pack-years)†
Smokers	9	16	36 (21-53)	23 (4-54)
Intermittents	18	19	39 (23-63)	26 (2-70)
Quitters	6	7	35 (24-53)	20 (1-84)

* Mean, range given in parentheses.

† One pack-year = 20 cigarettes per day for one year.

TABLE 2
MEAN STANDARD SCORE* FOR VARIOUS MEASUREMENTS
BY GROUP AT INITIAL TESTING

Group	FEV ₁	MMEF ₂₅₋₇₅	ΔN ₂ /L	CV/VC	CC/TLC
Smokers	-0.18	-0.66	1.38	1.36	1.45
Intermittents	0.09	-0.25	0.79	1.03	1.21
Quitters	0.01	0.25	0.64	0.50	0.83

Definition of abbreviations: FEV₁ = 1-sec forced expiratory volume; MMEF₂₅₋₇₅ = mean maximal mid-expiratory flow; ΔN₂/L = slope of the alveolar plateau (Phase III); CV/VC = closing volume as a percentage of expired vital capacity; CC/TLC = closing capacity as a percentage of total lung capacity.

*Standard score (z) = $\frac{\text{observed value} - \text{predicted value}}{\text{standard error of the mean}}$

purpose of evaluating the effect of reduced cigarette consumption on lung function, we also defined a group of "Modifiers." This excluded the Quitters but included all those from the other 2 groups who had reduced their cigarette consumption at each test period by at least 25 per cent of the initial amount smoked.

The subjects were tested 5 times: before stopping smoking, and at 1, 3, 6, and approximately 12 months after the initial testing. Testing consisted of a respiratory symptom questionnaire, spirometry, and the single-breath N₂ test for the measurement of CV. The questionnaire used in the initial test was the National Heart and Lung Institute (NHLI) respiratory symptom questionnaire recommended for epidemiologic studies (10). On return visits questionnaires designed to elicit changes in symptoms from the immediately preceding test occasion and from the initial test occasion were used.

Spirometry involved 3 maximal forced expiratory maneuvers performed on a Vitalograph wedge-type spirometer for the measurement of forced vital capacity (FVC), FEV₁, and MMEF₂₅₋₇₅. The largest value for each measurement was taken. The FVC of the curve from which the MMEF was derived had to be within 200 ml of the largest FVC.

The single-breath N₂ test was performed in triplicate in accordance with the standardized procedure for the measurement of CV, as recommended by the NHLI (11). All single-breath curves were read

by the same observer without the knowledge of the group to which a subject belonged.

Closing volume was taken from the single-breath curve as the volume between the abrupt increase in N₂ concentration and residual volume (RV) and was expressed as a percentage of the expired vital capacity (VC) taken from the single-breath N₂ curve, i.e., CV/VC%. Closing capacity (CC) was taken as CV plus RV; RV was derived by subtracting the expired VC from the total lung capacity (TLC). The TLC was estimated from the single-breath N₂ curve by planimetric determination of the area under the N₂ versus volume curve, followed by application of the alveolar dilution equation as described previously (11, 12). Closing capacity was expressed as a percentage of TLC, i.e., CC/TLC%. The distribution of ventilation was determined by measuring the slope of the alveolar plateau of the single-breath curve, i.e., increase in N₂ concentration per liter, ΔN₂ (%)/L. For each measurement obtained or derived from the single-breath curve, a mean was obtained from the technically acceptable single-breath curves. The criteria for acceptance of a curve were those described in the standardized procedure (11).

All volumes, both from spirometry and the single-breath N₂ test, were corrected to BTPS.

A standard score (z) was calculated for each individual measurement at the initial test period using the equation: $z = (O - P) \div \text{SEM}$, where O was

TABLE 3
MEAN CHANGE FROM INITIAL TESTING IN 1-SEC FORCED
EXPIRATORY VOLUME (FEV₁) AND MEAN MAXIMAL MID-EXPIRATORY FLOW
(MMEF) AT EACH TEST PERIOD

Time from Initial Testing (months)	FEV ₁ (Δ, liter)		MMEF ₂₅₋₇₅ (Δ, liter/sec)	
	Smokers	Quitters	Smokers	Quitters
1	-0.01	-0.04	-0.04	-0.06
3	-0.08	-0.07	-0.37*	-0.11
6	-0.02	0.03	-0.18*	0.04
12	-0.03	0.06	-0.28*	-0.16

*Significant (P < 0.05) by t test.

the observed value, P was the predicted value, and SEM was the standard error of the mean. This corrected for sex, age, and height differences. There is a normal probability distribution for z with a mean equal to zero and a standard deviation equal to one. The predicted values and SEM were from Morris and associates (13) for spirometry and from Buist and Ross for CV (12) and the slope of the alveolar plateau (14). Measurements that were more than 2 standard scores below the predicted value for spirometry or more than 2 standard scores above the predicted value for CV and $\Delta N_2/L$ were considered abnormal.

No attempts were made to validate a subject's statement about smoking habits at each test occasion by using objective measurements, such as carboxyhemoglobin concentrations. Care was taken, however, not to project a condemnatory attitude, with the expectation that this would help the subject give a truthful answer to questions relating to smoking habits.

After each test, the subject was informed of spirometry results but was not given any results for the single-breath N_2 test.

Results

The 3 groups are described in terms of age, sex, and amount smoked at the initial testing in table 1. The mean and range for age were very similar for the Smokers and the Quitters. The Intermittent group had a slightly higher mean age and a few more older subjects than the other 2 groups. The smoking habits were slightly different in the 3 groups, with the Intermittents tending to smoke the most and the Quitters the least. The group means for pack-years, however, were not significantly different.

To determine whether the 3 groups differed

in pulmonary function at the onset of the study, a mean standard score was determined as described previously for each test and for each group (table 2). It can be seen that at the start of the study there were differences between the groups for all tests except FEV_1 . In each case, the Smokers had the worst pulmonary function, i.e., the group mean was lower for spirometric variables and higher for CV/VC, CC/TLC, and $\Delta N_2/L$, and the Quitters had the best pulmonary function. The percentage of subjects considered abnormal initially was very similar in the Smokers and the Quitters. For the Smokers, 5 of 25 were abnormal in one or more of the spirometric tests and 8 of 25 were abnormal in one or more of the measurements derived from the CV test. For the Quitters, 2 of 13 were abnormal in the spirometric tests and 4 of 13 were abnormal in the CV test.

The mean changes from the initial values for FEV_1 and MMEF for the Smokers and the Quitters at each testing are shown in table 3. Neither of the groups demonstrated a significant change in FEV_1 at any of the test periods. The only significant mean change ($P < 0.05$) by t test was seen in the Smokers group, which showed a deterioration in MMEF, as compared to the control value, at 3, 6, and 12 months. The FVC is not shown because no significant changes were seen in any group throughout the year.

Mean changes from initial values in CV/VC, CC/TLC, and $\Delta N_2/L$ for the Smokers and the Quitters are shown in table 4. For CV/VC, both groups showed a trend toward improvement. This change was significant ($P < 0.05$) by t test in the Smokers only at 3 months and in the Quitters at 6 and 12 months.

TABLE 4

MEAN CHANGE FROM INITIAL VALUES IN CLOSING VOLUME AS A PERCENTAGE OF VITAL CAPACITY (CV/VC), CLOSING CAPACITY AS A PERCENTAGE OF TOTAL LUNG CAPACITY (CC/TLC), AND SLOPE OF THE ALVEOLAR PLATEAU (PHASE III) ($\Delta N_2/L$) IN SMOKERS AND QUITTERS

Group	Time from Initial Testing (months)	CV/VC (Δ , %)	CC/TLC (Δ , %)	$\Delta N_2/L$ (Δ , %)
Smokers	1	-0.6	0.3	-0.0
	3	-2.5*	0.8	0.4
	6	-1.5	-2.3*	-0.1
	12	-1.9	0.2	0.1
Quitters	1	-1.6	-0.8	-0.3*
	3	-1.9	1.6	0.0
	6	-4.1*	-5.7*	-0.4*
	12	-3.6*	-2.6*	-0.3*

For definition of abbreviations, see table 2.

*Significant ($P < 0.05$) by t test.

TABLE 5
MEAN CHANGE FROM INITIAL VALUES IN CLOSING VOLUME (CV),
VITAL CAPACITY (VC), AND TOTAL LUNG CAPACITY
(TLC), IN SMOKERS AND QUITTERS

Group	Time from Initial Testing (months)	CV (Δ , liter)	VC (Δ , liter)	TLC (Δ , liter)
Smokers	1	-0.00	0.04	0.07
	3	-0.11*	-0.13	0.06
	6	-0.08	0.06	-0.23*
	12	-0.09*	-0.06	-0.04
Quitters	1	-0.06	-0.04	0.01
	3	-0.11	-0.31	-0.10
	6	-0.15*	-0.12	-0.24*
	12	-0.15*	-0.07	-0.05

*Significant ($P < 0.05$) by t test.

For CC/TLC the Smokers showed a significant improvement ($P < 0.05$) at 6 months but no appreciable change from the initial value at the other test periods. The Quitters showed a significant improvement ($P < 0.05$) at 6 and 12 months.

For $\Delta N_2/L$, no significant change was seen in the Smokers at any test period and no trend was apparent. In the Quitters, there was a trend toward improvement, with significant improvement ($P < 0.05$) at 1, 6, and 12 months.

Because changes in CV/VC and CC/TLC may result from changes in both the numerator and the denominator, we also determined the mean changes from initial values for each group in CV, VC, and TLC (table 5). A significant decrease ($P < 0.05$) was found for CV at 3 and 12 months in the Smokers and at 6 and 12 months in the Quitters. There was no significant change in VC at any test period for either group. For TLC, a significant decrease ($P < 0.05$) was found in both groups only at the 6-month test period. We attribute the significant decrease in CC/TLC in the Smokers at 6 months to TLC

because CV and VC did not change. On the other hand, it is difficult to determine the relative effect of TLC on CC/TLC at 6 months in the Quitters because both CV and TLC decreased significantly. The significant decreases in CV/VC at 3 months in the Smokers and 6 and 12 months in the Quitters, and in CC/TLC at 12 months in the Quitters are clearly due to the significant decreases in CV.

In addition to examining the effect of stopping smoking on lung function, we also examined the effect of modification of the smoking habit on the various tests. The mean changes from initial values for smoking consumption and for the various measurements at each test occasion for the Modifiers are shown in table 6. The only significant ($P < 0.05$) improvement seen in this group was in CC/TLC at 6 months. The apparent increase in smoking during the year in this group was largely due to the increase in the number of Intermittents smoking at each time period from 0 of 17 at one month to 8 of 17 at 3 months, 11 of 17 at 6 months, and 16 of 17 at 12 months.

TABLE 6
MEAN CHANGE FROM INITIAL VALUES FOR VARIOUS
MEASUREMENTS IN 23 MODIFIERS*

Time from Initial Testing (months)	Δ Cigar- rettes/ Day	FEV ₁ (Δ , liter)	MMEF ₂₅₋₇₅ (Δ , liter/sec)	CV/VC (Δ , %)	CC/TLC (Δ , %)	$\Delta N_2/L$ (Δ , %)
1	-30	0.03	0.18	-1.6	-1.3	-0.1
3	-24	-0.03	-0.10	-0.5	1.8	0.2
6	-22	0.04	-0.03	-2.0	-3.7†	-0.1
12	-16	0.06	0.02	0.1	0.1	-0.1

For definition of abbreviations, see table 2.

*Cigarette consumption reduced 25 per cent or more from initial test period.

†Significant ($P < 0.05$) by t test.

TABLE 7
 MEAN CHANGE FROM INITIAL VALUES IN CLOSING VOLUME AS A PERCENTAGE OF VITAL CAPACITY (CV/VC), CLOSING CAPACITY AS A PERCENTAGE OF TOTAL LUNG CAPACITY (CC/TLC), AND SLOPE OF THE ALVEOLAR PLATEAU (PHASE III) ($\Delta N_2/L$) FOR 7 SUBJECTS WHO STOPPED SMOKING FOR 6 MONTHS

Time from Initial Testing (months)	CV/VC (Δ , %)	CC/TLC (Δ , %)	$\Delta N_2/L$ (Δ , %)
1	-3.6	-3.6*	-0.3
3	-4.4	-3.1	0.1
6	-4.8*	-5.9*	-0.6
12	-2.8	-2.9*	-0.3

*Significant ($P < 0.05$) by t test.

It was difficult to analyze the data from the Intermittents, because at any one test occasion some were smoking and some were not. Seven of this group, however, were still not smoking at 6 months and can therefore be considered quitters up to this point. The mean changes in CV/VC, CC/TLC, and $\Delta N_2/L$ for this group are shown in table 7. For all 3 measurements, the mean change from initial values at each test period was, for the most part, greater for the 6-month quitters than for the 12-month quitters.

To examine the effect of stopping or modifying the smoking habit on respiratory symptoms such as cough, expectoration, shortness of breath, and wheezing, a symptom ratio was derived for each group. This was defined as the ratio of the number of symptoms observed within a group to the total number of possible symptoms within that group. For the purposes of this analysis, subjects were grouped as Quitters, Modifiers, and Nonmodifiers. The symptom ratio for each group at each test period is shown in figure 1. Initially, all 3 groups had very similar symptom ratios. The Quitters showed a dramatic decrease in symptoms within one month and sustained this improvement for the rest of the year. The Modifiers also showed a definite, although smaller, decrease in symptoms that was constant throughout the year. Those who did not decrease their cigarette consumption by at least 25 per cent (Nonmodifiers) showed very little change during the year.

Discussion

Our finding that CV, the slope of the alveolar plateau, and the number of respiratory symptoms decreased significantly in those who stopped smoking provides additional evidence that some of the early structural changes and

functional impairment resulting from cigarette smoking are reversible.

In interpreting these findings, an important consideration is the characteristics of the subjects. In our study, the Quitters and Smokers were similar in age, smoking history, respiratory symptoms, and the percentage of subjects with abnormal function at the beginning of the study. In terms of functional level, as measured by mean standard score (z), the Quitters were slightly closer to the nonsmokers' predicted values than were the Smokers. Basically, we were dealing with relatively healthy smokers; few of our subjects had clinically overt disease and/or abnormal spirometric tests. For the group as a whole, the prevalence of functional abnormali-

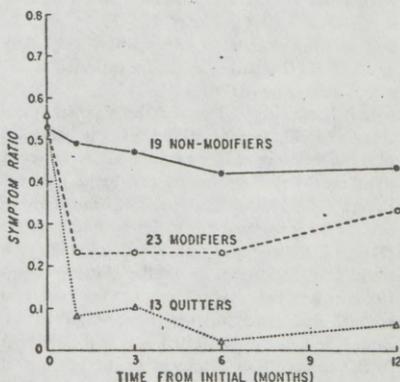


Fig. 1. Symptom ratio (number of observed symptoms to number of possible symptoms) in Nonmodifiers, Modifiers, and Quitters at each test period. The symptoms are cough, sputum production, wheezing, and shortness of breath.

ties was almost identical to that found recently in smokers in a random sample of 507 persons working in the city of Portland (15). We were, therefore, dealing with a group of smokers in whom the structural and functional changes caused by smoking might be expected to revert to normal with smoking cessation (4).

Structural changes that are found in smokers who are older or who have marked functional impairment may not be so likely to revert to normal when the subject no longer smokes. It has been widely thought that by the time a smoker develops an abnormal FEV_1 , the disease process is largely irreversible (4, 16). This belief may no longer be tenable, however, because Fletcher (17) has demonstrated that an abnormal FEV_1 may revert to normal after stopping smoking and that the accelerated decline in FEV_1 with age in smokers with chronic bronchitis usually changes with cessation of smoking to the more gradual decline found in nonsmokers.

In the absence of pre- and post-smoking cessation morphologic studies, it is impossible to know the nature of the structural changes that occur on cessation of smoking, but it seems reasonable to propose that in the airways there is a regression of the inflammatory changes, restoration of more normal epithelial structure, and a reduction in the amount of mucus and number of mucous plugs in the peripheral airways. Other possible changes may be a return to normal amounts of surface-active material, as demonstrated by Finley and Ladman (18), and to normal mucous composition (19). Any or all of these changes might be expected to result in a decrease in CV.

