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**PANEL DISCUSSION**

**MORTALITY OF SMOKERS AND NONSMOKERS**

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**WILLIAM J. NOVEMBER:**

In opening the panel discussion, I should like first to thank Mr. Edward A. Lew for the work he did in organizing it and laying out the basis for the discussion. He was originally slated to be the moderator, but illness has prevented him from appearing on the program today.

There have been discussions at Society of Actuaries meetings of what account might be taken of smoking habits in the underwriting of new applications, but the relationship of smoking to mortality has not in itself come under scrutiny at our meetings. Actuaries naturally have a great interest in the forces which act on mortality. It therefore appeared appropriate that we should expose ourselves officially to the evidence that has been developed and to the various points of view that exist on the interpretation of the evidence.

The topic has been one of continuing lively interest to the public. Press accounts and reports and articles on the subject matter of the panel have appeared with sufficient regularity to make it unnecessary for me as moderator to set a background for the discussion that is to follow. Instead we shall rely on the experts who have graciously consented to appear on the panel to jump right in and to deal with the subject without benefit of a preliminary statement from the chair.

**E. CUYLER HAMMOND, SC.D.:**

Truly new ideas appear to be rare. I would hazard the guess that some ancient Mayan suggested that smoking is harmful if not sinful and that some fellow citizen countered by saying that smoking is pleasurable, a

tribute to the gods, and the best means of avoiding swamp sickness. At a later date, Europeans had an opportunity to express themselves on the subject, and a prominent French diplomat, Jean Nicot, did so quite forcibly in 1560. He claimed that tobacco smoke is a marvelous cure for disease; but other equally distinguished personages saw only sin and destruction in the use of the weed. Such divergent expressions of opinion mixed with emotion have continued to this day. The situation has changed only to the extent that there is now evidence on the subject; and I suspect that members of your Society prefer evidence to opinions. Therefore, I will review the evidence briefly and let you draw your own conclusions.

As early as 1859, a French physician named Bouisson reported that all his patients with buccal cancer were smokers and that pipe smokers tended to develop cancer at the point where they held the stem between their lips. He concluded that smoking caused the cancer and speculated on whether the heat or the tobacco tar or a combination of the two was responsible. In 1936, Ochsner and De Bakey made a similar observation, this time in relation to cigarette smoking and lung cancer. Their clinical findings, together with reports that lung-cancer death rates were increasing and that cigarette consumption was increasing, led them to suggest that cigarette smoking could cause that disease. This view was strengthened when, in 1939, Roffo induced skin cancer in rabbits by painting them with tar extracted from tobacco.

Some years later, other investigators carried out more formal studies and made statistical analyses of clinical data using what has come to be called the "retrospective" or "case-control" method. In such studies, the investigator ascertains the habits or environment of patients having a particular disease such as lung cancer and also ascertains the habits or environment of a sample of patients with other diseases or of a sample of persons drawn from the general population. In no less than twenty-eight studies of this type, more cigarette smokers (and particularly more heavy cigarette smokers) were found in the lung-cancer group than in the control group.

Cancer was by no means the only disease studied in relation to smoking. Clinical studies strongly indicated that smoking is a factor of great importance in relation to a rare disease of the peripheral arteries (Buerger's disease); many physicians suspected that smoking has a deleterious effect upon patients with gastric ulcers; and the known acute effect of nicotine led many physicians to suspect that smoking is bad for patients with heart disease. Furthermore, in several retrospective studies, more cigarette smokers were found among patients with coronary artery dis-

ease than among persons without this disease; and similar studies showed a relationship between cigarette smoking and chronic bronchitis.

Raymond Pearl was probably the first to study smoking in relation to death rates. His data, published in 1938, appeared to show that smokers have a shorter life-expectancy than nonsmokers.

Many people were unwilling to accept the evidence described above. It was pointed out that the retrospective method can sometimes yield very misleading results. Pearl's methods were severely criticized. The acute toxic effects of nicotine do not necessarily indicate that the amount of nicotine absorbed from cigarette smoke produces serious chronic or long term effects. Findings in animal experiments, whether positive or negative, do not necessarily apply to man.

Largely because of these doubts, several large-scale prospective studies were undertaken. Doll and Hill sent questionnaires on smoking to all physicians in Great Britain; Hammond and Horn ascertained the smoking habits of 187,000 men between the ages of 50 and 69; Dorn mailed questionnaires to veterans holding national service life insurance; Best, Josie, and Walker sent questionnaires to Canadian pensioners; Dunn, Linden, and Breslow ascertained the smoking habits and occupational exposures of men in certain occupations in California; Dunn, Buell, and Breslow ascertained the smoking habits of California members of the American Legion and the wives; and, in the latest such study, American Cancer Society volunteers enrolled over 1,000,000 men and women and asked each subject to fill out a lengthy, confidential questionnaire on family history, disease history, physical complaints, occupational exposures, and many other factors, including smoking. In each of these studies, the subjects were traced for a number of years, and copies of death certificates were obtained on those who died. Total death rates and death rates from various diseases were calculated in relation to the smoking habits of the subjects and in relation to certain other factors.

It should be pointed out that these studies were very similar in design to studies made by life insurance companies on death rates in relation to various attributes of policyholders. Indeed, Dorn's study was actually carried out on life insurance policyholders. Death rates of the subjects were lower than death rates of the general population, since there was some tendency to exclude high risk individuals. For example, Hammond and Horn attempted to avoid enrolling subjects who had cancer, subjects who were extremely ill, illiterates, and persons (such as migrant laborers) who could not have been traced. In five of the seven studies, questionnaires were sent out by mail; and this tended to exclude seriously ill persons, since such persons would be unlikely to reply. Owing to the partial exclusion of ill persons, age-specific death rates were especially low during

the first few months but rose in succeeding years. Thus, in many respects, the mortality experience of the study population was similar to mortality experience among life insurance policyholders. In the latest study, I was able to make an analysis confined to persons, most of whom would have been accepted for ordinary life insurance; that is to say, I excluded subjects who at the time of enrollment said that they were "sick at present" or had a history of cancer, heart disease, stroke, or high blood pressure. The differential mortality between cigarette smokers and nonsmokers in this selected group was somewhat greater than the differential mortality between cigarette smokers and nonsmokers in the rejected group.

Most of these studies were either deliberately confined to men or were largely confined to men due to the nature of the group (e.g., the majority of British physicians and the majority of American veterans are men); and no findings have yet been published on the female subjects of the studies which included women. The results of the seven studies are in good agreement with each other.

It is of interest that the first prospective studies were undertaken primarily to ascertain whether or not there is a significant relationship between smoking habits and death rates from lung cancer. Such a relationship was found. What is far more important, a high degree of relationship was found between smoking habits and total death rates from all causes combined. This may be summarized as follows:

The rank order of age-standardized death rates among men classified by their lifetime smoking history was (from lowest to highest): (1) men who never smoked regularly; (2) men who smoked only pipes; (3) men who smoked only cigars; (4) men who smoked cigarettes and also smoked pipes or cigars; and (5) men who smoked only cigarettes. The death rate of pipe and cigar smokers was only slightly higher than the death rate of men who never smoked; but the death rate of men who smoked only cigarettes was far higher than the death rate of men who never smoked. The mortality ratio of men with a history of only cigarette smoking (i.e., the death rate of these smokers divided by the death rate of men who never smoked regularly) varied from 1.44 in Doll and Hill's British physician study based upon 269,000 person-years of exposure to 1.79 in Dorn's United States veterans study based upon 1,312,000 person-years of exposure. This variation was due at least in part to the fact that the studies covered different age ranges. During the first thirty-four months of follow-up of 422,094 men enrolled in the latest study, the mortality ratio of men with a history of only cigarette smoking was 2.37 in age group 40-49, 2.01 in age group 50-59, 1.65 in age group 60-69, 1.49 in age group 70-79, and 1.18 in age group 80-89.

Among current cigarette smokers with a history of only cigarette smok-

ing, age-specific death rates increased with number of cigarettes smoked per day and with degree of inhalation of the smoke; and death rates were far higher among those who started smoking at a young age than among those who started smoking at an older age. For example, among current cigarette smokers aged 40–69 in the latest study, the mortality ratios were: (1) 1.57 for men who smoked 1–9 cigarettes a day and 2.18 for men who smoked 40 or more cigarettes a day; (2) 1.64 for men who claimed that they did not inhale the smoke and 2.22 for men who said that they inhaled deeply; and (3) 1.42 for men who started smoking after the age of 25 and 2.29 for men who started smoking under the age of 15. Most of the pipe and cigar smokers said that they did not inhale the smoke or only inhaled slightly. Those few who said that they inhaled moderately or deeply had death rates considerably higher than the death rates of nonsmokers.

Of particular interest was the death rate of ex-cigarette smokers as compared to the death rate of those who were currently smoking cigarettes at the time of enrollment. The death rate of *recent* ex-smokers (i.e., those who had stopped smoking less than a year prior to enrollment) was *higher* than the death rate of current smokers. A further analysis showed that the recent ex-smoker group was heavily weighted with men who had stopped smoking following a heart attack or some other illness or physical complaint; and this was sufficient to account for their high death rate. However, the death rate of ex-cigarette smokers who had given up the habit some years prior to enrollment was considerably lower than the death rate of current cigarette smokers (though not as low as the death rate of men who had never smoked).

Now let us turn to death rates by cause of death as stated on death certificates.

The relationship between smoking habits and death rates from *coronary artery disease* closely paralleled the relationship between smoking habits and death rates from all causes combined. This is not surprising, since coronary artery disease accounted for a large proportion of all deaths. Among men under the age of 70, over half of the excess mortality associated with cigarette smoking was accounted for by the excess mortality of cigarette smokers from coronary artery disease.

For many diseases, the death rate of cigarette smokers was slightly higher than that of nonsmokers, and for several diseases the mortality ratio of cigarette smokers was very high indeed. Notable in this respect were lung cancer; cancer of the buccal cavity, pharynx, larynx and esophagus; gastric ulcers; emphysema and chronic bronchitis; and non-syphilitic aortic aneurysm. The death rate of cigarette smokers was sever-

al times as high as the death rate of nonsmokers from all the diseases just mentioned.

The death rate from lung cancer was about ten times as high for men with a history of cigarette smoking as for men who never smoked, and the mortality ratio increased greatly with amount of cigarette smoking. The excess death rate of cigarette smokers from lung cancer accounted for about 16 per cent of the total excess death rate of cigarette smokers as compared with nonsmokers. Ex-cigarette smokers who had given up the habit some years prior to enrollment had lower lung-cancer death rates than did current cigarette smokers.

Pipe and cigar smokers had far higher death rates than did nonsmokers from cancer of the buccal cavity and pharynx; but cancer of these sites accounts for only a very small proportion of all deaths.

The findings on death rates in relation to smoking habits are based upon over 4,200,000 person-years of exposure to risk of approximately 1,123,000 men enrolled in seven prospective studies designed in the same way as studies made by life insurance companies on their policyholders. The validity of the findings on total death rates (from all causes of death combined) can hardly be doubted. Findings on death rates from specific diseases are subject to inaccuracy for the reason that the cause of death entered on a death certificate is not always correct. However, if (due to inaccuracy of diagnosis) the mortality ratio of cigarette smokers from some particular disease (e.g., coronary artery disease) has been overestimated, then it follows that the mortality ratio from one or more other diseases has been underestimated (since the total death rate is the sum of the death rates from each specific cause).

It has often been said that the finding of an association between two variables does not necessarily prove that a direct causal relationship exists between them. This, of course, is true. However, the term "causation" seems to mean different things to different people. Therefore, in this discussion I will avoid the use of the words "causation" and "cause." The important point to consider here is whether or not the evidence is such as to warrant the conclusion that the death rate of a group of cigarette smokers is higher than it would have been among this same group of people had they refrained from smoking.

The principal evidence on the question has come from the studies on human beings which I have just described. Additional evidence has come from microscopic studies of the lungs of people who died of various causes. These studies have shown a very high degree of relationship between cigarette smoking and damage to the bronchial tubes and the lung parenchyma. Findings in the bronchial tubes are such as to give very strong

support to the theory that cigarette smoking leads to a series of changes which in some individuals eventually leads to lung cancer. Findings in the lung parenchyma give strong support to the theory that cigarette smoking leads to damage which impairs lung function (and this is supported by evidence that cigarette smoking leads to shortness of breath).