It is harder to understand the significant decrease found in the slope of the alveolar plateau, because this measurement is believed to reflect the elastic properties of the lung parenchyma (20). However, if smoking causes alveolitis, as demonstrated by Anderson and Foraker (21) and Niewoehner and associates (22), this could lead to alterations in the elastic properties of the alveoli, which might then revert to normal with smoking cessation. Another, although less likely, possibility is that the changes in the composition of mucus in the peripheral airways of smokers, described by Ebert and Terracio (19), may lead to an increase in the intralobar inhomogeneity, which would be reversible when the composition and amount of mucus returned to normal.

There are good theoretic reasons, therefore, for the changes found in both CV and the slope of the alveolar plateau. Other investigators have reported finding an improvement in MMEF with cessation of smoking (3, 6). We did not find this, possibly because only 2 of our Quitters initially had an abnormal MMEF. The fact that the MMEF did not improve with smoking cessation in our study could be interpreted as evidence that the MMEF is somewhat less sensitive to minor changes in peripheral airway function than CV or the slope of the alveolar plateau. However, the MMEF was able to distinguish between the Smokers and Quitters in that during the course of a year there was a significant deterioration in MMEF in the Smokers but no change in the Quitters. It is also interesting that the mean decrease in MMEF in the Smokers was approximately 6 times the decrease predicted by the regression equation of Morris and associates (13).

It seems reasonable to predict that if stopping smoking results in functional improvement, reducing the amount smoked may also result in improvement, although to a lesser degree; however, this was not found to be the case, with the exception of CC/TLC at 6 months, for the group of subjects who had reduced their smoking consumption by 25 per cent or greater.

One of the most interesting findings of this study was that respiratory symptoms almost completely disappeared in the Quitters and decreased in the Modifiers, but showed no appreciable change in those who did not decrease their smoking consumption. Presumably, this reflects a regression of the hypersecretion of the mucous glands in the central airways, which is the structural hallmark of mild chronic bronchitis (23). In this respect, therefore, respiratory symptoms could be considered the most sensitive indicator of structural changes that occur after stopping smoking.

The conclusions we draw from this study are (1) that stopping smoking causes definite improvement in both respiratory symptoms and function and that decreasing consumption by at least 25 per cent results in improvement in respiratory symptoms but no measurable improvement in function; (2) that the time course of change after smoking cessation may be quite slow with some improvement occurring within one month, followed by continuation of improvement up to 6 months after smoking is stopped.

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Mr. SEGAL. Are you aware of any studies that you have done on small airways relating to teenagers?

Dr. HOCKETT. We have such a study going on, I believe now, here in this city.

Mr. SEGAL. Who is doing it?

Dr. HOCKETT. Dr. Paul Hamosh, I believe is involved in this. And the plan of this is that it should go on for 3 years in order to obtain data that will really be useful. This is required to work out some methods of measurement that can be used in the field but are still accurate and reliable in the methodological problems. I couldn't tell you, any conclusion from this work, which has really only just begun. There are no conclusions, and I guess there wouldn't be any now until the end of about the 3-year period, which would be sometime in 1981.

Mr. SEGAL. Are you familiar with a study that was published in 1974 by Niewoehner of 39 cases, 20 nonsmokers and 19 smokers, to determine if a histiologic abnormality could be identified in peripheral airways of young smokers, and they found definite pathologic changes in the cigarette smokers.

Dr. HOCKETT. I don't remember the specific papers. There are a multitude of papers that contradict one another.

Mr. SEGAL. Do you have any contradictory findings that show this?

Dr. HOCKETT. I would have to see what kind of pathological changes these were.

Mr. SEGAL. These were pathologic changes in the small airways.

Dr. HOCKETT. I can't comment on that.

Mr. SEGAL. You did before state that you did not fund research into small airways until the last few years. Is that correct?

Dr. HOCKETT. No; I don't think I said that. We have had some such research going on for some time. We went into the emphysema problem quite a few years ago. We have a large project, but they are not these clinical things. They are concentrations on the biochemical angles, what goes on in there that produces this disease. That is what we need to know to be able to determine the effect of any environmental factor.

Mr. SEGAL. When you talk about an individual, evidence on an individual, which is what you keep coming back to, it is more than the biochemical effects. We are also talking about clinical observations, are we not?

Dr. HOCKETT. We have a certain amount of clinical research. But, as I mentioned originally, the great concentration was on the pathogenesis of these diseases, which is not well understood. We feel until it is well understood, we cannot answer such questions as this—what is the significance of a change of this kind.

Mr. SEGAL. Are you aware of any studies that have been done relative to any kind of clinical observations, not only on children, but adults on the smaller airways?

Dr. HOCKETT. There are a great many of them.

Mr. SEGAL. That you have funded? Do you believe a study methodology is correct with conclusions that are valid?

Dr. HOCKETT. No; I don't think we have a study that would be analogous to this kind of thing. Most of what we are doing is right down at the test tube biochemical level, studying these enzymes,

how they are put together, what the structures are, where they break, where the antielastases are located in the lung, and what their chemical nature is, and how they act. We are trying to get to the mechanics of these things.

Mr. SEGAL. Let's talk about it, an elastase breakdown; is that right?

Dr. HOCKETT. That is the idea, and the stretchy parts of the lung.

Mr. SEGAL. What happens if there is too much elastase?

Dr. HOCKETT. If there is too much elastase, at least dog model experiments show that the substance of these small, the very small air sacs, the wall will dissolve and the little ones will coalesce together, into larger ones, and then they lose their elasticity; they won't expand and contract.

The real problem in emphysema is in exhalation, because the lungs don't collapse back in the normal fashion. The lung capacity—actually, the capacity is increased, but the functional capacity, the amount of air that goes out and in with each breath becomes reduced.

That has to go quite a long way before any clinical symptoms are observed. It is irreversible. If the little air sacs, the walls, are destroyed, they don't rebuild.

There are some people that are just born with a lack of the antielastases that defend against such destruction of the air sacs or impede their normal function. They used to call it the alpha I antitrypsin, but this term has been abandoned. We talk about them as antiproteases now. There are a number of them. Originally we thought there was one; now we find there are many.

Mr. SEGAL. Would you suggest to those people who are known to be deficient in alpha I antitrypsin or the new ones you alluded to, would you suggest to them that smoking might be a danger?

Dr. HOCKETT. Yes, I would; it might be, but you see they are probably going to get emphysema whether they smoke or not.

Mr. SEGAL. But they might get it in their second or sixth decade of life—

Dr. HOCKETT. I don't know any clear picture of that.

Mr. SEGAL. Isn't it clear from studies those people who do have homozygous deficiency of alpha I antitrypsin, they are more likely to have emphysema than those that don't, if they smoke?

Dr. HOCKETT. It may be so.

Mr. SEGAL. Those were among the studies you helped fund?

Dr. HOCKETT. But there are not very many of these homozygotes. The problem has been with the heterozygotes; they are more suspicious.

Mr. SEGAL. There have been studies that demonstrate that. Do you believe those conclusions that—

Dr. HOCKETT. That is still controversial.

Mr. SEGAL. A lot of things are controversial. Do you believe it?

Dr. HOCKETT. I don't make judgments because I don't feel I am qualified to make judgments. I try to keep the arena open so the gladiators can have a good fair fight and work these things out.

Mr. SEGAL. Who makes the scientific recommendations to the council?

Dr. HOCKETT. This is the scientific advisory board. They discuss what kind of work might be relevant, what kind of work should be done.

Mr. SEGAL. Do you staff that group?

Dr. HOCKETT. We are the staff. That is, the staff of the council carry out the decisions or judgments of the advisory board. What happens usually is that people learn that we sponsor research. We get multitudes of applications and inquiries, whether we would be interested in research along one line or another. These inquiries are all submitted to committees of the board for consideration on their relevance and their scientific merit, and many times a little committee will go and visit somebody, discuss things, see their setup, what kind of team they have, and it may or may not be funded.

Naturally we get many more requests than we can fund. I would say less than 1 in 10 is funded, maybe 1 in 20.

Mr. SEGAL. Based on your scientific judgments, would you recommend to people who have a heterozygous deficiency of alpha I antitrypsin, that they smoke or not smoke?

Dr. HOCKETT. It is not my business to advise people.

Mr. SEGAL. We are asking you as the person sent by the Council for Tobacco Research, as a scientific researcher, to present some testimony to the Congress, and the question that is being posed is, would you recommend it? You don't have to answer it, but the question is, would you personally recommend it?

Dr. HOCKETT. I think what I would say, if he knew he was heterozygous, that he acquaint his physician with that fact, so it could be taken into consideration and follow the advice of his own physician.

Mr. SEGAL. Would you smoke if you were a heterozygote?

Dr. HOCKETT. I might be for all I know. I don't know. I don't know whether I am a heterozygote or not. I smoke, and so far haven't had any trouble.

Mr. SEGAL. Are you familiar with the report which finds cigarette smoke condensate suppresses the usual activity of antiproteases?

Dr. HOCKETT. Yes; I am familiar with that work.

Mr. SEGAL. That let's off excess of proteases in the lungs; right?

Dr. HOCKETT. That is right.

Mr. SEGAL. That is greater among smokers than nonsmokers?

Dr. HOCKETT. I don't think that is the question. As I understand, his suspicion is that various inhaled substances might affect the release of those proteases from the places where they are stored, so they get out from the leukocytes or monocytes.

Mr. SEGAL. So there is excess proteases, and we know what that does.

Dr. HOCKETT. Right.

Mr. SEGAL. It breaks down the elastic walls. What else does it do? What if it is collagen?

Dr. HOCKETT. I don't think it affects collagen very much. It apparently acts chiefly on elastic tissue.

Mr. SEGAL. Proteases only act on elastic tissue?

Dr. HOCKETT. The ones we are concerned with, the elastase acts chiefly on—

Mr. SEGAL. What about collagenase?

Dr. HOCKETT. Well, there are collagenase in the lung, but we don't think they are very much involved in emphysema. The attention seems to be focusing on elastasis.

Mr. SEGAL. Have there been studies that have shown that there is also an imbalance with excess collagenase versus inhibitory—

Dr. HOCKETT. I don't recall specifically whether that has been shown. But there are believed to be imbalances between the proteases and the antiproteases in the lung.

Mr. SEGAL. And more smoking leads to further imbalance; right?

Dr. HOCKETT. We don't know. If we get a method of measurement for checking this, we will find out. He used condensate, and that is not equivalent to fresh smoke. That work is not yet conclusive.

Mr. SEGAL. Condensate in vitro and in vivo, there have been studies on both; right?

Dr. HOCKETT. The condensate, itself, chemically and physically, is not equivalent to the kind of smoke people ordinarily inhale. They are very different.

Mr. SEGAL. Then when you get to the kind that people inhale, then you are talking about human clinical observations, and you said you don't deal with that.

Dr. HOCKETT. We can't do that, but we have to do experiments, preferably experimental observations in order to evaluate what the smoke will do.

Mr. SEGAL. Is that with an individual?

Dr. HOCKETT. We do it with mice.

Mr. SEGAL. Let's talk about people.

Dr. HOCKETT. We are not doing experiments of that kind on people at present.

Mr. SEGAL. But we talked about the Seely study, and that was done on people.

Dr. HOCKETT. Yes, but we didn't do that. That wasn't our study.

Mr. SEGAL. So any studies that are outside the bounds of the \$40 million-plus that you distributed on behalf of the Tobacco Institute is beyond the world of research for you to consider?

Dr. HOCKETT. No; we consider them, but we weren't involved in that. I thought you implied that we had some kind of involvement in it, and we are not at the moment conducting that type—well, we are conducting that type, but we are not conducting any experiments with humans at this moment to find out how smoke affects these proteases. There are dog experiments underway.

Mr. SEGAL. Do the dog experiments show results that are different than the humans?

Dr. HOCKETT. We don't know yet. This takes a long time.

Mr. SEGAL. Who is doing the dog studies?

Dr. HOCKETT. There is a dog study going on in Philadelphia at the Einstein Institute.

Mr. SEGAL. What about the work done by Harris on human alveolar macrophages. They came out concluding that cigarette smoke condensate could cause the release of elastase from leukocytes. Do you agree with those?

Dr. HOCKETT. I am not going to comment on one particular study. This is being studied by a lot of different people, and they are still more or less in controversy as to what happens. We know that the

elastases in macrophages, if you break them up with ultrasonic sound waves, will produce emphysema in a dog lung, but the question is whether they ever get loose in the case of humans in real-life situations. That is another question, which is pretty tough.

Mr. SEGAL. We can't just say that is one study. The way one accumulates evidence, be it a scientist or lawyer or anybody else, is to take specific findings based on a hypothesis. We all learn about Koch's postulates and certain kinds of things in isolation, and we move from there until we come to conclusions. You have not yet reached the point of conclusions that others have, but I don't believe, unless you choose to say so, that you can disregard pieces of evidence along the way.

Dr. HOCKETT. What I intended to say was I can't comment minutely on the specific paper you bring up because I can't remember the details offhand well enough to know what they are all about. But I have a general picture of the kind I described in the 1975 report on the status of this subject, now a little out of date, and we follow these things that are going on, but I can't comment individually on the merit of any one of them on short notice like this.

But we have a general picture, and we feel that these are some of the things that have to be solved, the kinds of things I mentioned, what influences can bring about the release of these enzymes from the macrophages and other structures, and there are a number of people working on this subject. We probably have 10 or 12 grantees that are involved with this, and this is a constantly changing picture. I couldn't tell right up to the minute what stage any one of them has reached, but they are moving along, I think, in a very productive fashion.

Mr. SEGAL. Let me ask it in a more general way. The earliest effects of cigarette smoking have now been identified as occurring not in the bronchi or the large bronchus, but the smaller peripheral airways of the tracheobronchial tree. Do you agree with that?

Dr. HOCKETT. I don't know whether I agree or not. I am aware of it, and I watch what goes on to find out whether it is true or not. I don't think you should ask me really whether I agree or not, because it isn't my business to agree or disagree, but to be aware of these things and watch, like watching a football game.

Mr. SEGAL. But as the person sent by the tobacco industry to be a scientific expert to tell Congress who has to decide what kinds of legislation ought to be enacted or not enacted, or regulated or not regulated, we are attempting to acquire what seems to be the conventional wisdom of science as it exists today, and the question to you, as the science expert sent by the industry, is, do you agree with that kind of statement?

Dr. HOCKETT. Well, I tried to explain what our role is. We try to make it possible for people to do independent research with the hope that will go into the stockpile and the truth will emerge.

Mr. MAGUIRE. If the gentleman will yield, I am reasonably confident that if the chairman of your Council were here, or if Mr. Yeaman or Mr. Kornegay had appeared on behalf of the Tobacco Institute and had been asked these questions, that they would have referred them to you in view of the fact that you are the scientific research director. Now, the questions are, in fact, being asked of

you, and you are here telling us that it is not your role to make any judgments.

I find——

Dr. HOCKETT. These things are not conclusive.

Mr. MAGUIRE [continuing]. I find that difficult to understand.

Dr. HOCKETT. That is the nature of science. Many things are not conclusive. They go gradually. All scientists should be very careful in making judgments as to what the significance of this or that is, because in 3 years they may find it isn't so.

Mr. MAGUIRE. So you never make a judgment on the basis of all the scientific——

Dr. HOCKETT. I am——

Mr. MAGUIRE. Let me finish asking a question, if you wouldn't mind. You never make a judgment on the basis of any scientific evidence you have helped to fund or any of the evidence that has been marshaled by other researchers. Is that correct?

Dr. HOCKETT. I might make limited and tentative judgments in order to lead to the next step, but I think I should be cautious about making judgments. Let them make the judgments.

Mr. MAGUIRE. Let who make the judgments?

Dr. HOCKETT. The people who are doing the experimental work, not individually, but the judgments have to emerge out of the interaction of people working various angles of a particular problem. There is no way out of that.

Mr. MAGUIRE. Fine, if you could then answer Mr. Segal's questions by giving us your limited and tentative judgments, rather than saying you have no judgments or you can't respond, that would be appreciated by the committee.

Dr. HOCKETT. I have been trying to do that.

Mr. MAGUIRE. Mr. Segal.

Mr. SEGAL. Thank you. Let me ask it in relation to an individual; you know Dr. Richard A. Bing; he serves on your board. Is that correct?

Dr. HOCKETT. Yes.

Mr. SEGAL. Are you aware he was also a member of the AMA's——

Dr. HOCKETT. Yes, I am.

Mr. SEGAL. Do you know he, after synthesizing the materials and others that were there, also was a researcher who carried on these kinds of studies that you alluded to, as you said, the researchers ought to come to the conclusions, and he concluded the point that Mr. Waxman read to you before, for those individuals with preexisting coronary artery disease, they had a grave risk by smoking, and that was his conclusion. Isn't that correct?

Dr. HOCKETT. I don't think it is altogether correct. I think he probably has said there are people who shouldn't smoke because it might hurt their cardiovascular systems. As I recall, he indicated he did not favor the inclusion of any conclusions in the book. I haven't checked that with him, but I believe that is the case.

Mr. SEGAL. We may get the answer to that specific one tomorrow, but he nevertheless did not have a scientific or other dissenting view comment on the report that came out.

Could you give us a tentative statement on the sentence I read you before about cigarette smoking having an effect on small airways. Do you believe it does?

Dr. HOCKETT. I would say this remains as a plausible possibility, but I think quite a lot still has to be done to determine the extent or the significance of this.

Mr. SEGAL. Do you think all of the existing mechanical and scientific equipment that has come forth that can now in some ways measure things that were not measurable before, do you think we do not yet have sophisticated enough scientific apparatus to measure?

Dr. HOCKETT. I think it has improved a lot, and there are some things that are possible that weren't possible before, but many of these have been developed in recent years, and some have been discarded. It is still a period of constant change.

Mr. SEGAL. Do you believe all the studies that have come out in one direction, using all of the equipment that has been used, are still not worthy to come to any kind of conclusion about a factual item that small airways disease and cigarette smoking are related?

Dr. HOCKETT. That is a pretty broad—that is the kind of broad conclusion I hesitate to draw. I think that should be drawn by a group of experts in the field, you see. I watch them; I listen to them.

Mr. SEGAL. Like the group that was here that drew that conclusion?

Dr. HOCKETT. That was not a large group of experts in that particular pulmonary field.

Mr. SEGAL. How large a group do you think ought to be assembled to come to this kind of conclusion?

Dr. HOCKETT. Well, I don't know. I think these people have to draw their own individual conclusions.

Mr. SEGAL. Individually or as a group?

Dr. HOCKETT. It is better individually, really. They can confer, and other people will probably dispute. You will have this constant seesaw and zigzag toward the truth. You could say at any one time the consensus seems to be so-and-so. Three years later, it may be different.

Mr. SEGAL. What does the consensus seem to say today?

Dr. HOCKETT. I don't know.

Mr. SEGAL. This is not a consensus; that was a small group?

Dr. HOCKETT. That is too small a group in a limited program over a few years, and they drew that conclusion.

Mr. SEGAL. Over 14 years, and \$15 million.

Dr. HOCKETT. Ten years or a little more.

Mr. SEGAL. Over 10 years.

Dr. HOCKETT. I am saying—all I am saying is that they didn't draw sweeping conclusions. I think they were right not to do that. They expressed their feeling about what they had there, and I am not going to dispute it. But I don't think that really settles all the numerous questions that are involved.

Mr. MAGUIRE. I can't help observing that the standard which you apply before you are willing to draw a conclusion or make a judgment is one which, if it were applied generally to other things that we decide individually or as a society to do or not to do, would,

I think, result in a situation in which we would never do or not do anything. It is most perplexing.

Mr. SEGAL.

Mr. SEGAL. I just want to continue one last line on some factual evidence that we would like to know whether you feel ought to be included in a body of evidence that you would agree to or not.

Do you believe when the normal balance between the two groups of proteases and antiproteases is upset that the excess proteases can lead to the destruction of the elastin and to the dissolution of the lung parenchyma? You believe that; right?

Dr. HOCKETT. I think that seems quite convincing, as of now. That is all I can say.

Mr. SEGAL. It is convincing to you?

Dr. HOCKETT. Yes, I think it is a good working hypothesis that has quite a lot of support.

Mr. SEGAL. Has this hypothesis not emerged in relation to a number of studies that have shown that the damaging effects of cigarette smoke on the macrophages and the white blood cells, the polymorphonuclear leukocytes that normally function, help create this imbalance?

Dr. HOCKETT. I don't think it has happened yet. It is under investigation; it is under suspicion.

Mr. SEGAL. The things that led to this suspicion have been those studies that have used cigarette condensate, have they not?

Dr. HOCKETT. That is right, and they have to be confirmed with whole smoke.

Mr. SEGAL. What we are trying to find out is what it is that you believe as evidence. You are saying that cigarette condensates have been shown to create this imbalance; that causes an excess of proteases that leads to the destruction of the elastic qualities within the lung?

Dr. HOCKETT. It is under suspicion, and I think it would be a fine thing if people try to find out if that is so; what is it that does it. It probably could be removed.

Mr. SEGAL. When you say that, do you mean like removing tar or nicotines?

Dr. HOCKETT. No. Tar is not smoke, you know. It is condensed smoke that has undergone chemical changes and a great deal of the original smoke is gone off as gases. I object to the term tar. But, anyway—

Mr. SEGAL. We would like to know what this tar is, anyway.

Dr. HOCKETT. It is just condensed smoke, caught in a cold trap.

Mr. SEGAL. But it is ingredients, right, polycyclic aromatic hydrocarbons?

Dr. HOCKETT. There are tiny traces of that. There are at least 10,000 different compounds present and nearly that many identified.

Mr. SEGAL. Is beta naphthylamine one that has been found?

Dr. HOCKETT. In small amounts.

Mr. SEGAL. Do you know what that does relative to urinary cancer?

Dr. HOCKETT. I know it is a urinary tract carcinogen.

Mr. SEGAL. Right, if you were interested in possibly smoking as an adult or just turning an adult and wanted to smoke, but seemed

to be impressed with the fact that Beta naphthylamine was a known human carcinogenic and some of the products in the market had it, and some didn't, wouldn't you like to know which ones had that ingredient?

Dr. HOCKETT. I think that information would be useless to people unless they had the quantitative concept. These days we have to know how much you are talking about.

Mr. SEGAL. So you would like to know whether it is zero, whether it is one microgram, whether one nanogram, or one milligram; is that what you mean by quantitative?

Dr. HOCKETT. Yes, we have to know that sort of thing in relation to physiological effects.

Mr. SEGAL. We have to know whether or not it is present before we know how much it is?

Dr. HOCKETT. That can be deceiving; that is going to raise all kinds of alarms. If you take anything apart, if you look at coffee the same way, by chromatography, it is full of all kinds of things we know are poisons, the same is true of flavorings, perfumes, spices, and so forth. They all contain such things; they are everywhere. And there is no reason to single out tobacco, and tobacco smoke, for headlining and spotlighting under this situation. It is only one of many environmental things, and we are faced with how to evaluate all of these in relation to health.

Mr. SEGAL. Do you know how much Beta naphthylamine is involved in cigarettes?

Dr. HOCKETT. Well, I don't remember offhand; I know it was years before anybody was able to find it, and the techniques had to become refined and refined. It is probably in terms of micrograms as benzopyrene is.

Mr. SEGAL. Does it have an acceptable threshold better—

Dr. HOCKETT. Threshold is a philosophical question. The mathematical question is very hard to settle. There is a great battle on now, as you very well know, between the mathematical people, that is, the statisticians, and physiologists. They are divided on this question, but we know there are levels of, what you might call carcinogens, if you like, that can't possibly be harmful. They are too low, and yet theoretically, mathematically, there is no such thing as a threshold.

Mr. MAGUIRE. Doctor, you invoke the notion of many hazards in the environment, many possible carcinogens, different dosage levels, and so on. Are you aware of the fact that the consensus of scientific evidence seems to be that among adult men some 40 to 50 percent of the cancers are linked to smoking?

Dr. HOCKETT. Linked. And that raises the question of statistical association, the same link between cause and association, and that is not any simple problem. And this has been pointed out very strongly by both Dr. Berkson, of the Mayo Clinic, and Dr. Wilson, whom I mentioned, as our advisor in the early days, and practically every one of the two dozen or so statistical men that I have had conferences with over the years. That is the basis of the fallacy that I am worrying about.

The other thing is that human smokers select themselves into the groups: smokers and nonsmokers. If we are going to do an animal experiment, we use litter mates and separate them at

random, so but we know any differences between those animals are due to the treatment they get. It isn't true in any of these studies.

Human beings classify themselves in smoking and nonsmoking, and we don't know what influences go into that determination. It is a horribly complex problem. And a pure statistician will tell you, these are not comparable groups. They should be——

Mr. MAGUIRE. Would you have recommended that we not take any steps to protect people against polio because we didn't fully understand the causal relationship?

Dr. HOCKETT. There are certain things they can do. I think that the obligation of the Public Health Service is to let people know the facts, but without scare tactics, without exaggeration, to simply acquaint them with the facts and let them make up their own minds how they want to act. Not campaigns, not scare tactics; that was Dr. Little's view.

Mr. MAGUIRE. Would you have recommended that we not take steps to protect people against polio on the basis that we didn't have the causal relationship fully and scientifically established?

Dr. HOCKETT. The only thing we could suggest was they avoid crowds when an epidemic was around, and that sort of thing.

Mr. MAGUIRE. We also developed a vaccine that was administered widely in the population in order to protect people.

Dr. HOCKETT. That was carefully checked out and found to be effective before it was applied to the population and, fortunately, it worked.

Mr. MAGUIRE. Yes, but we didn't have all the causes of polio in mind any more than we can demonstrate——

Dr. HOCKETT. Yes, we did. We couldn't make the vaccine until we isolated the virus responsible for polio and found out how to grow it in an egg or monkey kidney. That was central to the process.

Mr. MAGUIRE. Did we understand all the linkages? Did we understand what happened to which cell?

Dr. HOCKETT. Not all of them, but the important ones.

Mr. MAGUIRE. Do we understand how it developed in each individual one, whether there were individual differences and whether large enough groups of scientists had drawn the conclusions, and so on?

Dr. HOCKETT. That was done in the preliminary experiments. They had control, and so on, to determine its effectiveness. But the analogy between infectious diseases and the kind of constitutional diseases we are talking about is pretty wide, because all these constitutional diseases are really due to a kind of breakdown of some part of the body's defensive system over a long period of time of wear and tear and people differ enormously in their susceptibility.

Mr. MAGUIRE. Of course they do. I must remind you that we are finally only dealing with individuals, and not with some abstract notions; we are dealing with individuals who smoke or do not smoke. If people were to stop smoking, Doctor, do you think the rate of lung cancer would go down in this country, based on your best scientific estimates and your best tentative judgments?

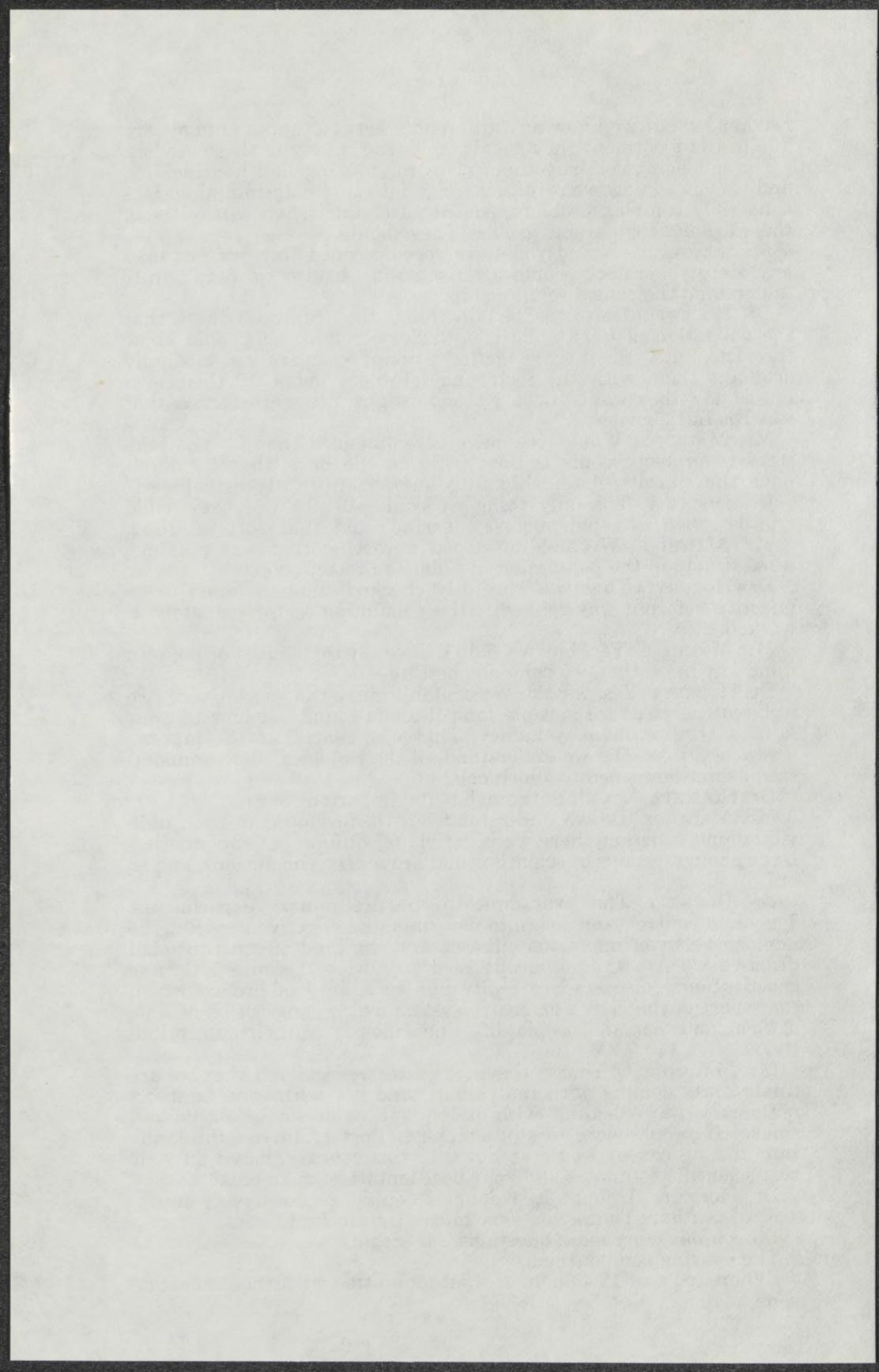
Dr. HOCKETT. I doubt it. I doubt it would go down very much.

Mr. MAGUIRE. Thank you very much, Dr. Hockett.

Do you have any more questions, Mr. Segal?

The hearing is adjourned.

[Whereupon, at 12:45 p.m., the subcommittee adjourned, to reconvene at 10 a.m. October 5, 1978.]



CIGARETTE SMOKING AND HEALTH—UPDATE

1978

FRIDAY, OCTOBER 6, 1978

HOUSE OF REPRESENTATIVES,
SUBCOMMITTEE ON OVERSIGHT AND INVESTIGATIONS,
COMMITTEE ON INTERSTATE AND FOREIGN COMMERCE,
Washington, D.C.

The subcommittee met, pursuant to notice, at 10 a.m. in room 2322, Rayburn House Office Building, Hon. Henry A. Waxman presiding. [Hon. John E. Moss, chairman.]

Mr. WAXMAN. The meeting will come to order.

I am pleased to convene this second day of the subcommittee's investigation into the relationship between smoking and disease. At yesterday's hearing we received testimony from a representative of the tobacco industry's research organization. The witness went out of his way to deny any linkage between smoking and increased risk of heart disease and cancer. Though such testimony was not unexpected, it was surprising in the face of almost two decades of scientific evidence to the contrary.

It had been the subcommittee's intention also to hear from a representative of the Tobacco Institute, the industry trade association. Mr. Horace Kornegay, president of the institute, however, declined to testify.

Due to this refusal we will not have the opportunity to ask industry spokesmen about the adequacy of their efforts to reduce the skyrocketing increases in the numbers of teenagers and preteenagers starting to smoke.

The trend in tobacco industry advertising seems to characterize cigarette smokers as vigorous, active, and virile young people. The act of smoking is portrayed almost as a badge of maturity and sophistication. I am personally anxious to discuss these advertising campaigns with the Tobacco Institute. I am interested in exploring what role these ads play in the decision of young people to start smoking. They may serve to mislead and possibly deceive children into thinking cigarettes are healthy. These are very serious issues and I am hopeful the subcommittee will have the opportunity to address them at a hearing in the near future.

Today we will hear from two scientists who will hopefully be more candid and forthcoming about the serious health risks associated with cigarette smoking.

Our first witness will be Dr. Gio Gori, Federal Officer of the National Cancer Institute's Division of Cancer Cause and Prevention. Dr. Gori is the author of a controversial study on the comparatively less hazardous nature of low tar and nicotine cigarettes

that appeared in the September issue of the Journal of the American Medical Association.