Experiments on human beings have shown that, due to nicotine, cigarette smoking produces acute effects upon the heart and circulatory system; and that, due to carbon monoxide, cigarette smoking results in a conversion of some of the hemoglobin in the blood to carboxyl-hemoglobin. An experiment on cigarette smokers with gastric ulcers showed that recovery is more likely if the patients give up smoking than if they continue to smoke.

Opinions vary as to whether or not findings in animal experiments have an important bearing on the problem at hand. Since mice and other animals (except for a few monkeys) have not been persuaded to smoke cigarettes, there is no direct evidence of the effects which cigarette smoking might produce in animals. There is no doubt of one thing: mice and other rodents have an extremely high death rate if placed in a chamber with a fairly high concentration of cigarette smoke. Cigarette smoke contains several carcinogenic agents, these being defined as chemicals capable of producing cancer in experimental animals; and cancer of the skin has been produced in experimental animals by painting the skin with material condensed from cigarette smoke. So far as I know, no one has succeeded in inducing lung cancer in experimental animals by placing them in a chamber filled with cigarette smoke; but if the concentrate of smoke in the chamber is as high as the concentration of smoke inhaled by some human cigarette smokers, the animals usually do not live long enough to develop cancer of the lung or any other site.

Such is the nature of the evidence. My own conclusion is that cigarette smoking is seriously harmful.

ROBERT C. HOCKETT, PH.D.:

You have heard an account of the epidemiological studies that are considered to have established a statistical relationship between cigarette smoking and mortality from a number of diseases, especially carcinoma of the lung. It is logical that I should follow this presentation with my discussion, since the research program of the Council for Tobacco Research\* was set up in large part for the purpose of investigating the meaning of these statistical relationships.

The question is: Just what can we conclude from the reported associa-

\* Formerly called the Tobacco Industry Research Committee.

tions and how best can they guide us to practical measures for reducing the toll of these diseases?

Of course, it is being said in some quarters that these epidemiological studies are sufficient and that further research is not needed except to clean up details. It is being stated, vociferously and often, that the statistical relationships show conclusively that cigarette smoking is the primary cause of lung cancer and of several other diseases and also is a contributor to the etiology of still others.

There is one very basic and overwhelmingly important question, however, that is not answered by these statistical associations, and this question has nothing to do with the mathematical handling of the data. The question is simply this: Do people really contract these diseases because they smoke, or is it rather that the kinds of people most likely to develop the diseases need or wish to smoke? The answer to this question is very important for the development of preventive measures. Both insurance companies and the processors of tobacco have a humanitarian interest in preventive measures. In addition, they both have an economic interest in the long survival and continuing health of their clientele.

When a population is divided into two groups on the basis of a single characteristic such as the smoking or nonsmoking of cigarettes, I think no actuary would expect these two groups to be perfectly balanced with respect to all other characteristics. From casual observation, we all think of a "typical" heavy cigarette smoker as being quite a different person in many respects from a nonsmoker or even from a "typical" pipe or cigar smoker. The only question on which there is any difference of judgment so far as I am aware is in respect to the magnitude or significance of such differences and the extent or degree to which they may render these divisions of the population noncomparable for the purposes in view.

We might put the matter in another way. Nonsmokers actually constitute a minority of the total adult male population. Are nonsmokers cautious and self-protective in a multitude of different ways which, as a pattern of life, contribute materially to their relative longevity? Has tobacco use actually anything at all to do with it?

There are numerous hints and leads accumulating to show that smokers and nonsmokers, as well as the various types of smokers, are really quite different kinds of people. It will require considerable more investigation, however, to evaluate such data fully.

There are other schools of thought, though these have been less vigorously advanced and have not impinged upon the public attention to the same degree. Thus it has been suggested that the victims of lung cancer arise from a small minority of the total population who are especially pre-

disposed to the disease by reason of constitutional characteristics, perhaps combined with the effects of many kinds of life experiences. The adherents of this concept are inclined to regard tobacco abolition attempts as rather like burning down a house in order to roast a pig or abolishing lobster and mushrooms because a few people are allergic to these foods. Their disposition is to look for ways of identifying the relatively small minority of lung-cancer susceptibles so that this minority can be given special protection without disturbing the habits and practices of the great majority. This concept or theory does suggest a considerable number of investigations that can be carried out to gain a better picture of the antecedents of lung cancer.

A third school might be called the "indirect association school." If heavy cigarette smokers eat more saturated fats than nonsmokers, and if a high intake of saturated fats is really conducive to arterial disease, then there might be an association of smoking with arterial disease which, however, would reflect no direct causal contribution to its etiology. This hypothesis has been put forward quite seriously but is cited here only by way of illustration, since it has not yet been confirmed. The principle is one very familiar to statisticians.

The research program of the Council for Tobacco Research is experimentally oriented. In order to investigate tobacco use and health, we conceive it to be our task to investigate each of the diseases which is reported to be statistically associated with the smoking of tobacco products in order to determine in each case whether the association reflects direct causation or an indirect contribution or whether it is due to an incidental or fortuitous relationship that is meaningless to etiology.

Let me emphasize that we do *not* believe that the methods of epidemiology alone can solve the problem of causation or degree of causation. To determine whether tobacco use causes or aggravates a disease, we feel we must have some direct knowledge of *how* and *to what extent*. The history of medicine is full of examples of the misinterpretation of associations. Malaria was associated with swamps and stagnant water, and this led to the interpretation that it was *caused* by inhalation of "bad air," whence the name that still persists. Pellagra was associated with the eating of corn and was long believed to be *caused* by a poison or infection in the grain. To be sure, the associations were right in suggesting that stagnant water had *something* to do with malaria, for mosquitoes do breed in the swampy water. Corn had *something* to do *somehow* with pellagra—but only because certain impoverished persons who ate corn were not getting a balanced diet. Such vague knowledge as a general association is not adequate in the present sophisticated age, and *effective* measures of control depend

on acquisition of better and more complete knowledge. Though both diseases are now well on the way toward world-wide eradication, malaria was not eradicated by abolition of swamps or pellagra by abolition of corn, although the complete eradication of corn as a food was once proposed!