Our second witness will be Dr. Robert Hunter, chairman of the board of trustees of the American Medical Association. The AMA's Education and Research Foundation recently published what many consider to be a definitive report confirming the relationship between smoking and the development of chronic pulmonary disease.

Dr. Gori, would you come forward please.

Good morning. Let me swear you in.

Do you promise to tell the truth, the whole truth and nothing but the truth, so help you God?

Dr. GORI. I do.

Mr. WAXMAN. Please be seated.

TESTIMONY OF GIO B. GORI, PH. D., DEPUTY DIRECTOR, DIVISION OF CANCER CAUSE AND PREVENTION, NATIONAL CANCER INSTITUTE, NATIONAL INSTITUTES OF HEALTH, PUBLIC HEALTH SERVICE, DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Mr. WAXMAN. I understand you don't have a prepared statement.

Dr. GORI. No, I don't.

Mr. WAXMAN. Please proceed in any way you would like.

Dr. GORI. I would like to specifically be instructed by you, Mr. Chairman, of exactly what questions you would like me to answer at this point.

Mr. WAXMAN. Let me start off with staff asking questions of you.

Mr. SEGAL. Dr. Gori, I would like to identify for the record, if I might, a publication which came from your office. It is entitled "Smoking and Health, A Program to Reduce the Risk of Disease in Smokers," was it a status report dated December 1977?

Dr. GORI. Yes. This is the latest of a published series of reports on the smoking and health program of the National Cancer Institute.

Mr. SEGAL. Fine. I would like to just refer to a few items in that. On page small roman 6, in the beginning—Health Consequences of Smoking. You will note at the bottom of that a chart that was prepared.

Could you explain under the lists of diseases, bronchitis and emphysema, for example, 85 percent male, 85 percent female. Could you explain that chart and that specific notation?

Dr. GORI. If you read the paragraph before the chart, it says that these are estimates of the portion of these diseases that may be attributable to the use of cigarettes. It is self-explanatory, therefore.

Those percentages refer to each of the diseases listed, to what we think—from the epidemiologic evidence we have today the portion of these diseases is that is attributable or linked to the smoking of cigarettes.

Mr. SEGAL. So what you are saying is that you are attributing 85 percent of emphysema and bronchitis in this country to the results of cigarette smoking, is that right?

Dr. GORI. Yes, this is the implication of available epidemiologic studies.

Mr. SEGAL. If I could then refer you to page 6 of the text, there is a chart, table 1, and then at the bottom table 2, and if we look at table 2 and look at bronchitis and emphysema again, you describe the number of deaths that you would expect in this country from emphysema and bronchitis, is that correct?

Dr. GORI. That describes the percent of the present population living in the United States that would incur these diseases, if the trends present today—namely, if the death rates and the incidence rates present today—would continue indefinitely.

Mr. SEGAL. Based on those estimates, you would expect that 2,150,000 people will die of bronchitis and emphysema?

Dr. GORI. Yes.

Mr. SEGAL. Of those people living today?

Dr. GORI. That is correct.

Mr. SEGAL. And then on the right hand column it says premature deaths having tobacco-related causes. Does that lead to the understanding that 1,828,000 people would be expected to die prematurely of bronchitis and emphysema because of cigarette smoking?

Dr. GORI. That is correct. If the present trends and the present patterns of cigarette consumption would continue, one may expect—this is an estimate of course—that approximately that number would shorten their lives of some period of time because of the habit.

Mr. SEGAL. And that compares with two other larger items within that same table; namely, diseases of the heart and cancer of the lung where you have lower percentages but higher numbers.

Dr. GORI. That is correct.

Mr. SEGAL. Now, yesterday the question was posed to the Council on Tobacco Research that was asked whether or not cigarette smoking was the most important cause or an important cause in leading to chronic obstructive lung diseases.

Would you be prepared to say that you believe it is?

Dr. GORI. Based on the epidemiologic evidence that we have, and the consensus that I sense from the experts in this field, I would think that the major contributor to obstructive pulmonary disease today is smoking.

Mr. SEGAL. You would say the major contributor. Would you go as far as one of the Surgeon General's reports that said the most important factor? Would you say that?

Dr. GORI. Yes, I would say that.

Mr. SEGAL. If I could just ask one more question along those lines. When you are looking at emphysema and bronchitis, in your table, on page 6, you include three items that are smoke components—this is under table 1—that are contributory to what you believe causes bronchitis and emphysema, is that right?

Dr. GORI. I must point out that we can only say that smoking contributes to bronchitis and emphysema. Exactly what in smoke contributes to it is a question that still needs a precise answer today.

We feel that nitrogen oxides, hydrogen cyanide, acrolein, and a number of other components of smoke may be incriminated when we speak of emphysema and bronchitis. But this still needs to be proved beyond doubt.

Mr. SEGAL. But you believe that with current scientific knowledge, the state of the art being what it is today, that nitrogen oxides, acrolein, and hydrogen cyanide found in cigarette smoke are contributors to emphysema and bronchitis.

Dr. GORI. I would say it appears so. But, I would like to repeat that we need considerably more research to establish that precisely.

Mr. SEGAL. Do you know what kinds of levels of those three components and another component that is not normally made available to the public would be in cigarettes, for example, do you know how much carbon monoxide or how much hydrogen cyanide might be found in a given brand?

Dr. GORI. Brands may differ, depending on the construction of the cigarette, of course. But we are speaking on average in the order of approximately 10 to 20 milligrams of carbon monoxide, and of a few hundred micrograms of hydrogen cyanide per cigarette.

Mr. SEGAL. So you would say that carbon monoxide is found in much greater concentrations.

Dr. GORI. That is correct.

Mr. SEGAL. In some cases is it found in greater concentrations than nicotine?

Dr. GORI. Yes. Usually we find 10 to 20 milligrams of carbon monoxide in a cigarette, and nicotine usually is present in much lower quantities; namely, 1 to 2 milligrams per cigarette, or less.

Mr. SEGAL. So, there is a much higher order of magnitude of carbon monoxide in cigarettes.

Dr. GORI. Certainly there is much more carbon monoxide than nicotine.

Mr. SEGAL. OK. In the paper that you published recently in the Journal of the AMA—Mr. Chairman, I would like to request that this article be placed in the record.

Mr. WAXMAN. Without objection, that will be done.

[The paper referred to follows:]

Toward Less Hazardous Cigarettes

Current Advances

Gjo B. Gori, PhD, Cornelius J. Lynch, PhD

• Critical levels of selected cigarette smoke constituents have been expressed in terms of maximum numbers of pre-1950 cigarettes that a smoker may consume daily without increasing his mortality risk substantially above that of a nonsmoker. This could still imply an important risk, although it may be difficult to detect. We relate these levels to the yields of 27 current low tar and nicotine commercial cigarettes, as measured at the Oak Ridge National Laboratory. In addition, the yields of these selected constituents concomitant with the yield of 1 mg of nicotine are provided as a guide for the smoker who titrates or adjusts his smoking pattern to accommodate a fixed daily intake of nicotine.

(*JAMA* 240:1255-1259, 1978)

SINCE the Surgeon General's report on smoking and health,¹ considerable attention has been focused on cigarette smoke constituents implicated in the cause of tobacco-related diseases. Many such components have been considered, the most frequently

*For editorial comment
see p 1271.*

cited being total particulate matter (tar), nicotine, carbon monoxide (CO), nitrogen oxides (NO_x), hydrogen cyanide (HCN), and acrolein. Several investigations document the contribu-

From the National Cancer Institute, Bethesda, Md (Dr Gori), and Enviro Control, Inc, Rockville, Md (Dr Lynch).

The views expressed herein are those of the authors and do not necessarily reflect the views or policies of the National Cancer Institute, Public Health Service.

Reprint requests to Division of Cancer Cause and Prevention, National Cancer Institute, Biog 21, Room 11A03, Bethesda, MD 20014 (Dr Gori).

tion of these components to cancer, chronic pulmonary disease, or cardiovascular impairment.²⁻¹¹ Many studies also indicate that there is a dose response between the number of cigarettes smoked and disease incidence and morbidity.^{12,13,16} Since publication of the Surgeon General's report, average tar values of commercial cigarettes have decreased by 29%, and nicotine yields have decreased by 21%, indicating a continuing preoccupation toward reduced hazard.¹⁴

Evaluation of health benefits resulting from these reductions would be premature because of the long latent periods involved. Available data suggest that, for the present, smoking-related diseases have not abated substantially, with the possible exception of cardiovascular diseases. However, factors contributing to the decline in these latter diseases are not yet clear. On the other hand,

mortality from tracheal, bronchial, and lung cancer has continued to rise; projections for the immediate future indicate that further increases may be expected. This probably is because smokers now in the age groups in which cancer is most likely to develop have spent a considerable part of their smoking history using high tar and nicotine cigarettes. As younger smokers who are exposed to lower tar and nicotine cigarettes approach cancer-susceptible ages, a reduction in morbidity and mortality rates could be expected. However, considering tar and nicotine alone may give an incomplete and misleading picture of hazard reduction: the full impact of low tar and nicotine cigarettes on health effects should be evaluated in

Table 1.—Average Critical Levels of Pre-1950 Cigarette Consumption^a

Disease	No. of Pre-1950 Cigarettes
Cancer of the oral cavity	8.2
Pharyngeal cancer	2.5
Esophageal cancer	7.3
Pancreatic cancer	9.0
Laryngeal cancer	6.8
Lung cancer	5.7
Bladder and kidney cancer	9.5
Coronary artery disease	4.2
Coronary heart disease	3.5
Aortic aneurysm	4.5
Emphysema, bronchitis, or both	10.0
All causes for current smokers	2.0

terms of all the major toxic smoke components mentioned previously.

Critical levels of daily smoke inhalation have been discussed recently in terms of the maximum number of pre-1960 cigarettes that may be smoked daily without detectable increase to the average smoker's risk of mortality beyond that of a nonsmoker.¹¹ These are by no means safe levels but merely imply that, for a smoker whose daily consumption does not exceed these levels, any attendant tobacco-related mortality risk may be epidemiologically indiscernible from that of a nonsmoker.

This could still imply a substantial although less readily apparent risk. For instance, if the smoker's risk of developing lung cancer could be reduced from the present level of approximately 10:1 to some value less than 2:1, this risk, while considerable, could be difficult to establish epidemiologically. The inability to verify this reduced risk might lead to its being considered socially tolerable.

Average critical levels relative to diseases to which smokers are particularly susceptible are listed in Table 1, expressed as daily numbers of pre-1960 cigarettes. The last entry in Table 1, "All causes for current smokers," is a comprehensive category representing the effect of cigarette consumption on mortality in general. These values are based on typical yields per cigarette of pre-1960 cigarettes: 43 mg of tar, 3.0 mg of nicotine, 23 mg of CO, 270 µg of NO_x, 410 µg of HCN, and 130 µg of acrolein.¹²

CURRENT COMMERCIAL CIGARETTES

Most commercial brands today have yields that are below the typical pre-1960 levels, with particular interest in lowered tar and nicotine yields. A recent publication from the Oak Ridge National Laboratory¹³ lists the yields of the six constituents referred to previously for 32 brands of commercial low tar and nicotine cigarettes. Twenty-seven of these brands have measured tar yields that do not exceed 10.0 mg by more than two SEs. Results of testing for these 27 brands are summarized in Table 2. The lowest measured tar yield is 1.2 mg, and the highest is 10.3 mg (SE, 0.40 mg).

Table 3 presents the percentage

Table 2.—Analytical Data for Selected Low Tar and Nicotine Cigarettes¹³*

Brand	Tar, mg/cigt [†]	Nicotine, mg/cigt	Carbon Monoxide, mg/cigt	Nitrogen Oxides,‡ µg/cigt	Hydrogen Cyanide, µg/cigt	Acrolein, µg/cigt
Benson & Hedges Lights	10.1	0.81	12.1	135	116	81
Carton	1.5	0.15	2.6	34	18	15
Carton Menthol	1.2	0.14	2.0	12	12	10
Decade	5.5	0.46	4.3	57	49	38
Decade Menthol	6.6	0.69	4.4	61	50	47
Iceberg 100's	3.1	0.32	5.7	44	44	42
Kent Golden Lights	8.9	0.71	9.2	61	51	47
Kent Golden Lights Menthol	8.3	0.66	8.3	71	62	37
King Samo	5.8	0.29	11.5	196	79	35
King Samo Menthol	5.3	0.25	13.6	205	102	44
L&M Flavor Lights (King)	7.2	0.80	4.8	40	65	30
L&M Long Lights (100's)	6.5	0.67	5.5	41	69	47
Lark II	7.5	0.61	7.3	83	84	44
Lucky 100	3.1	0.28	5.3	68	34	28
Merit	8.5	0.60	12.1	168	151	49
Merit Menthol	8.4	0.61	10.2	172	140	52
Newport Lights Menthol	10.3	0.85	12.5	86	133	67
Now	1.9	0.19	2.4	25	16	15
Now Menthol	1.6	0.16	2.1	30	9	13
Pal Mall Extra Mild	5.1	0.47	5.8	78	65	38
Real	10.2	1.01	12.9	99	155	76
Real Menthol	7.9	0.81	10.2	84	105	44
Stride	3.3	0.36	1.8	5	<10	12
Tareyton Lights	7.8	0.72	2.6	85	75	31
Tempo	6.9	0.56	10.1	168	98	31
True	4.8	0.48	5.2	72	34	29
True Menthol	5.2	0.42	5.7	64	43	31

*Data analyzed by Oak Ridge National Laboratory.

†Abbreviation cig indicates cigarette.

‡Total oxides of nitrogen.

Table 3.—Reduction in Yields as Percent of Pre-1960 Cigarette Yields

Brand	Tar, mg/cigt [†]	Nicotine, mg/cigt	Carbon, Monoxide, mg/cigt	Nitrogen Oxides, µg/cigt	Cyanide, µg/cigt	Acrolein, µg/cigt
Benson & Hedges Lights	77	73	47	50	72	53
Carton	97	95	89	67	96	88
Carton Menthol	97	95	91	96	97	92
Decade	87	85	61	79	88	71
Decade Menthol	85	77	61	77	86	64
Iceberg 100's	93	89	75	84	89	68
Kent Golden Lights	79	76	60	77	88	64
Kent Golden Lights Menthol	81	78	64	74	85	72
King Samo	87	90	50	27	61	73
King Samo Menthol	88	92	41	24	75	66
L&M Flavor Lights (King)	83	73	79	85	84	77
L&M Long Lights (100's)	85	78	76	65	83	64
Lark II	83	80	68	69	80	66
Lucky 100	93	91	77	75	92	78
Merit	80	80	47	38	63	62
Merit Menthol	80	80	56	36	66	60
Newport Lights Menthol	76	72	46	66	68	56
Now	96	94	90	91	96	86
Now Menthol	96	95	91	89	96	90
Pal Mall Extra Mild	88	84	75	72	84	71
Real	76	66	44	63	62	42
Real Menthol	82	73	56	69	74	66
Stride	92	88	92	98	>96	91
Tareyton Lights	82	76	69	69	82	76
Tempo	84	81	56	39	76	78
True	89	85	77	73	92	76
True Menthol	88	86	75	76	90	76
Average	86	83	69	69	83	71

*Abbreviation cig indicates cigarette.

Table 4.—Critical Levels of Selected Smoke Constituents

Brand	No. of Cigarettes Required						Lowest Row Entry	Highest Row Entry
	Tar	Nicotine	Carbon Monoxide	Nitrogen Oxides	Hydrogen Cyanide	Acrolein		
Benson & Hedges Lights	9*	7	4†	4†	7	4†	4	9
Carlton	57*	40	18	16†	51	17	16	57
Carlton Menthol	72*	43	23†	45	55	26	23	72
Decade	16	13	11	9	17*	7†	7	17
Decade Menthol	13	9	10	9	16*	6†	6	16
Iceberg 100's	28*	19	8	12	19	6†	6	28
Kent Golden Lights	10	8	5†	9	16*	6	5	16
Kent Golden Lights Menthol	10	9	6†	8	13*	7	8	13
King Samo	15	21*	4	3†	10	7	3	21
King Samo Menthol	16	24*	3†	3†	8	6	3	24
L&M Flavor Lights (King)	12	8†	10	14*	13	9	8	14
L&M Long Lights (100's)	13*	9	8	13*	12	6†	6	13
Lark II	11*	10	6†	7	10	6†	6	11
Lucky 100	26*	21	9	8†	24	9	8	26
Ment	10*	10*	4	3†	5	5	3	10
Ment Menthol	10*	10*	5	3†	6	5	3	10
Newport Lights Menthol	8*	7	4†	6	6	5	4	8
Now	46	32	19	22	51*	17†	17	51
Now Menthol	48	36	22	18†	51*	20	18	51
Pall Mall Extra Mild	17*	13	8	7†	13	7†	7	17
Real	8*	6	4	5	5	3†	3	8
Real Menthol	11*	7	5†	6	8	6	5	11
Stride	26	17†	26	106*	>82	22	17	106
Tareyton Lights	11	8	18*	6†	11	8	8	18
Tempo	12*	11	5	3†	8	8	3	12
True	18	13	9	8†	24*	9	8	24
True Menthol	17	14	8†	8†	19*	6†	8	19

*Highest row entry.