I would like to make a few points about lung cancer.

How much has lung cancer actually increased in this century? We are being told that it is skyrocketing and approaching epidemic proportions. There is much doubt whether this is true. Indeed, the incidence curves show signs of approaching a plateau.

There is a considerable literature showing that lung cancer was well known during the entire nineteenth century by those pathologists who happened to be interested in making autopsy examinations, though not by physicians generally. But lung cancer was usually recognized only post mortem and was rarely diagnosed clinically. Since autopsies were uncommon, we have no way of knowing the true prevalence of the disease in that era.

There is good qualitative evidence, however, of the progressively increasing ability of clinicians to diagnose the disease during the present century, especially as new diagnostic tools such as radiography, bronchoscopy, exfoliative cell examination, and others were perfected and used more generally. A number of hospitals scattered about the civilized world have published reports comparing clinical diagnosis with post-mortem findings. During the early years of the twentieth century, carcinoma of the lung was missed in these series as often as nine times out of ten during diagnosis of the living patient. As time went on, the score improved until at present 80 or 90 per cent of such cases in some series have been correctly diagnosed in living patients. If the diagnostic score was formerly no better than this in the hospitals that have published such reports, it is very likely that it was far less accurate among practitioners generally. The effect of a general increase in awareness of the disease coupled with ability to diagnose it *in vivo* would be to swell the figures for *reported* mortality for bronchogenic carcinoma.

It is certain, therefore, that the curves showing *reported* mortality from this disease through the years are considerably steeper than the "true" curves ought to be. We do not know how much too steep they are, and it is not likely that we ever shall. Opinions differ from the view, at one extreme, that there has not been *any* real increase at all, to the view that *most* of the apparent increase is real. The difference between these two views may be the difference between concern and panic. The difference between concern and panic may add up to the difference between enthusiasm for concentrated, assiduous, and thoughtful analytical investigation and

demand for immediate and precipitous action, whether or not well grounded, and despite the risk that it may degenerate into fruitless gesturing.

We can all agree that the existence of any lung carcinoma at all is sufficient reason for concern and for concentrated hard work.

The several theories cited suggest many experimental approaches to the problem. It has been pointed out that only a small minority of persons develop this disease, even though they are heavy smokers of cigarettes. This fact is strong evidence that some combination of factors must distinguish those who do from those who do not.

A number of studies suggest a relation between lung-cancer incidence and a history of previously damaging lung diseases. Tubercular infections rank high in these reports. Autopsy studies also indicate that lung-cancer victims have had a high prevalence of chronic or repetitive lung disease, as well as pathological changes in the endocrine glands. These may be clues to predisposing conditions.

Animal experiments also promise to help solve the lung-cancer problem. We know the role that animal experimentation has played in the conquest of infectious diseases. The study of constitutional diseases is more difficult experimentally, but there are very hopeful signs of progress.

When the statistics linking lung cancer with cigarette smoking were first published, a number of investigators plunged into animal experiments. It was assumed that tobacco smoke must contain some carcinogenic substance. The assumption was that this might be identified and removed. In many laboratories, mice, rats, hamsters, hens, rabbits, and dogs were induced to inhale cigarette smoke by means of a variety of ingenious mechanical devices. No doubt it was expected that these animals would develop lung cancers. Then, it was supposed, the work of fractionating smoke to identify and remove the dangerous substances could begin. But there was a hitch in the plan. The smoking animals did *not* develop cancers of the lung. This raised questions. Are these species capable, biologically, of developing lung cancers of the type prevalent in humans? This was answered in the affirmative by the use of several radioactive materials planted into their lungs. Another question arose. Can the agents that produce cancers readily on the skins of mice really act similarly in the lung? To find out, mice were made to inhale fumes of pure methylcholanthrene. These mice did not develop lung cancers either.

Evidently, the resistance of the normal intact lungs of mice is very great. Under certain conditions these potent agents sometimes produced cancers if introduced intratracheally in solutions or suspensions containing high concentrations. Trauma and infections often seemed to play a role in this "success" under rather extreme conditions.

At this point, historically, a kind of breakthrough occurred on the experimental front. Dr. Kotin and his group in southern California subjected mice to a series of three adapted influenza virus infections and then exposed them to a carcinogenic synthetic smog. A considerable proportion of these mice developed lung carcinomas resembling the prevalent human type. The smog alone produced no carcinomas, and the infections alone also produced none, though the viruses did induce proliferative changes and squamous metaplasia.

At about the same time and quite independently, the Leuchtenbergers infected mice with a virus that damages the respiratory tree and followed this by subjecting the mice to inhalation of cigarette smoke. They obtained no cancers, but some of the mice, rather erratically, showed inflammatory reactions. Indeed, some of the control mice, which had neither virus nor smoke, also showed such changes. Those receiving smoke alone showed them just a little more often. Virus-treated animals had a high prevalence of inflammatory changes, while those receiving both virus and smoke showed still more, with evidence of a synergistic effect.

These studies tempt speculation. Can we conclude that human lungs must first be damaged by viruses or something that produces equivalent changes before they become susceptible to the action of inhaled carcinogenic agents, which are ubiquitous in our modern environments? I do not think we can draw any *conclusions* at present, but such a speculation does appear to provide a quite reasonable working hypothesis. More thorough and sophisticated clinical studies may help test the hypothesis. We are in the pilot stage of developing such a prospective clinical study, which we hope will be under way shortly.

Perhaps we are now close to having an animal model by which many other questions about lung cancer can be tested. If we can regularly produce a predicted level of incidence of bronchogenic carcinoma in animals by a combination of virus infection and inhalation of a potent carcinogen, we can begin to test the effects of a whole series of other factors, added to the system or subtracted from it, one by one. Such factors include the effects of liver damage, which impairs its ability to oxidize and inactivate carcinogenic substances; the effects of age, sex, and nutritional condition; the effects of stimulation or inhibition of the reticulo-endothelial system; the effects of overabundance, insufficiency or compositional changes in tracheobronchial mucus; the effects of added hormones or their diminution; the effects of stimulation or inhibition of the ciliary activity. There are many others. Thus it seems probable that the interaction of numerous factors can eventually be pieced together to show how they combine to produce respiratory tree malignancies, at least for mice. But extension of

such studies across strain and species lines can undoubtedly produce hints and leads for human clinical studies.