†Lowest row entry.

Table 5.—Yields of Selected Constituents Concomitant With Yield of 1 mg Nicotine*

Brand	No. of Cigarettes Necessary to Yield 1 mg Nicotine	Tar, mg	Nicotine, mg	Carbon Monoxide, mg	Nitrogen Oxides, μ g	Hydrogen Cyanide, μ g	Acrolein, μ g
Benson & Hedges Lights	1.2	12.1	1.0	14.5	162	139	73
Carlton	6.7	10.1	1.0	17.4	228	107	101
Carlton Menthol	7.1	8.5	1.0	14.2	85	85	71
Decade	2.2	12.1	1.0	9.5	125	108	84
Decade Menthol	1.4	9.2	1.0	6.2	85	70	66
Iceberg 100's	3.1	9.6	1.0	17.7	136	136	130
Kent Golden Lights	1.4	12.5	1.0	12.9	85	71	66
Kent Golden Lights Menthol	1.5	12.5	1.0	12.5	107	93	56
King Samo	3.4	19.7	1.0	36.4	666†	269	119
King Samo Menthol	4.0	21.2	1.0	54.4†	820†	408	176
L&M Flavor Lights (King)	1.3	9.4	1.0	6.2	52	85	39
L&M Long Lights (100's)	1.5	9.8	1.0	8.3	62	104	71
Lark II	1.6	12.0	1.0	11.7	133	134	70
Lucky 100	3.6	11.2	1.0	9.1	245	122	101
Ment	1.7	15.0	1.0	20.6	286	257	83
Ment Menthol	1.6	13.4	1.0	16.3	275	224	83
Newport Lights Menthol	1.2	12.4	1.0	15.0	103	180	68
Now	6.3	10.1	1.0	12.7	133	85	80
Now Menthol	6.3	11.3	1.0	13.2	189	57	82
Pall Mall Extra Mild	2.1	10.7	1.0	12.2	160	137	80
Real	1.0	10.2	1.0	12.9	99	165	76
Real Menthol	1.2	9.5	1.0	12.2	101	126	63
Stride	2.8	9.2	1.0	5.0	14	<28	34
Tareyton Lights	1.4	10.9	1.0	3.6	119	105	43
Tempo	1.8	12.4	1.0	18.2	299	176	66
True	2.2	10.6	1.0	11.4	168	75	64
True Menthol	2.4	12.5	1.0	13.7	154	103	74
Critical Value	Not Applicable	86.0	6.0	46.0	540	820	260

*Each column also gives constituent/nicotine ratio. For example, entries under column headed "Tar" give tar/nicotine ratios.

†Exceeds critical value.

reductions in yields of these brands compared with yields of typical pre-1960 cigarettes. Reductions range from a high of more than 98% (Stride HCN yield) to a low of 24% (King Sano Menthol NO₂ yield). On the average, the brands under consideration have had the greatest percentage reduction in tar yield (86%) and the least percentage reduction in CO and NO₂ yields (69%) compared with pre-1960 cigarettes.

The numbers of these cigarettes smoked daily without exceeding critical levels have been calculated from the data in Tables 1 and 2, as in the following example: since the critical level for all causes is two pre-1960 cigarettes, each yielding 43 mg of tar, the number of Benson & Hedges Lights (10.1 mg of tar each) with the equivalent tar yield is 8.5. Thus, 8.5 Benson & Hedges Lights have a total tar yield equal to the critical level for the "All causes for current smokers" category.

Similar values for all of the brands and constituents considered in this article are given in Table 4, rounded off to the nearest integer. Critical levels were calculated under the assumption of a smoking pattern uniformly distributed over a ten-hour period for any given day. Deviations from such a smoking pattern could alter some critical values, such as those associated with CO effects.¹²

The lowest entry in each row of Table 4 represents the maximum number of cigarettes of that brand that if smoked daily would not exceed the critical level for any of the smoke constituents considered. The highest row entry represents the maximum number of cigarettes of that brand that if smoked daily would not exceed at least one of the smoke constituents considered. The range from highest to lowest row entries provides the smoker with intermediate goals for gradually reducing his smoking habit through progressively less hazardous smoking stages: proceeding in this manner, he would gradually reach the lowest row entry as a daily maximum cigarette consumption level. At this point, the smoker is likely to be more receptive to taking the final step toward total cessation.¹³ Similar considerations apply to brands not explicitly addressed in this article. If the majority of smokers proceeded

along these lines, it would be reasonable to predict a substantial decrease in tobacco-related morbidity and mortality.

It should be noted from Table 4 that the highest row entries for 14 brands are for tar yields, whereas the lowest row entries for 13 of the brands are for NO₂ yields. In addition, lowest row entries for nine brands occur for CO and acrolein. These values suggest that the cigarette manufacturers should concentrate on the further reduction of NO₂ yields, while still attempting to reduce further the yields of other constituents, particularly CO and acrolein.

With the introduction of relatively low nicotine yields, it has been suggested that some smokers may compensate by increasing the total number of cigarettes smoked to maintain a fixed daily level of nicotine intake.¹⁴ The daily intake of other constituents for such a person would depend on the nicotine compensation rate. Table 5 lists the yields of selected smoke constituents concomitant with the yield of 1 mg of nicotine for the brands under consideration. For example, for Benson & Hedges Lights, about 1.2 cigarettes yield 1 mg of nicotine. This same number of cigarettes yields 12.1 mg of tar, 14.5 mg of CO, 162 µg of NO₂, 139 µg of HCN, and 73 µg of acrolein. Thus, a smoker compensating to 1 mg of nicotine would be exposed also to these yields of other smoke constituents.

Compensating to other nicotine values would affect associated yields proportionately. Table 5 allows a smoker to estimate his smoke constituent intake, depending on his own nicotine compensation rates. The 1 mg of nicotine yield (one sixth or 17% of the critical value) is accompanied by an NO₂ yield that exceeds the critical level for two brands and by a CO yield that exceeds it for one of these brands. For the remaining brands, the nicotine intake can exceed 1 mg before concomitant yields of other constituents exceed critical values.

COMMENT

Pre-1960 cigarettes have contributed most to the current epidemic of tobacco-related diseases, and epidemiologic studies show a relationship between number of cigarettes smoked daily and the risk of the development

of disease. From these studies, we can define the critical daily smoke intake that would not appreciably increase the risk of the smoker over that of the nonsmoker.

Because different cigarette brands deliver different amounts of smoke of different compositions, this critical smoke intake can be met by smoking different numbers of cigarettes, depending on brand.

Today, cigarettes having toxic constituent yields considerably below pre-1960 cigarettes are feasible, and forerunners of such cigarettes are commercially available. Twenty-seven brands that fall into this category were tested at the Oak Ridge National Laboratory, and the numbers of these cigarettes smoked daily without exceeding critical values have been estimated for six major toxic smoke constituents. These critical values may serve as intermediate goals for a smoker who is intent on reducing his smoking habit through progressively less hazardous smoking stages. These calculations are based on the assumption that the smoker of the low tar and nicotine cigarettes will not change his smoking habits in terms of depth of inhalation, frequency of puffing, and butt length. Findings of recent studies support this assumption.¹⁵

Although the yields for the 27 brands are considerably below the yields of the typical pre-1960 cigarettes, additional reductions are warranted, particularly with respect to NO₂, CO, and acrolein. Otherwise, smokers who compensate for fixed levels of nicotine intake, even though these levels do not exceed critical values for nicotine, may be subjecting themselves to daily intakes of other toxic smoke constituents in excess of their estimated critical values.

Methods for further reductions in yields of toxic smoke constituents have been developed through research such as that conducted by the National Cancer Institute's Smoking and Health Program.¹⁶ One of the principal objectives of this program is to identify those characteristics of cigarettes that lead to toxic and other adverse effects and to develop methods for reducing or eliminating such factors. Progress has been made thus far in improving methods for reducing tar yields through the use of

reconstituted tobacco sheet and in reducing nicotine yields through tobacco extraction processes and reblending. Other smoke yields have been adjusted through selected combinations of filters and smoke-dilution devices, the use of high-porosity paper, the use of tobacco blends rich in nitrates, and the adjustment of the cigarette's burning rate. Further incorporation of these and other state-of-the-art advances coupled with flavor acceptability characteristics can

improve commercially available cigarettes to the point where they may properly be termed less hazardous.

The rationale for developing less hazardous cigarettes rests on the fact that despite the publicity given to the health risks associated with smoking, more than 50 million Americans still smoke. While programs to discourage smoking should continue, these educational efforts should be coupled with others directed toward reducing the risks to persistent smokers.

Persuading the smoker to wean himself to progressively less hazardous cigarettes may provide an alternative to smoking cessation that is perhaps more effective than the self-denial approaches of current anti-smoking messages. Although this would not eliminate the risks to the smoker, it is an approach that has the potential to reduce the current epidemic of smoking-associated diseases to a considerably less serious public health problem.

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Mr. SEGAL. You have on table 2 analytical data analyzed by the Oak Ridge National Laboratory.

Dr. GORI. That is correct.

Mr. SEGAL. And if we could just go through that. Could you explain some of the relative differences between nicotine and carbon monoxide? In some cases it is higher, is that right? Benson & Hedges, the first one, 0.8 for nicotine and 12.1 for carbon monoxide.

Dr. GORI. Yes, carbon monoxide usually is higher than nicotine.

Mr. SEGAL. So that would be the general trend?

Dr. GORI. The general trend, yes.

Mr. SEGAL. As you know, right now there are advertising pieces and information put out by the industry where they identify the amount of tar and the amount of nicotine, but do not identify the amount of carbon monoxide.

Do you think that amounts of carbon monoxide should be made available to the public?

Dr. GORI. I believe they should. There is always the possibility of building cigarettes that may be low in tar, and low in nicotine, but still high in carbon monoxide. This apparently may have in fact happened in other countries.

I am not aware that it has happened in the United States, but simply to prevent this particular danger, I think that knowledge of carbon monoxide delivery from cigarettes would be desirable.

Mr. SEGAL. OK. So your conclusion is that it is possible, under current situations, to have lower tar and nicotine, but not necessarily lower carbon monoxide?

Dr. GORI. That is right.

Mr. SEGAL. And that might present an unknown danger to the American public?

Dr. GORI. That is correct.

Mr. SEGAL. And to compensate for that you would feel that the carbon monoxide level should be made public?

Dr. GORI. Yes, so that the consumer may have a reasonable choice.

Mr. SEGAL. Would you say that same thing should hold for cyanide?

Dr. GORI. I believe that the methods for reducing hydrogen cyanide go hand in hand with the methods for reducing tar and nicotine and carbon monoxide. If we had lower tar, nicotine, and carbon monoxide, they would be fairly well representative of the reduction of other components as well.

So, I don't think it would be necessary to indicate the hydrogen cyanide content of cigarettes.

Mr. SEGAL. OK. I have one further line of questions, Dr. Gori.

Could you explain what the tobacco working group was within your organization?

Dr. GORI. The Cancer Institute about 10 years ago realized that in spite of all public education efforts many Americans were going to continue to smoke. Indeed today, after over 15 years of public education, we still have between 50 million and 60 million Americans who smoke.

The feeling was that something should be done to reduce the risk of all these people who smoked in spite of warnings.

One of the approaches was to be the development of less hazardous cigarettes. At that time the technology for producing and manipulating the engineering of cigarettes resided, and still resides today, with the cigarette industry.

It is a very specific art not widely known or widely practiced. It was felt at that time that it would be desirable to have the assistance of the technicians from the cigarette industry for the modification of certain characteristics of cigarettes, and the production of these cigarettes, so that the Cancer Institute program would be able to test them.

Therefore, the decision was made at that time to utilize the research directors of some of the cigarette industries, as advisers, technical advisers, to the tobacco working group in the design and production of the experimental cigarettes that the program tested.

Mr. SEGAL. They were formal members of a duly constituted advisory committee.

Dr. GORI. That is correct.

Mr. SEGAL. Of the Department of HEW?

Dr. GORI. Yes.

Mr. SEGAL. And they participated in the meetings and voted as voting members?

Dr. GORI. Yes, but they were never in a majority position. The committee had always between 12 and 15 members, and we always had no more than three or four people from the tobacco industry.

Mr. SEGAL. Mr. Chairman, could I request that the list of members of that committee be inserted into the record at this point?

Mr. WAXMAN. Without objection, that will be done.

Mr. SEGAL. Thank you.

[The material requested, had not been supplied at the time of printing.]

Mr. SEGAL. Now, they knew that the purpose of this was to have a less hazardous cigarette.

Dr. GORI. Correct.

Mr. SEGAL. The current kinds of cigarettes that were on the market maybe, if lowered in tar and nicotine, might lead to less hazard than the current ones. They participated with that kind of thing in mind.

Did they submit the actual kinds of cigarette uses for the studies? Did they take protocols that you developed and supply the cigarettes themselves?

Dr. GORI. I would like to clarify that they personally participated in the tobacco working group as private citizens, not as legal representatives of their industry. There was some legal arrangement at that time to this effect.

At the beginning I remember some discussions about safety and hazards of cigarettes which proved to be of some embarrassment to the members from the tobacco industry.

Mr. SEGAL. Could you elaborate on that? What kind of embarrassment?

Dr. GORI. Well, the question of whether cigarettes are dangerous or not came up in the first discussions. Of course, this was a genuine issue with the tobacco industry at that time.

The result was that we tried to stick with technical issues from there on because that was the problem that we were facing. We were facing the necessity to modify and test modifying cigarettes, so that they could be made them less hazardous.

Therefore, most of the discussion at the tobacco working group referred specifically to methods for producing different cigarettes and how to test them.

The cigarettes were then produced by different tobacco companies on the specifications prescribed by the tobacco working group.

Mr. WAXMAN. Did you ever have scientists from the tobacco industry, who worked with you, say they didn't want to get into a discussion of the hazards from cigarettes because they did not want to acknowledge this linkage in any way out of a fear of legal liability for the tobacco industry?

Dr. GORI. No, I don't think we have had that particular problem. But you can tell in a discussion of a group, say, whether a particular issue is a welcome one or not.

Mr. WAXMAN. A particular issue is what?

Dr. GORI. Is a welcome one or not. It was clear that if we wanted to do something about the engineering of cigarettes, and changing of the cigarettes, it would be best that we stick to that particular issue.

Mr. WAXMAN. Now, you are saying some issues, particularly the linkage issue of whether cigarette smoking causes health hazards, was an unwelcome one to some of the scientists from the industry.

These are scientists. They were at least, in the thought of most of us, acting based on the rigid discipline of the scientific method as

opposed to acting as public relations representatives from the industry.

Did this interfere in their scientific judgment? Did you find that they would be responsive to the industry's fears as opposed to that rigid discipline that we expect of scientists?

Dr. GORI. You must keep in mind that the tobacco working group and the smoking and health program were not set up to prove that tobacco smoking was dangerous, or anything of the sort.

Our purpose was to manufacture cigarettes that would be less hazardous, to produce them, to test them. In a sense, therefore, the questions of the hazards of tobacco in the population, epidemiologic studies and all that, were not really germane to what we were trying to do within the tobacco working group.

We had the particular task of reducing the hazards of cigarettes. We tried to stick to that particular task. In that context, they were functioning at a scientific level.

Mr. LUKEN. Would the gentleman yield?

Mr. WAXMAN. Yes.

Mr. LUKEN. Thank you, Mr. Chairman.

I would just like to ask you one question. That is in reference to the statement of the American Medical Association.

Now, they support the contention that the development of obstructive diseases—heart disease, lung disease—are caused by cigarette smoking.

What would be your comments on that?

Dr. GORI. I just supported that particular contention myself 5 minutes ago, Mr. Luken. Frankly, I am even surprised that today, in 1978, this question could be posed again.

I think in scientific circles today it is well understood, well accepted, that a large portion of chronic pulmonary obstructive diseases is due to the use of cigarettes.

Mr. LUKEN. May I just ask one little additional question, Mr. Chairman. How about cigar smoking? It is not discussed very much, very often, in this context. Yet, I hear it discussed in public quite a lot.

Some people say 1 cigar is as bad as 10 or 15 cigarettes. I have a personal interest in this question.

Dr. GORI. From your smile, it looks like that to me.

Mr. LUKEN. I look like a cigar smoker, don't I?

Dr. GORI. Apparently the scientific evidence—

Mr. LUKEN. I will hide it.

Dr. GORI. Don't hide the scientific evidence, hide the cigar. The scientific evidence indicates that cigars have, probably have some effect in producing certain cancers in the upper respiratory tract, in the mouth, in the pharynx, in the tongue.

But, they do not seem to be associated with cardiovascular diseases or with respiratory diseases or lung cancer, mostly because cigar smokers do not inhale.

Mr. LUKEN. If they do, of course—

Dr. GORI. If they do—

Mr. LUKEN. But cigarettes are worse?

Dr. GORI. That is correct. But most cigar smokers fortunately do not inhale. Therefore, the statistics are not as dramatic as we have from cigarette smoking.

Mr. LUKEN. Thank you, Mr. Chairman.

Thank you, Doctor.

Mr. WAXMAN. Dr. Gori, when we talk about cigarette smoking, you acknowledge what I think most Americans, including smokers, believe—that cigarette smoking is hazardous. So, people who continue to smoke do it with the understanding that they are taking a risk.

What they hope to do at times is minimize that risk. We have millions of Americans who feel that they cannot stop smoking. You have been working on the development of what could be called a less hazardous cigarette.

How much safer is this less hazardous cigarette that we hear is now being produced and sold on the market?

Dr. GORI. You used two phrases. One is less hazardous cigarette and the other is how much safer is it.

I would again state that I don't think we can use the word safe in connection with a cigarette. There is no such thing as a safe cigarette. We can speak of decreased hazard, but there is always going to be a residual hazard associated with any cigarette that one smokes.

The paper that was published in the Journal of the American Medical Association indicates that certain cigarettes today represent a 90-percent decrease in tar and nicotine over the average cigarette of 10 years ago.

This is a dramatic reduction, one that leads us to believe, based on the epidemiologic evidence of the last 20 years, that if all smokers were going to smoke a moderate number of these cigarettes then their risk would fall dramatically.

In other terms, the current epidemics of smoking-related diseases could reasonably be expected to perhaps disappear.

Now, this again is based not on experimental studies in animals but on very large long-term epidemiologic studies in man, conducted during the last 20 years.

Mr. WAXMAN. Earlier you discussed other chemicals, carbon monoxide and nitrogen oxides, that are still present in these less hazardous cigarettes.

Are the dangers of heart and lung disease from carbon monoxide, cyanide, and nitrogen oxides as great in these less hazardous cigarettes.

Dr. GORI. Of course not, because these components are reduced in these cigarettes. Apparently the industry has been able to reduce not only tar and nicotine, but also carbon monoxide and a variety of other components, including nitrogen oxides, hydrogen cyanide, and acrolein.

Mr. WAXMAN. When we talk about less hazardous cigarettes, could one who smokes assume that the main benefit is going to be a delay in the arrival of cigarette-induced ailments, or can one anticipate that some of these ailments may never come about at all?

Dr. GORI. It all depends on the type of cigarette that is smoked and the number of cigarettes that are smoked during 1 day.

In the paper that I have published in JAMA with Dr. Lynch, we indicate what the maximum limits of smoking compatible with a risk that may not be detectable.

In other terms, the limits that we have listed would still have a residual risk of developing disease, but epidemiologically this risk probably could not be detected.

This, of course, doesn't mean that an individual does not have some residual risk or that special classes of individuals at high risk, like asbestos workers, or uranium miners or coal miners, may not have a greater risk than the general population.

But in general the limits that we have indicated would be, or would seem to be, compatible with a level of risk that epidemiologically would not be distinguishable from the risk of the nonsmoker.

Mr. WAXMAN. Have you looked into or conducted any studies on physiologic or psychological reasons that people continue to smoke?

Dr. GORI. We have done some studies, although our prime concern, in the beginning at least, was to try to develop and evaluate methods for the production of less hazardous cigarettes.

Some behavioral studies are being conducted right now. It is important to see, for instance, whether people that may switch to low tar, low nicotine cigarettes smoke more cigarettes.

If this happened on a large scale and on a continuing basis, it would negate some of the beneficial effects of smoking less hazardous cigarettes.

Mr. WAXMAN. Do you know whether people who smoke low tar and nicotine cigarettes are smoking more cigarettes?

Dr. GORI. The evidence we have today indicates that if the switch is made gradually—namely, so that the next cigarette is no lower than 10 percent in tar and nicotine than the previous one—there is apparently no compensation.

If the switch is very large, say from a heavy cigarette yielding around 15 milligrams of tar to a cigarette around 2 or 3 milligrams, then likely there may be compensation.

This suggests that smokers who are seriously intent at getting rid of the habit, or at least decreasing the risk, should do so gradually.

The Federal Trade Commission publishes tables of tar and nicotine contents of all current brands in the United States so that any consumer concerned about his own safety and health would be able to have a guide to select cigarettes that are progressively lower in tar and nicotine, and therefore cigarettes that would not increase his risk of compensating.

Mr. WAXMAN. If a heavy smoker of many years' duration decided to switch to a safer cigarette—

Dr. GORI. Less hazardous.

Mr. WAXMAN [continuing]. Less hazardous cigarette—excuse me—and if that individual switched overnight to a less hazardous cigarette, is it likely that this individual may well compensate by smoking more of those cigarettes because of the habit that has already been formed and is so clearly part of his present pattern of behavior?

Dr. GORI. As I said before, if the new cigarette that he smokes is much lower, say 50 percent lower than the previous one, it is likely that he may compensate to some extent. How much it is not possible to say.

We are conducting studies right now. Probably the degree of compensation will have great individual variability.

All we know is that for a reduction in the order of 10 percent to 20 percent, this compensation is not likely to occur.

Mr. WAXMAN. I have heard some smokers say, upon switching, that the low tar and nicotine cigarettes are not as satisfying. For whatever reasons that they smoke, they do not get the same pleasure out of smoking low tar and nicotine cigarettes.

Do you know of people who have returned to the other cigarettes after having tried the low tar and nicotine cigarettes?

Dr. GORI. This depends again on the same issues that we have indicated for the compensation. It depends on the difference in tar and nicotine emission of the new cigarette.

If the cigarette is much lower in tar and nicotine, then the flavor, of course, is going to be much different and this may induce the smoker to go back to his previous cigarette.

However, again, if the switch is made gradually, the flavor perception does not change so dramatically and the smoker may eventually be able to wean himself out of the heavy cigarettes into something that is less hazardous.

Therefore, what we suggest here is that people should go at it gradually, should not try to make very drastic switches. This may be a dangerous proposition, may discourage them, and there is nothing worse for a smoker who wants to quit than getting discouraged.

The important thing I feel is to build a degree of confidence in the smoker that he can actually do it. But he will be able—he would be much more able to succeed, able to do it, if he is taking it easy, and is not going to try to go overboard very quickly.

Mr. WAXMAN. You obviously believe that the only safe cigarette is the cigarette that is unlit.

Dr. GORI. Yes, I have said that before.

Mr. WAXMAN. And if a smoker, knowing the dangers, realizes that is the safest route to take and wanted to give up smoking, he should switch gradually to a low tar, low nicotine cigarette would this be a reasonable way to stop by phasing out as opposed to trying to end smoking by a tremendous assertion of self-discipline and immediately ceasing all cigarette smoking?

Dr. GORI. The question needs to be proven yet experimentally. But today we believe that people smoke because of the flavor they get from the tar, and of the pharmacological action that they get from nicotine.

In other terms, people may be hooked to the cigarette because they are hooked to nicotine and the tar.

Conceivably, therefore, if they wean themselves to cigarettes that have very little tar and very little nicotine, their habituation to the cigarette may be lower and it might be easier for them to make the final switch to nonsmoking.

Mr. WAXMAN. Yesterday we heard testimony from Dr. Hockett, Director of Research for the Council for Tobacco Research. He acknowledged that there is a special problem for children who smoke cigarettes, although he did not acknowledge any health problems for adults.

He said his only concern over young children who smoke is that it might hinder their muscle development. But he implied that additional protein in the diet might alleviate this problem.

Do you agree with this statement? Is it a scientific statement? Have you looked at or conducted any studies on that subject?

Dr. GORI. I don't know that I can make scientific statements about this issue because I don't know of scientific studies that have been done about it.

These are suppositions I believe that can be reasonably made, but the verification of this, to my knowledge, is not available.

Mr. WAXMAN. I suppose there is research being conducted by behaviorists who try to determine why people are attracted to smoking and what role advertising may play, and whether advertising directed into a certain market, or for a certain age group, may well be effective in reaching that age group.

I assume that is not your particular area of expertise?

Dr. GORI. Within the National Cancer Institute program, we also conduct these kinds of studies. We have been able to start them only in the last couple of years, and I would think that it may take another 2 or 3 years before some reasonable results can be achieved.

The problem here arises from the apparent variety of reasons for which people smoke. In other words, not all smokers smoke for the same reasons. They don't have the same motivation, they don't have the same genetic or the same physiologic background.

So that to try to reach generalizations from research is somewhat difficult, and it will take more time than we had hoped it would take at the beginning.

Mr. WAXMAN. Is it safe to say—excuse me again for using the word safe—is it fair to say that the reason people continue to smoke is not only psychological but physiological, that there is some kind of dependence upon the tar and the nicotine in their system?

Dr. GORI. Again, precise research in this area is lacking, Mr. Chairman. But there is a belief among scientists in this field that nicotine dependency may play a substantial role in the need for continuing smoking in a large portion of smokers.

Mr. WAXMAN. Do you believe that children run a special or increased health risk from smoking?

Dr. GORI. We don't have data about this, but I would say simply on the basis of the greater vulnerability of children to toxic materials and insults, that children should be particularly shielded from that habit, keeping in mind that if they start that early, their chances of developing smoking-related disease when adults, are going to be enhanced.

Mr. WAXMAN. Do you know whether it is harder for people who have started smoking as teenagers to give up the habit later on?

Dr. GORI. I don't know that there is firm evidence to this extent, Mr. Chairman. I don't know of studies that have established this particular issue.

Mr. WAXMAN. What dangers or risks are posed to nonsmokers from smokers? Is there evidence to suggest that there may be an increase in the risk of heart and lung disease to a nonsmoker by being in the presence of smokers?

Dr. GORI. This is a difficult question, Mr. Chairman, because the answer that I have to give as a scientist may not please always my antismoking colleagues.

The fact remains that we really do not have conclusive scientific evidence about the adverse health effects of passive smoking on the bystander.

Of course, we may have some peculiar situations. You may have a small car with windows up, and four people smoking a cigar in there, which may create rapidly very toxic conditions.

But in the usual situations under which smoking is practiced, the evidence does not indicate that the casual bystander is seriously harmed by smoking.

Now, this doesn't mean that smoking does not cause annoyance, irritation, and all that, and simply on the basis of irritation and of his desire to breathe clean air, a nonsmoker may have a point.

Mr. WAXMAN. Have there been studies on the health consequences or risks to nonsmokers?

Dr. GORI. Yes.

Mr. WAXMAN. And they don't establish any danger at this point?

Dr. GORI. They may establish that there is some transfer of smoke components in the bloodstream of the bystander, but at levels that certainly do not appear sufficient to cause any specific harm at this point.

Mr. WAXMAN. In your opinion, as a scientist, do women who use contraceptive pills run an increased risk of heart disease if they smoke?

Dr. GORI. I believe so, yes.

Mr. WAXMAN. In a presentation before another subcommittee, the Tobacco Institute testified that the FDA's antismoking warning on oral contraceptives wasn't valid.

Are you aware of any studies published on the subject which do not show an increased incidence of risk among women who smoke and use the pill?

Dr. GORI. No, but simply on the physiologic and epidemiologic evidence that we have, I believe it is reasonable to think that women that take contraceptives may have a higher risk of developing cardiovascular problems if they smoke.

Mr. WAXMAN. What other individuals might run a higher risk from smoking cigarettes than the rest of the population?

Dr. GORI. Asbestos workers, uranium workers, coal miners, people that work in cotton mills and textile mills, generally people that work in atmospheres with high dust concentrations.

Mr. WAXMAN. Are some people genetically predisposed to a higher risk if they smoke?

Dr. GORI. Some have suggested this particular hypothesis. But I don't know of any scientific tests or scientific studies that have been done on this subject yet.

Mr. WAXMAN. The tobacco researcher yesterday told us that some people are predisposed genetically to a higher risk and until we could ascertain who these individuals are he didn't see that there was conclusive evidence that smoking was harmful to all people.

The conclusion he drew from that was we ought to let everybody smoke. Do you know whether the tobacco researchers are doing any tests in trying to ascertain which individuals may be predisposed to higher risk of lung disease?

Dr. GORI. I am not aware of these studies, but I cannot say they are not being conducted.

Mr. WAXMAN. Do you think it would be a prudent area of scientific research to try to discern whether there are some individuals who run very little risk by smoking so that those who know that they have a higher risk will be advised that they are taking a greater risk in smoking cigarettes?

Dr. GORI. I believe that it should be one of the priorities in our research, but not the highest at this point. The introduction of less hazardous cigarettes would have a much greater effect in reducing tobacco-related diseases than any other approach that we may think of today.

However, it is possible that genetic differences could be at the base of some of the differences in susceptibility that certain smokers have. Indeed, the National Cancer Institute has sponsored some research in this area, with rather discouraging results so far.

But as the methodology to screen for genetic variability improves, we may indeed come close to verifying this hypothesis.

Mr. WAXMAN. The National Cancer Institute is looking at a whole range of questions. People are often deciding nowadays, when they hear about dangers in various substances, whether to take a risk or not.

Some people throw up their hands and say, well, I guess we are always taking a risk even by crossing the street, so why not smoke cigarettes, why not take other food items that may well have carcinogens in them.

There is no doubt in your mind that cigarette smoking does involve carcinogenic components; is that correct?

Dr. GORI. If we look at the epidemiologic record, I don't think we can come to any other conclusion.

Mr. WAXMAN. But what I guess is most on people's minds is the anecdotal evidence that they themselves see. There is always someone who can tell you about a friend or neighbor or relative who smoked cigarettes for 50 years and died at a ripe old age and the story of an individual who never smoked but died of lung cancer at the age of 50.

Obviously we are not dealing with poisons that have certain effects on people the way taking cyanide would have, regardless of their genetic predispositions.

Dr. GORI. Correct.

Mr. WAXMAN. How do we explain this? Why is it that some people are going to be selected and others are not, even though they might expose themselves to the same amount of toxicity?

Dr. GORI. We have, of course, a variety of interesting hypotheses about this. Certainly this should be one of the most interesting avenues of research in our times.

Genetics, of course, as you have indicated before, could be a factor here, although we cannot pinpoint how and why genetics may operate.

Different behavioral characteristics may be a factor. A person that smokes two or four packs a day may actually inhale less tar than a person that efficiently smokes one or two packs a day because of different patterns in smoking.

The studies that have been done so far unfortunately are not very precise in determining actual inhalation of tar and nicotine by any individual smoker. We can have some idea of what happens to

the general population. But when we go to individuals, our knowledge about intake is minimal at this point.

Then you may have other factors of resistance or susceptibility determined by the environment or by nutrition, which seems to be increasingly one of the major factors in determining resistance or susceptibility of an individual.

You could have other illnesses which may predispose one individual rather than another to develop a particular disease subsequently in his life.

Then you have, of course, competing diseases, people that die of lung cancer may not die of heart disease and vice versa. We don't live long enough to develop all diseases that we could develop if we were allowed to do so.

Mr. WAXMAN. Do you believe the warning labels on cigarette packages or advertising are effective in discouraging people from smoking?

Dr. GORI. I would like to believe so, but if we look at the number of smokers in this country, one tends to conclude that the warning does not motivate well enough those 50 or 60 million Americans out there who are still smoking.

Mr. WAXMAN. Do you think of the 50 to 60 million Americans who continue to smoke, that they are not aware of the fact that there is some danger to their health in smoking?