What about tobacco smoke? We seem to have left it behind in this discussion. I think we have left it behind quite properly. We need a better picture of the basic factors that can combine to produce this disease before we can hope realistically to determine whether tobacco smoke really can play any role, major or minor, or attempt to define what that role may be.

The idea that cigarette smoke acts simply and directly as a carcinogenic agent has not borne fruit. In our program we have tried a full score of bio-assay methods in the attempt to estimate its "carcinogenic activity" in relation to that of substances known to be active. None of these methods has been wholly satisfactory, but they add up to a picture of tobacco smoke as having a very equivocal level of activity or none at all. The chemists tell us that by refined techniques they can detect infinitesimal traces of benzpyrene and of its cousins and its aunts in tobacco smoke. But chemistry today can detect such things at levels far below that of any biological significance. The chemists have applied the same kind of techniques to coffee, flavoring extracts, and a great variety of foods. Most of these contain whole lists of substances that in high concentrations would be considered poisons. In trace quantities, most of these substances are harmless, and we could not avoid them without starving to death. The proof of the pudding lies not in chemical identification of ingredients, though chemistry is infinitely helpful, but in properly conceived *biological* studies to relate significant over-all effects, if they exist, to their origins. It is entirely irrelevant whether tobacco smoke contains one, ten, or one hundred carcinogenic substances unless these really exert some biological effect. The search for the "effective carcinogen" in smoke appears today to have been a wild-goose chase, biologically speaking.

Now there are several new working theories. One is that tobacco smoke, though not an effective carcinogen, may act as a *promoter* of cancer development once the initiation has been accomplished by an agent from some other source. But the whole theory of initiation and promotion is quite controversial, and nearly all of the work that has given rise to the concept has been carried out on animal skin. Before the concept can be carried over to the lung tissues, I think that it will be necessary to establish the very existence of any basic relationship between initiators and promoters in the lung, using single chemically defined agents. I think this can and probably will be done, but the hypothesis that tobacco smoke can act as a promoter of lung cancer will have to await further basic studies before it can be evaluated.

Then there is the concept that tobacco smoke might make an indirect contribution to lung-cancer etiology by paralyzing the activity of the cilia which propel the mucus blanket upward over the bronchial epithelium to carry away inhaled debris. According to this hypothesis, the main effect of tobacco smoke and of many other inhaled agents might be merely to prolong contact of lung tissue with carcinogenic agents from other sources. This concept also needs testing in whole, intact animals in order to shed light on the relative importance of ciliary activity in the total machinery of lung cleansing. Such studies are under way at present.

In the search for the origins of lung cancer, there has been much reliance on the presumption that an inspired agent, entering the lungs by inhalation, may be the main causative factor. However, Stanton has shown that intravenous administration of carcinogenic agents can produce carcinoma in the lungs of animals in which areas of infarction, scarring, and regeneration have been produced by injection of plastic microspheres. Also, Herrold has obtained pulmonary carcinomas in 100 per cent of a small group of animals following introduction of a carcinogenic agent directly into the stomach. These studies show that introduction of agents by routes of entry other than inhalation can produce cancers of the lung. Indeed, it has been somewhat easier to produce lung cancer in animals by intravenous or intragastric administration of agents than by inhalation or intratracheal infusion.

The implications of these observations are tremendous. We do not really know that inhalation of substances has anything at all to do with the origin of human lung cancer. Yet, in the general preoccupation with the theory of inhalation, especially that of cigarette smoke, scientists may have been neglecting the study of mechanisms that now appear to have valid experimental support. Scientific investigation cannot afford to rely on presumptions.

I have not left much time for discussion of cardiovascular diseases, but I would like to make a few points.

The first is statistical. It has been reported that a number of cardiovascular diseases are more prevalent among smokers of cigarettes than among nonsmokers. Since these diseases are much more frequent causes of mortality than cancer of the lung, the "excess deaths" from cardiovascular disease are much greater in total numbers than the "excess deaths" from lung cancer. The moderate increase in the age-adjusted death rates from arteriosclerotic heart disease certainly does not match the sharp rise in cigarette smoking.

A second point is the remarkable difference, statistically, between the incidence of cardiovascular diseases in cigarette smokers and in nonsmokers

of pipes and cigars. Because nicotine produces measurable effects on the cardiovascular system, there was formerly a tendency to assume that this alkaloid might somehow be responsible for the reported increase in the incidence of heart and artery diseases among smokers. Yet there are a number of studies which suggest that the amounts of nicotine actually absorbed by pipe and cigar smokers are quite similar to those absorbed by cigarette smokers. If these various smoker types actually do get about the same amount of nicotine, then nicotine certainly cannot be responsible for the greater amount of circulatory system diseases in cigarette smokers. To understand better this important point, we have set up several studies aimed at refining the methods for determining nicotine absorption and total "physiological" smoke exposure by humans and by animals.

As mentioned before, we know from psychological and anthropometric studies that typical cigarette smokers are statistically different people from typical pipe or cigar smokers. If the suggested role of nicotine is definitely ruled out of the picture, we can concentrate upon the study of other possible factors. It could be that the smoking of cigarettes appeals especially to persons of a temperamental disposition that tends also to predispose them to cardiovascular diseases. Friedman, Russek, and many others have produced evidence that tense, anxious people under stress, or persons who have certain built-in ways of reacting to stress, may be the special candidates for heart diseases. These people may also be especially likely to smoke cigarettes or to smoke them more heavily than the general population.

Several prospective epidemiological studies have shown that smokers do not develop chronic angina any oftener than nonsmokers and some studies show a lower attack rate in smokers. Since angina is considered to be a manifestation of relatively generalized and progressive coronary artery sclerosis, this observation suggests that smoking has no effect in *producing* or *accelerating* the atherosclerotic process. Animal experiments tend to reinforce this conclusion. Moreover, autopsy studies by Wilens and Plair have revealed no significant difference in the degree of sclerosis of the coronary arteries related to the amount of smoking. Hence the statistical correlations between cigarette smoking and cardiovascular disease mortality still remain largely a mystery. Hypotheses which have been advanced by way of explanation include (1) the selection hypothesis mentioned above and (2) the possibility that, when an advanced atherosclerotic condition has developed, smoking might in some way help precipitate an acute episode. Since the mechanisms leading to thrombosis and infarction are still veiled in mystery, it may be difficult to test this latter speculation realistically for some time to come. It also may be worth noting here that habitual smokers tend to have lower prevailing

blood pressures than nonsmokers, and deaths from diseases related to hypertension have been declining for some years.

It has become quite clear that in cardiovascular disease research a clear distinction must be maintained between (1) measurable physiological effects of nicotine or smoking, on the one hand, and (2) contribution to production of a disease, or (3) aggravation of existing disease, on the other. We know a good deal about nicotine and smoking effects. Several substantial monographs are filled with such data. But I am unaware of any clear evidence that any such effects actually contribute to the *production* of any cardiovascular disease. As for aggravation, there are a few suggestive indications in a few uncommon conditions.

Recently there is concern over the reported rise in chronic respiratory diseases such as various forms of bronchitis and emphysema. I am afraid there is no opportunity for adequate discussion of these because of my time allotment. We seem still to be in the stage of trying to define the various clinical entities within this group, to establish differential diagnoses, to describe their natural histories, and to correlate symptoms and functional changes with pathological observations. Until a great deal of this has been accomplished, the problems of etiology will be very difficult to resolve. We are considerably augmenting our program of study in this field at the physiological level through animal work and at the clinical level.

In summary, then, I would reiterate the following points:

1. Comparisons of smoking and nonsmoking populations cannot determine whether smoking affects mortality rates as a whole or from any particular disease because of the factor of selection. It is quite uncertain whether populations separated on the basis of smoking practices are comparable in other respects.
2. Solutions to problems of etiology must come from suitably devised and penetrating experimental and clinical studies of the disease entities in question.
3. Distinction must be preserved between measurable *effects* of tobacco use, on the one hand, and possible contributions to disease *causation* or the *aggravation* of pre-existing disease, on the other hand. The possibility of such contributions can be evaluated only against an adequate picture of the interacting basic factors, intrinsic and extrinsic, which can produce the disease.
4. Progress in such experimental and clinical studies shows definite promise of producing practical preventive measures, as, for example, the use of immunization procedures to prevent and control respiratory-tree infections that appear to predispose to carcinoma of the lung.
5. Recent animal experiments employing routes of administration of

carcinogens other than through inhalation suggest that the inhalation route may have been overstressed in the study of human carcinoma of the lung.

6. The epidemiological picture of the cardiovascular diseases makes it difficult to rationalize a significant role for tobacco use in the complex of causative or aggravating factors.

7. The Council for Tobacco Research is committed to a continuing and expanding program of experimental and clinical studies of the diseases reputed to be associated with smoking.

W. HARDING LE RICHE, M.D.:

As I could not exactly foresee what Dr. Hammond and Dr. Hockett were going to say on this interesting subject, I have had to use a little clairvoyance. So I will start my talk in the sphere, not of parapsychology, but of ordinary psychology. May I tell you something about the psychology of the physician.

In general, the physician is taught to think on his feet. As he observes, he analyzes, and he acts. He cannot, by the nature of his activities, always wait until the final word is said on a problem. He reviews all the evidence, and then he recommends action for the public good. And, in connection with the public good, the following bodies or groups have, to my knowledge, clearly stated that cigarette smoking is harmful: the American Cancer Society, the American Heart Association, the Canadian Medical Association, the British Ministry of Health, the Canadian Department of National Health and Welfare, the American Public Health Association, the Royal College of Physicians of London, the Canadian Cancer Society, the British Medical Association, and the Medical Research Council of Great Britain [5, 10].

On the other hand, no one claims that cigarette smoking is the one and only cause of lung or other pathology. There must be other associated causes as well [5].

This is where the epidemiologist enters the scene. He is usually, but certainly not invariably, a physician, often trained in another discipline as well, and, in addition, in preventive medicine and public health.

The epidemiological approach is comprehensive. Methods of study include clinical observation, laboratory determinations of all types, and statistical analysis. The epidemiologist studies group manifestations of disease.

In his approach he is interested in the total ecological picture. He tries to see a dynamic interaction between the host, the agent or agents, and the total environment. By the very nature of his outlook and timing, he

tries to be fair, judicious, and reasonable. Of course, this paragon of all the virtues does not really exist, but we do try!

As to the interest of the actuary in this whole business, I suppose that he wants to know whether smokers should have their premiums raised. As has been shown by Dr. Hammond, there is excess mortality among smokers. Whether this is large enough to move the actuary to action, I do not know, but the fact that we are discussing the matter means that he is certainly considering the question.

In regard to environmental sanitation, neither Dr. John Snow in London, who in 1849 recommended the removal of the handle from the water pump round Broad Street, in order to control cholera, nor Lemuel Shattuck, who tried to clean up Boston at about the same time, knew anything at all about the germ theory of disease. But they were sure that impure water carried certain diseases. This was based on reasoning supported by observation and arithmetic. They did not know all the minute details of etiology. Nevertheless, action to clean the water helped vastly to control many water-borne diseases like cholera, typhoid, and dysentery.

In our era the same situation applies to air sanitation. We do not know all the details of related disease etiology, but we can clean the air. Not only do we pollute our circumambient air by industrial processes, internal-combustion engine fumes, and atomic explosions, but owing to some strange perversion, we deliberately place smoke in our lungs.

Our immediate need therefore is to clean our air by eliminating industrial contaminants and automobile fumes and by developing some less harmful vices than cigarette smoking. I do not see why we, as adults, should play at being smoke-breathing dragons.

On the assumption that Drs. Hammond and Hockett will already have discussed lung cancer at some length, I will try to state a unitary theory to cover certain other diseases in which there is excess mortality in cigarette smokers. These conditions are coronary artery disease, aneurysm and Buerger's disease, gastric and duodenal ulcer, chronic pulmonary disease, cancer of the bladder, liver cirrhosis, and probably also cancer of mouth, tongue, lip, larynx, pharynx, and esophagus [10].