Dr. GORI. I don't know that there is any smoker today in this country, or perhaps in the world, who is not aware of the dangers associated with cigarette smoking.

Mr. WAXMAN. So people reject obviously that warning and those of us in government who are concerned about public health wonder what, if anything, we should be doing. Since this is a personal decision, obviously ultimately it is up to the individual involved.

Should we be acting in some more positive way? We already provide warning labels and television advertising of cigarettes is banned. Yet, people continue to smoke.

There has been a decrease of smoking in adults. Is that your understanding?

Dr. GORI. Yes, there has been a decrease, largely, I must say, because of the action of the cigarette industry, which has responded by introducing on the market cigarettes with low tar and low nicotine.

Mr. WAXMAN. I mean, a decrease in people smoking at all. Do you think that is related to low tar and low nicotine cigarettes?

Dr. GORI. I don't know if there is a decrease of people smoking at all. The percent of people smoking in the United States has remained more or less constant for the last 10 years, as far as I know, so perhaps the absolute number of smokers may have actually increased somewhat.

The proportions in that percent may have changed, with a greater number of younger people, young women in particular. But the overall percentage has remained pretty constant in the last 10 years or so.

Mr. WAXMAN. Do you think that we ought to look into legislation to either require a listing of the carbon monoxide content of cigarettes or mandating that cigarettes be manufactured only with a certain level of low tar and nicotine?

Dr. GORI. Well, there have been, as you know, several attempts at proposing legislation of this nature. I think that any action by the Government that would result in lowering the intake of smoke by the smokers would be a desirable one.

Exactly what action I am not prepared to say because I am not an expert in legislation, any action that would tend to decrease the amount of smoke that the smokers inhale, would obviously have a beneficial impact, not necessarily immediately, but with a certain delay, on the public health.

This delay feature is something that we should keep in mind because it may indeed discourage some of us, or some of you in Congress that may wish to present this kind of legislation, because the results will not be felt immediately.

They will be seen and felt with a delay of 5 to 10 to 25 years, depending on the disease that we are talking about. But any measure in this direction would inevitably have a positive effect on the health of the Nation.

Mr. WAXMAN. Do you believe that if we prohibited smoking of cigarettes, similar to our prohibition on alcoholic beverages in the 1920's, that people would still continue to smoke?

Dr. GORI. Yes, sir.

Mr. WAXMAN. Do you feel that if we prohibited the manufacture and sale of cigarettes with high tar and high nicotine that people would turn to a black market to get cigarettes with high tar and high nicotine?

Dr. GORI. At the cost of sounding un-American, I must say that I am a gradualist. In this country, it is my opinion that we are looking for drastic solutions and drastic decisions, black and white.

I believe that with the habit of cigarette smoking, we must adopt a gradualistic approach. Any drastic solution to this problem I don't think is going to lead us to a desirable conclusion.

It is my opinion that we have the methods now to gradually reduce the hazards associated with smoking, perhaps to levels—as I have indicated in my article—that may even become to be considered socially tolerable.

I know that the word "tolerable" has raised a number of objections. But again, if we could not distinguish epidemiologically in the health effects attributable to smoking, then perhaps we could stop thinking of smoking as a problem.

Mr. WAXMAN. In some areas, for example the Clean Air Act, we mandate by legislation technological advancements. We tell the automobile industry, that by a certain period of time in the future, we expect them to meet certain standards for auto emissions that we consider will help reduce in the dirty areas of the country the amount of pollution in the air, obviously with health consequences in mind.

What would you think of the idea of setting standards for the tobacco industry to reduce certain chemical properties in cigarettes we know to be harmful by a certain period of time, and by mandating that they meet these levels. We would in effect also mandate that they devote resources to greater technology and research into accomplishing that result?

Dr. GORI. Scientifically, that is a reasonable approach and in keeping with my philosophy of a gradual approach, as long as we

can make sure that the changes are not going to be so drastic that the smoker will compensate or will turn to a black market. We need to educate the smoker to accept as normal cigarettes that are much milder than what he smokes today. This is possible.

The cigarette of 10 years ago was almost twice as strong as we have today. The average contemporary cigarette would have been considered as effeminate 15 years ago, a sissy cigarette.

Yet today, the average smoker can still ride his Marlboro horse and feel very strong about it. So, I can think that the smoker can be persuaded gradually to accept and consider acceptable cigarettes that are much milder than what we have today.

Mr. WAXMAN. How do you suppose that the notion of macho in cigarette smoking has come about? Years ago it was considered socially unacceptable for a woman to be seen smoking in public and also unacceptable for a man to smoke a cigarette as opposed to a cigar. Now we see those virile, happy people smoking cigarettes and riding on their horses.

Is that due to advertising or is it due to social pressures that come about through ways other than inducement by advertising?

Dr. GORI. Perhaps males in this country, with all the pressures that our life style puts on males, may have a greater need for cigarettes than other members of our population.

Mr. WAXMAN. But the increase we see in cigarette smoking is from females.

Dr. GORI. Perhaps because we have saturated the number of males that need to smoke, perhaps. But these are really presumptive discussions, Mr. Chairman.

Probably part of the image comes from the role that the First and the Second World War have had in the diffusion of cigarettes in our country and the imagery that Hollywood has been able to associate with cigarettes in our population.

There is a certain mythology associated with smoking today that follows a particular cliché.

Mr. WAXMAN. Do we break down that mythology by counteradvertising, let's say, on television, showing how harmful it is to the individual and to certain basic human values of life to continue smoking cigarettes?

Dr. GORI. I have mixed feelings about that, Mr. Chairman, because if I look back at the effectiveness of the educational campaigns, the scare tactics, television advertising, and other things that we have seen, I must conclude that if they had some effectiveness, it had to be marginal because so many people still smoke.

My impression is that people don't like to be scared, don't like to be insulted because they smoke. I think they need help. I think they may need some reassurance that they are still in command of their own habits, and—to the risk of getting boring here—my previous proposition of a gradualistic approach may offer the average smoker a way out.

Most smokers that I know would like to quit smoking, don't particularly feel proud about their habit, but somehow they need it. They cannot get rid of it. It is obvious that this is the situation.

Rather than a scare message, we could offer a message that gives them hope, a chance of winning it by themselves, without heroic efforts.

I would think that even if we had to take a few years to reduce the number of smokers in this country by a gradual approach, it would still be a great public health victory.

Mr. WAXMAN. We see now, I think, an amazing phenomenon, to me anyway, of sort of a polarization of smokers and nonsmokers who I think are going beyond the bonds of trying to protect their own comfort by trying to make those who smoke feel guilty.

As I drove into work the other day, I passed a car that had a license plate saying, in fact the letters were "I Smoke," and the bumper sticker on the car said "Califano May be Hazardous to Your Health." Obviously this is a radicalization, a strong reflection of antagonism between smokers and nonsmokers that they have some issue to resolve—over and above the question of how you deal with people who have a habit that they decide they don't want to break or cannot break.

Have you seen that?

Dr. GORI. Yes, of course, I have seen it. I have experienced personally this particular problem.

Mr. WAXMAN. I believe you have been somewhat of a victim, haven't you? I was amazed at the reaction to your report by some people who are very much concerned about smoking, who felt that it was a disservice to the cause because you suggested that maybe some people could smoke a less hazardous cigarette as opposed to going cold turkey.

Dr. GORI. I don't consider myself a victim, Mr. Chairman. I think that I am fortunate to be able to work in society and with this particular important problem, so I have to take the smiles and the bad news.

Mr. WAXMAN. I know, we have to also. We have to face election.

Dr. GORI. Personally, I like to cherish the hope of being able to do something for the public health of this country, and I believe that smoking is one of the few issues in prevention where we can make a contribution if we go at it patiently, not with radical solutions, because I don't believe that radical solutions have worked so far, or that they are going to work in the future. I think that we could approach our goals much faster if we adopted a gradualistic approach.

Mr. WAXMAN. In our society, if I can move to another subject that is somewhat related, there is a tremendous controversy over marihuana smoking, and the approach has been by our society to place criminal sanctions on those who smoke marihuana with the reaction by smokers of marihuana that they should have the right to make those decisions themselves.

As a consequence of this ongoing political dispute, many jurisdictions are either decriminalizing, making no criminal penalty or minimizing criminal penalty to the point of being no realistic sanction at all.

Do you foresee as a consequence of this, more people turning to marihuana and that maybe we are going to end up with a new kind of health problem, because we just don't know all of the health effects of marihuana? We didn't know the adverse health effects of cigarettes when they started catching on in this country?

Dr. GORI. You are asking me to look at my crystal ball rather than to express a scientific judgment on this issue. Marihuana is

not, in my opinion, going to be such a public health problem as tobacco is, because from what I know of marihuana and of tobacco, it is unlikely that people would smoke three or four packs of marihuana cigarettes a day. The weed just does not lend itself to that kind of consumption. So the doses of tar and carbon monoxide and other components taken in with marihuana smoking are likely going to be lower than the doses from smoking cigarettes.

Mr. WAXMAN. One contention that smokers make and even our witness yesterday from the Tobacco Research Council made, was that smoking cigarettes relaxes people. It relieves anxieties and tensions.

Is there any scientific evidence for that conclusion?

Dr. GORI. Yes, there is, and this is obviously a problem that we are conveniently forgetting when we speak of eliminating smoking. But it is obvious that a great number of smokers out there would like to quit, but too many cannot quit, and are somehow dependent for their daily lives, for their daily behavior, on the consumption of cigarettes.

Mr. WAXMAN. We are also told that there are some beneficial results from peristalsis with cigarette smoking.

Is there scientific evidence on that?

Dr. GORI. That I don't know. Many people smoke a cigarette after meals, and obviously, nicotine will have some effect on the autonomic nervous system.

Mr. WAXMAN. You have been most helpful. I want to thank you for appearing before us and answering these questions.

Dr. GORI. Thank you, Mr. Chairman.

Mr. WAXMAN. It sheds a great deal of light on a subject that sometimes is obscured by a lot of emotions. Thank you very much.

Dr. GORI. Thank you.

Mr. WAXMAN. Our next witness will be Dr. Robert B. Hunter, chairman of the board of trustees of the American Medical Association.

Dr. Hunter, do you promise to tell the truth, the whole truth, and nothing but the truth so help you God?

Dr. HUNTER. I do.

Mr. WAXMAN. Dr. Hunter, you have two gentlemen accompanying you. Do you have plans for them to testify?

Dr. HUNTER. They might.

Mr. WAXMAN. Let me administer an oath to both of them. Please identify yourself for the record.

Dr. SINGER. Ira Singer.

Mr. FENNINGER. Randolph Fenninger.

Mr. WAXMAN. Do both of you promise to tell the truth, the whole truth, and nothing but the truth, so help you God?

Dr. SINGER. I do.

Mr. FENNINGER. I do.

TESTIMONY OF ROBERT B. HUNTER, M.D., CHAIRMAN, BOARD OF TRUSTEES, AMERICAN MEDICAL ASSOCIATION, ACCOMPANIED BY IRA SINGER, PH. D., ASSISTANT DIRECTOR, DEPARTMENT OF UNDERGRADUATE EVALUATION; AND RANDOLPH FENNINGER, LEGISLATIVE DEPARTMENT

Dr. HUNTER. Mr. Chairman, I am Robert B. Hunter. I am a practicing physician from Sedro Wolley in the State of Washington, and I am chairman of the board of trustees of the American Medical Association. I also serve on the board of trustees of the AMA-Education and Research Foundation (AMA-ERF). With me today are Ira Singer, Ph. D., assistant director of AMA's Department of Undergraduate Evaluation and formerly secretary to the Committee for Research on Tobacco and Health, and Randolph Fenninger, a member of AMA's Legislative Department.

The American Medical Association is pleased to have this opportunity to respond to your interest in the recent publication "Tobacco and Health," the result of 14 years of research supported by the Project for Research on Tobacco and Health and sponsored by the American Medical Association—Education and Research Foundation.

THE AMA-ERF RESEARCH PROGRAM

In 1963, some 15 years ago, the AMA House of Delegates adopted a recommendation from its board of trustees that AMA-ERF be requested to undertake a comprehensive program of research on tobacco and health. This study was to be "devoted to the study of human ailments that may be caused or aggravated by smoking, the particular element or elements that may be the causal or aggravating agents, and the mechanisms of their action."

It was further recommended that this activity be financed not only by a contribution from the AMA, but that contributions also be solicited from other sources, and that only unrestricted contributions be accepted.

AMA-ERF is a nonprofit organization engaged primarily in the support of medical education through financial assistance to schools and students, and the fostering of scientific and medical research.

In 1964, with the AMA-ERF announcement of the formation of the Project for Research on Tobacco and Health, the tobacco industry declared its intention to support the project to the extent of \$10 million over a 5-year period. This money was freely given without limitations on objectivity. These funds came from individual cigarette companies: American Brands, Inc., Brown and Williamson Tobacco Corp., Liggett & Meyers, Inc., Lorillard Corp., Philip Morris, Inc., and R. J. Reynolds Industries, Inc. The amounts to be donated by each company were determined by the companies themselves and the funds were provided to the AMA Education and Research Foundation through yearly payments of approximately \$2 million. Four years later the companies, with the exception of American Brands, pledged additional funds to the Project for Research on Tobacco and Health.

By 1972, when this arrangement ended, the tobacco industry's total contribution to this effort had exceeded \$15 million.

Also in 1964, the AMA-ERF board of trustees appointed the Committee for Research on Tobacco and Health to determine how available funds might be used most effectively. This group proposed that requests for funds be initiated by individual investigators at recognized institutions and that the committee advise the AMA-ERF board of trustees on the merits of the proposed research project. The committee was active in recruiting scientists to participate in this research, and a postdoctoral fellowship program was instituted to encourage the interest of young scientists. As early as June 1964 the committee had recommended approval of the first awarded grants.

Three of the original committee were members of the Surgeon General's committee which developed the 1963 Surgeon General's report on the health hazards of smoking. The AMA-ERF committee usually met four times a year to consider applications for new grants, and to review the progress of research already funded.

Grants were awarded for periods ranging from 1 to 5 years and funds were transmitted to sponsoring institutions on a quarterly basis. The committee required the submission of a yearly progress report on committee forms in order to determine that the investigator was diligently pursuing his goals and to discover as well what serendipitous information might have been acquired during the course of the individual's researches. Equipment purchased with these funds ultimately became the property of the individual institution sponsoring the grantee. The only additional requirement placed on all grantees was that they publish the results of their research in reputable scientific journals of their own choice so that the information might be made public through the scientific media.

Our committee maintained liaison with the National Institutes of Health and other interested research groups. These organizations were advised of current trends in the committee's work and every attempt was made to minimize duplication of effort.

To further provide for informational exchange among grantees, the committee convened three workshops in Boulder, Colo. 1966; Scottsdale, Ariz. 1970; and Newport Beach, Calif. 1972. These workshops permitted scientific participants freedom of discussion and evaluated progress being made among research projects. In conjunction with the American Medical Association's annual meeting in San Francisco in 1968, a presentation of the work of the committee's grantees was held and constituted a report of progress and information to the profession and the public. At that time a statement was issued indicating that the research completed under the aegis of this project in no way altered the conclusions of the 1963 report of the Surgeon General.

Although after 1972 no new research grants were awarded, our committee continued to monitor grants already awarded so that the work originally contracted might be completed. The last funds paid out to a grantee to support this research were disbursed in 1976.

At its final meeting on May 27, 1977, the Committee for Research on Tobacco and Health reviewed the compilation of work which it had supported. In its own evaluation of this massive collection of disparate research efforts, the committee concluded, as stated in the volume, that:

Important contributions had been made to basic medical science as well as to problems associated with tobacco usage. Valuable information has been obtained relating to the distribution, metabolism, excretion, and toxicity of nicotine absorbed by the human body via cigarette smoking. In the area of carcinogenesis, the committee restricted the number of awards because cancer research was being generously financed by the National Institutes of Health and other agencies. Nevertheless, the demonstration of potent cocarcinogens in tobacco tar and the potential value of the measure of inducibility of aryl hydrocarbon hydroxylase as a determinant of susceptibility to lung cancer represent some of the more significant contributions in this area. Emphasis was placed on the impact of cigarette smoking on the physiology of the cardiovascular, respiratory and central autonomic nervous systems. The committee believes that the bulk of research sponsored by this project supports the contention that cigarette smoking plays an important role in the development of chronic obstructive pulmonary diseases and constitutes a grave danger to individuals with preexisting diseases of the coronary arteries. On the central and autonomic nervous system important findings were made related to the effects on behavior and on biochemical mediators elicited by nicotine. Gastrointestinal tract studies include new mechanisms by which nicotine may influence production of peptic ulcer.