Our evidence suggests that cigarette smoking is harmful [5, 8]. Whether such effect is due to irritation or to a direct carcinogenic action is possibly immaterial. Detailed mechanisms will be worked out. It may well be that the effect in the lungs may be mainly due to irritation, and in other parts of the body it may be due to a chemical effect. It could be that all effects in associated cancer etiology may be due to a biological trigger action, so that the final hatchet man may be a virus.

As far as the effect of air pollution, including smoking, is concerned,

there appears to be a biological gradient of lung disease. This has been shown experimentally by exposing mice to tobacco smoke and producing pathological changes [9] which are similar to what is produced in the bronchi of cigarette smokers. In this particular experimental approach cancer has not been produced in mice, but the stimulus has probably not been strong enough or for as long a period as may be necessary. It may be said here that bronchogenic carcinomata have been produced experimentally in hamsters and in dogs, the latter by a very interesting experimental design [2].

The gradient in man would be as follows: cough, shortness of breath, chest pains, hoarseness, chronic bronchitis, emphysema, and cancer, with other associated pathological changes.

Obviously, the interpretation of pathological changes is difficult and at times controversial. More recent work on lung pathology gives further support to this whole matter of a biological gradient of lung disease stimulated and enhanced by smoking [1].

The next clamant question which arises is why smoking should lead to excess deaths from coronary disease. Perhaps we might explain it by stating that the heart and lungs function as a unit, like the rest of the body. In the lungs, smoke, which includes carbon monoxide, and other air pollutants may lead to epithelial damage and alveolar rupture, which, in general, results in decreased oxygen absorption [8]. This situation is often associated with diseased hearts. The hearts are diseased in terms of atherosclerosis, hypertension, and in coronary artery thrombosis.

Incidentally, these three phenomena do not always coincide and may be three separated phenomena. It should be remembered that at least one-half of patients who suffer coronary infarctions have normal or low blood pressures.

Superimposed upon this situation is the pharmacological action of tobacco smoke, one of which is the constriction of the peripheral blood vessels. It does not appear that tobacco is associated directly with essential hypertension. In smoking patients with coronary heart disease there appears to be no increase in myocardial blood flow during exercise [3]. It would appear that in the already-diseased heart patient, cigarette smoking may be a final precipitating factor in development of a cardiac infarct. The exact mechanism involved is at present obscure.

There is some controversy about the role of smoking in gastric and duodenal ulcer. It could be that smoking merely exacerbates the situation rather than acting as a causative agent. This may also apply in conditions like cancer of the bladder, liver cirrhosis, and cancer in other sites.

As far as general occupational and other risks are concerned in lung

cancer, it is clear that it is dominantly a disease of males and that it occurs more commonly in certain occupations such as chromate and nickel miners. It is relatively uncommon in farmers, physicians, and clergymen, but it appears to be higher in the lower social and economic strata [7].

Urban males have more lung cancer than rural males. In females the same trends are evident, but differences are smaller [7].

The fact that British-born male migrants to New Zealand [6] and South Africa [4] have higher death rates from lung cancer than the local population suggests a long-term early environmental influence in the etiology of the condition. This influence could be all the chemicals involved in air pollution.

It is a matter of great interest that Seventh-Day Adventists [11], who do not smoke, have relatively far fewer lung cancer deaths than do smokers.

Obviously, in a democratic society, cigarette smoking cannot be stopped by edict, but active steps should be taken to teach children and young adults about its potential dangers. This, too, is the responsibility of a democratic society. And it is a responsibility which should be accepted seriously by all persons and agencies who are interested in the health of the people. This issue can no longer be ignored.

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*Upon completion of the formal presentations by the panel members, an informal discussion period followed during which comments and questions were invited from the floor by the Moderator.*

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MR. ROBERT E. BEARD: I was very interested when this subject came up for discussion because for some years I have personally been actively interested in the study of lung cancer and smoking. My interest in mortality started a long time ago, as may be noted from the *Journal of the Institute of Actuaries*. Various mortality laws have been suggested, but I have always looked for a set of data to throw light on how the organism deteriorated and how this deterioration led to ultimate death. I will confine my remarks to lung cancer and smoking because this is easier than bringing in the whole aspect of other diseases.

By way of background, some years ago I made a study which forced me to sort out some of the theoretical work, and I concluded that to get a picture of the mortality process it would be necessary to move away from the actuarial concept of mortality as a rate and move to a different concept, namely, the distribution of deaths by age. This becomes apparent in studying animal mortality or in analyzing specialized medical investigations. I required suitable data exhibiting sufficiently marked variation to provide a very sensitive test of any theories, and the mortality from lung cancer thus seemed a very good set of data. In England and Wales, for example, the mortality among males from lung cancer in 1962 was about 20,000 deaths, or one death in fourteen, as compared with two hundred or so deaths at the beginning of the century.

In building a model, I came to the conclusion that there were three factors to take into account: a factor related to the year of birth, a factor related to the calendar year of experience, and a factor dependent on age. The data for sixty years for the United Kingdom, males and females, were available, and by using a three-factor formula, I found it was possible to get a very reasonable numerical representation of the data extending over

sixty years and sixty years of age. In this calculation there is one arbitrary constant which had to be fixed by external considerations. After experiments which took me into the realm of genetics, biochemistry, and a host of other things, I finally concluded that there was a simple hypothesis which would work, namely, that the year of birth factor was related to and could be numerically specified by the proportion of smokers in the particular cohort. The calendar year of experience factor could then be regarded as the average amount smoked by smokers in that year.

Suitable figures for giving numerical values to these ideas were available for the United Kingdom: estimates of the age distribution of smokers over the years, the proportion of smokers, and the total cigarette consumption. These tabulations led to two sets of figures for males and females, respectively, with the age factor adjusted for the proportion of smokers and the average amount smoked. The curve for the age factor increased quite nicely age by age and showed a similar trend for males and females. A relative numerical factor for the males was .005 and for females .0045. This was the first quantitative agreement that I know of for the incidence of lung cancer and smoking between males and females.

Having now found this age variation, I inquired whether or not this threw any light upon the mechanism of the mortality process. It was obvious from the run of the figures that the Gompertz formula did not represent the data, and it was also clear that the logistic formula, which I had spent much time on, was equally of no use.