"Tobacco and Health," that is this volume, serves as a record of the foundation's sponsorship of 219 separate research projects related to health and the use of tobacco. This volume is the result of efforts of 844 researchers in 13 foreign and 85 U.S. research institutions and universities. These efforts resulted in 795 publications and reports concerning the relationship of tobacco and health. The volume includes abstracts of most of the published reports on research which were supported by the project. The responsible investigator, grantee, approved each abstract. Abstracts were grouped according to one of the seven areas of research to which they were related. These were: (1) Absorption, distribution, metabolism, excretion, and toxicology of nicotine, (2) carcinogenesis, (3) cardiovascular system, (4) central and autonomic nervous system, (5) the gastrointestinal tract, (6) reproduction, and (7) the respiratory system.

Members of our committee carefully studied the reports and condensed and collated them to the summaries which appear at the beginning of each section.

AMA ACTIVITIES ON SMOKING AND HEALTH

Now, other activities which our organization has had on smoking and health show a longstanding interest in the effects of smoking on health. As far back as 1953 our board of trustees banned the advertising of tobacco products in all of our publications; 10 years later, in 1963, the AMA House of Delegates, in addition to approving the research program, approved an educational campaign aimed at young people to discourage the use of all toxic materials, including tobacco. In the following year, the AMA House of Delegates adopted the official position that there is "a significant relationship between cigarette smoking and the incidence of lung cancer and certain other diseases, and cigarette smoking is a serious health hazard."

Since, the AMA House of Delegates has strengthened its positions against cigarette smoking by citing the hazards involved, urging all of our members to set an example for patients by not smoking, and by requesting AMA to expand its educational efforts with both the profession and the public. These antismoking admonitions were summed up in a resolution adopted by the house of delegates in December 1968.

In June 1972, the house of delegates adopted a resolution which stated "that smoking be discouraged during the sessions of the House of Delegates of the American Medical Association." In 1975, the House adopted a further resolution stating "that the American Medical Association urge physicians to continue to act as nonsmoking exemplars to the public."

At the recent annual meeting during June of 1978, the house of delegates reaffirmed existing policy of the association and urged physicians to continue to act as nonsmoking exemplars to the public, to advise patients of the health hazards of smoking, to discourage smoking by means of public pronouncements and educational programs, and to discourage visual presentation in the public media which influence young people by depicting smoking as the norm in the American culture. The House also voted to discourage cigarette sales and smoking in medical and health care facilities—that is, hospitals, doctors' offices, laboratories—to continue cooperation and participation with other organizations in implementing antismoking educational efforts, and to encourage the Federal Government to provide financial support for additional research into the health hazards relating to smoking.

Over the years the American Medical Association has been a member of the National Interagency Council on Smoking and Health, and our association participated in the planning of the Third World Conference on Smoking and Health. Liaison is maintained with appropriate units of the Department of Health, Education, and Welfare. The Department of Health Education of the American Medical Association answers requests on a daily basis for information on smoking and health. This department also publishes and distributes a pamphlet entitled "Smoking: Facts You Should Know." In addition, "No Smoking" signs are distributed for physicians' offices, and posters are available to those who wish to display them, including schools, in this country.

In conclusion, Mr. Chairman, the American Medical Association has a longstanding interest in the effects of smoking on health. In addition to its contributions of time, personnel, and facilities to the AMA-ERF research effort, which exceeded half a million dollars, the association has for many years engaged in a campaign to educate the profession and the public as to the hazards of smoking.

The recent release of "Tobacco and Health" by the American Medical Association-Education and Research Foundation complements these activities of the association. The information contained in this volume is not new, as all of it has been published in scientific journals over the past 14 years. However, it does represent a compilation and new summary of research that adds significantly to the body of knowledge concerning smoking and health.

We trust that these remarks are responsive to the questions raised in your letter inviting our testimony, and I am available for any questions that you may ask of me.

Mr. WAXMAN. Thank you very much for your testimony. We are being summoned to the floor for a vote, so I would like to recess for 10 minutes, and we will resume.

[Brief recess.]

Mr. WAXMAN. Dr. Hunter, thank you very much for your testimony. I gather it is very clear you and those who were involved in

the research project that cigarette smoking is a danger to people's health, that it has consequences both in terms of potential cancer, lung disease, and heart disease.

Dr. HUNTER. Yes, sir.

Mr. WAXMAN. You mentioned that the AMA urges physicians to continue to act as nonsmoking exemplars to the public, and certainly one of the groups that has dramatically dropped its level of smoking have been physicians. I think this has been very important in getting the message to the American people that those who are involved so directly with the public health recognize the danger.

I do not know if you were here earlier for Dr. Gori's testimony.

Dr. HUNTER. Yes, I was.

Mr. WAXMAN. We went into some questions as to why people smoke and why they do not stop, even though they know that they are taking a great risk. What work has been done by the AMA to develop in a sort of intimate doctor-patient relationship, some way of delving into the psychological or perhaps physiological problems that cause people to smoke.

Dr. HUNTER. I think the principal effort that we have made has been through the grant of this project, and that included some of the psychological aspects of the smoking habit, and the neurologic effects of smoking. We have a public educational program that is of real significance, but we do not have any current investigation going on that would lead to those answers.

Mr. WAXMAN. In your opinion what dangers or risks are posed to nonsmokers from smoking? Have you done any work in that field? Do you have any feeling about that issue?

Dr. HUNTER. Unless you are in a very closely closed environment, these dangers are much less significant than, for instance, the smog that you are familiar with occasionally in southern California. There is a significant irritant effect, and there is emotional stress that is very apparent in some people who are nonsmokers when they are involuntarily confronted with the smoker. I believe that these are the most significant.

In a closed environment the inhalation of toxic substances is real.

Mr. WAXMAN. Would you support national legislation to limit the concentration of tar and nicotine in cigarettes?

Dr. HUNTER. I think anything that reduces the hazards of smoking, which is a part of the life of, as you said earlier, 60 million Americans, is beneficial.

Mr. WAXMAN. Do you see health dangers from other chemicals in low tar and nicotine cigarettes such as carbon monoxide?

Dr. HUNTER. Do I see a hazard in them?

Mr. WAXMAN. Do you see a hazard?

Dr. HUNTER. Well, there is an ongoing hazard in any form of smoking.

Mr. WAXMAN. Mr. Segal.

Mr. SEGAL. Dr. Hunter, I would like to just get a little more elaboration on your particular report. Now this was done by—

Dr. HUNTER. You mean the total volume?

Mr. SEGAL. The total volume. This was done by a body of scientists selected by the AMA's research foundation; is that correct?

Dr. HUNTER. We appointed a special committee advisory to the research foundation. They selected the individual scientists who would do the research work.

Mr. SEGAL. That is that advisory body that came to the conclusions of grave danger for coronary artery diseases and also that cigarette smoking played an important role in chronic obstructive pulmonary diseases; is that right?

Dr. HUNTER. Yes. That statement is a statement of the committee.

Mr. SEGAL. Does the AMA generally then take a supportive position on that, or is the next step the kind that you testified to today, one that reinforces the contentions that you have had before about the dangers of cigarettes?

Dr. HUNTER. I would say that it was reinforcing to our previous positions and policy statements.

Mr. SEGAL. So this takes it beyond the specific items that were in previous AMA statements relative to lung cancer, but would now include some confirmatory statements relative to coronary artery disease and respiratory disease; is that it generally?

Dr. HUNTER. I believe that is true. Also, as I said, this is not new knowledge. As a practitioner, I have long held that belief, and I think that most practicing physicians in the country do.

Mr. SEGAL. There was testimony yesterday from the research director of the Council on Tobacco Research, when he was asked about the issue that clinicians have generally noted that pulmonary diseases are found to be more prevalent in smokers than in nonsmokers. As a clinician, is that the impression that you have as well?

Dr. HUNTER. Yes.

Mr. SEGAL. Do you feel that most physicians that you come in contact with and discuss it with have that same conclusion?

Dr. HUNTER. I believe so, yes.

Mr. SEGAL. When this report was issued, there was also a question raised as to whether or not there were dissenting views presented about it. Was the final conclusory statement made supported by all of the people on the advisory board?

Dr. HUNTER. Dr. Singer was the secretarial staff to that committee, and basically the editor of this book, and he said there were no objections raised by any member of the committee.

Mr. SEGAL. Was there a formal vote, and it was found to be unanimous, or was it just everybody through exchanges of communications said, "This is what we think is the conclusion of this large work."

Dr. HUNTER. I would have to defer to Dr. Singer on that.

Mr. SEGAL. Dr. Singer.

Dr. SINGER. The date on that committee's statement is May 27, 1977, which was the last time that the committee met, and the purpose of that meeting was to go through the abstracts, the summaries and so forth, and at that time the committee felt that it behooved the committee to point out what it thought were significant factors which had come up through this research which it had supported, and that statement is a composite statement of the committee through its deliberations. So as far as I know, there were no dissenting votes. It was simply a consensus statement.

Mr. SEGAL. Fine.

Earlier this morning Dr. Gori testified, indicating, in effect, that we now have an epidemic of smoking-related diseases, and one of the issues he raised was if people switched to lower tar and nicotine brands of cigarettes, that that epidemic might well disappear.

Do you have any comment on that kind of position?

Dr. HUNTER. I think I would have to say that it is the position of the American Medical Association that any form of cigarette smoking is hazardous, and that to make that prediction is an individual thought. The development of tobacco-related diseases is a very prolonged time interval, and to project a diminution of those diseases would involve a comparable period of time, and I am not able to project that far ahead. Most cigarette smoking, after all, has developed since World War I. Most cigarette smoking in women has developed since World War II. We use the figure of 40-pack years for the development of illness or disability as a result of cigarette smoking, and the diminution of cigarette smoking might have to be a comparable time frame.

Mr. SEGAL. So you believe that currently we do have an epidemic of smoking-related diseases, and your position would be that smoking less hazardous cigarettes is still a hazard.

Dr. HUNTER. It is still a hazard, that is right.

Mr. SEGAL. We also received some evidence that was brought out in testimony yesterday, the witness was asked that, and didn't have much in the way of confirmatory statements, that recent studies through new apparatus improvements have found that young smokers, even after as little as 1 to 5 years, find diseases of the small airways of the lung.

Is that something that was examined as part of the research work here, and do you have any kind of statements that lend support to the small airways disease syndrome?

Dr. HUNTER. No. Emphysema, in any form, is a disease of the terminal pulmonary anatomy, and the loss of elasticity, which is characteristic of the disease, is, in truth, what leads to chronic obstructive pulmonary disorders.

You can categorize them or you can call them different names, but that basically is the pathology of smoking-related illness, the loss of elasticity.

Mr. SEGAL. So you believe that the pathogenesis does begin in a spectrum that starts with the small airways, and it then progressively moves toward illnesses that can result in emphysema and bronchitis.

Dr. HUNTER. That is true.

Mr. SEGAL. Thank you, Mr. Chairman.

Mr. WAXMAN. Just to follow up on that point, are we talking about damage to the lungs that may be irreversible?

Dr. HUNTER. Yes, sir.

Mr. WAXMAN. There are many people who work on the assumption that while they smoke, if they smoke for 10 years and quit, they will be all right because the Heart and Lung Institute said a former smoker who has not smoked for 10 years runs no more risk of heart disease or chronic bronchitis than someone who had never smoked at all. But if we are talking about irreversible damage during the preceding 10 years of smoking, we may well be talking

about something that may well never be corrected and harm that can never be reversed.

Is that your opinion?

Dr. HUNTER. That is my opinion.

Mr. WAXMAN. It is commonly thought that low tar and nicotine cigarettes are safer, and they appear to reduce the risk of heart and lung disease.

Would you advocate that smokers adjust their smoking habits and avoid health risks by smoking these low tar and nicotine cigarettes if they feel that they cannot stop smoking?

Dr. HUNTER. If they must smoke, certainly they would be better off with the lower tar and nicotine cigarette.

Mr. WAXMAN. Do you feel that if we had a less hazardous cigarette on the market, that we may well avoid the kind of health consequences that we fear from cigarette smoking as we know it now?

Dr. HUNTER. No.

Mr. WAXMAN. In your answer to Mr. Segal's question, you didn't feel there would be a lessening of the epidemic that we are now experiencing and most likely to experience in greater consequence if people switched to low tar-nicotine cigarettes.

Do you think then that it is inconsistent with the idea that it is less harmful?

Dr. HUNTER. That would be conjecture on my part, because other than the creation of our current lower tar-lower nicotine cigarettes, I know of no less hazardous cigarette that has been produced.

Mr. WAXMAN. People want to know what risks they are taking. They enjoy the pleasures of smoking and want to psychologically deal with that. They often will decide that they are going to take a risk because, after all, everything we do involves some kind of risk.

By smoking a low tar-low nicotine cigarette are people reducing the risk of getting some of these diseases, such as lung cancer and heart disease, or are they just merely postponing the time in which they are going to get those diseases? What is your opinion?

Dr. HUNTER. We will have to wait 40 years to find out, because the progression of the development of these disease processes is that long. We presume it to be true, but the human experimentation takes 40 years to find out.

Mr. WAXMAN. What do you believe to be the cause of so many young people starting to smoke?

Dr. HUNTER. They are peer group pressure, the macho image that they associate with it, the desire of every youngster to be and appear older than he actually is or she is.

Mr. WAXMAN. Do you believe advertising plays an important role in the decision of a young person to take up the habit?

Dr. HUNTER. I don't know what influence advertising has. But as part of our antismoking campaign, we use advertising. So organizationally we do believe that it has an effect.

Mr. WAXMAN. Do you believe that cigarette smoking is physically addicting or that a physical dependency is developed by those who smoke?

Dr. HUNTER. It is habit forming, certainly. As to whether or not it is actually addicting, I have had only one clinical experience of a person who had withdrawal symptoms on the cessation of smoking.

They were very heavy smokers and their cessation was cold turkey, and they actually did have some withdrawal symptoms, which is the hallmark of addiction, as contrasted to habituation.

Mr. WAXMAN. Dr. Gori testified earlier that for some people, switching to a lower tar-low nicotine cigarette may be a good transitional way out of smoking.

Do you find that to be a scientifically valid statement?

Dr. HUNTER. I would say that it would have to be an acceptable alternative to the other side of the coin, which is not smoking.

Mr. WAXMAN. On another subject, do you believe that women on the pill run an increased risk of heart disease if they smoke? There is a controversy raised by the tobacco industry that that FDA's warning on oral contraceptives was unjustified. The industry felt there was not sufficient evidence to support the conclusion that smoking in conjunction with taking oral contraceptives increased the risk of heart disease.

Do you have any views or opinions on that?

Dr. HUNTER. I know of no laboratory evidence, and I am not a research scientist. There does seem to be a statistical correlation that justifies the insert information.

Mr. WAXMAN. Dr. Singer, I was interested in asking you about the AMA's report on the dangers of cigarette smoking. I noted that some of the researchers involved in the research for the AMA report were also involved with work for the tobacco industry.

Did any of these researchers object to the conclusions that were reached in the AMA report?

Dr. SINGER. No. As I said earlier, the conclusions were a consensus of committee beliefs, and there were no objections from anyone.

Mr. WAXMAN. In the summary of the AMA report it states that:

The committee believes the bulk of research sponsored by this project supports the contention that cigarette smoking plays an important role in the development of chronic obstructive pulmonary diseases and constitutes a grave danger to individuals with preexisting diseases of the coronary arteries.

By relating this to individuals with preexisting disease of the coronary arteries, would that mean that someone without a preexisting disease would not be susceptible to problems of both heart and lung disease from cigarette smoking?

Dr. SINGER. Not necessarily. They specifically mentioned that because they felt that that had been more or less borne out, not only in these studies, but in others.

Mr. WAXMAN. Did the committee look into the questions of genetic predisposition or other predispositions of individuals to adverse health consequences from smoking?

Dr. SINGER. Well, as you know from looking at this volume, they did support work in this area. The Swedish twin registry studies was one of this group.

Mr. WAXMAN. I want to thank you for your testimony.

Dr. HUNTER. Thank you, sir.

Mr. WAXMAN. We appreciate your attendance.

Dr. SINGER. Thank you.

Mr. WAXMAN. The committee meeting is adjourned, and I thank all those who have come to hear the testimony.

[Whereupon, at 12:15 p.m., the subcommittee adjourned.]