I turned to random (or Markov) processes to find a suitable probability model, but I found that a simple process would not fit the data. A closer study of the figures suggested that there were two types of recognized lung cancers involved, adenomas with incidence very weakly dependent on age and epidermoid types which nowadays are considered to be linked with smoking.

Confining my attention now to the epidermoid types, I applied available Norwegian data giving an age distribution for the two types of lung cancer to the United Kingdom data for males and females previously described. This gave me a curve of "pure mortality" rates for lung cancer arising from smoking. These figures could be adequately represented by a simple backward random process, that is, one in which the organism is assumed to start with a quantity of "organization" of which a small quantity is lost in successive intervals according to a simple probability scheme. The organism is assumed to die when the total remaining "organization" falls to a certain specified value.

The numerical pattern of my studies indicates a time lag of something like ten to fifteen years between the consumption of cigarettes and the

emergence of the deaths. This fits in perfectly well with the probability model used where the transition probabilities depend upon the number of cigarettes smoked. The model fits male and female data. It fits in with the fact that, when cigarette smoking is stopped, the mortality decreases and tails off (i.e., the transition probability is made zero), and from that point on the death process ceases; there is, of course, still a residual amount of damage from previous smoking, but this is below the critical stage.

This model answers a great many of the criticisms put forward against the linkage between smoking and lung cancer. I do not say that I have proved that smoking and lung cancer are related. All I can say is: Here is a model which quantitatively and qualitatively fits with all the facts that I have seen put forward. The process requires about fifteen years before any appreciable number of deaths arise, and hence I am not at all surprised that experiments on small animals have not revealed the linkage.

MR. NOVEMBER: Since the questions are a little slow in coming, perhaps I ought to start the ball rolling. Dr. Hammond, would you care to comment on the type-of-personality factor that Dr. Hockett emphasized in his talk? Might it be the kind of person you are that makes you vulnerable to certain diseases as well as giving you a need to smoke cigarettes?

DR. HAMMOND: Well, there is the question here as to which is cause and which is effect. Is it the smoker's personality which causes him to smoke or does smoking cause a change in his personality?

I can tell you what happens experimentally. If mice are exposed to smoke, they become quite irritated. In fact, they exhibit many of the characteristics that human beings exhibit under irritation. This is an experimental situation and not a test as to whether their personality made them smoke. I think we have a cause-and-effect relationship here, probably the result of nicotine, which has tremendous effects upon the nervous system.

MR. HARRY M. SARASON: I understand that a certain American tobacco company has been using arsenic as an insect controller for many years. Has any research been conducted as to the effect of this use on the tobacco and the possibility of transference to the human body?

DR. HOCKETT: Of course, we are very well aware of the speculations that have arisen around this matter of arsenic. There are traces of arsenic in tobacco, as in all plant material, but the present levels, as determined from cigarettes taken from the open market, are at the lowest point at-

tained in the last forty years. In other words, there used to be more forty years ago than there is now. There was some increase in arsenic for a period during which arsenical insecticides were used by farmers for hornworms, but at its maximum it still amounted only to traces, and, of these traces, only about 4-6 per cent was transferred to the smoke. The use of arsenical insecticides was discouraged by the Department of Agriculture beginning some years ago, and this has resulted in the decline mentioned.

Meanwhile, we have carried out several studies. One was to see how fast arsenic disappears from the soil after it has been applied. This study assured us that the suggested possibility that arsenic might have accumulated in tobacco soils during the period when it was used is not actually a problem.

With respect to animal experiments, we have several going on even now. Lung cancer has not been produced in animals with arsenic or with smoke from tobacco that was "enriched" with very much larger quantities of arsenic than have ever actually occurred in commercial tobacco.

We do not think that arsenic plays any part in the health picture. However, in line with our general policy, we are trying to do a complete and thorough investigation to make sure. It would actually be gratifying if arsenic had turned out to be the source of the supposed health hazard, since this was easily eliminated.

MR. ALTON P. MORTON: I think possibly that the panelists and perhaps many of the actuaries present too, whose duties are not directly related to the underwriting activities of their companies, may wonder about the comparative lack of activity in this very important problem. There is, however, no lack of interest.

I would like to point out, perhaps mainly for the benefit of the panelists, the nature of the underwriting process and the difficulties which the use of smoking habits as an additional underwriting factor would present.

We obtain what information we believe we need through an application form with questions suitably chosen to develop the characteristics of the risk which we need to evaluate for mortality classification purposes. The second step is to obtain whatever we consider necessary in the way of independent verification of the information supplied. Some questions may require one form of verification; others, some other form; and, still others, perhaps none.

I would personally feel that this problem of verification would be a nearly impossible one were we to try to use the applicant's statements as to smoking habits for underwriting mortality classification.

The second point I would like to make is a very practical one for any

life insurance company: How broad should be the base on which the company is willing to issue insurance at standard or normal premium rates? Most companies include nearly 90 per cent of all the people they insure in a single standard class. These are the people who are of average physical condition and who are average in occupation, recreation, and social habits, including use of alcohol.

As I remember the figures of one of Dr. Hammond's studies, they suggest that over 80 per cent of all adults smoke. It would be quite a radical departure for a company to change the base of its standard contracts by limiting their issue to nonsmokers. To narrow the breadth of the standard class from 90 to 20 per cent or so of all insurance applicants would be a radical step indeed.

A seemingly less radical approach, which might appeal to some actuaries as a more useful alternative for experimentation, might be to use nonsmoking as a factor in determining underwriting eligibility for a "preferred risk" classification. A few companies in the past have undertaken limited experiments in superselection—a process of subdividing the standard group so that a small proportion are identified by characteristics which it is hoped are indicators of greater than average longevity. "Preferred risk" policies may be issued at lower rates or made eligible for special higher dividends from extra mortality savings. Various factors have been tried in underwriting for eligibility for preferred classification. Nonsmoking at a glance might seem to be a useful additional factor.

However, it should be noted that perhaps the most obvious single factor as a valid indicator of increased longevity is a family history demonstrating marked longevity; those people lucky enough to have chosen parents, grandparents, etc., who lived to a very high age, say, 90 or beyond. Yet as a practical matter this factor is used very sparingly as an underwriting factor for any purpose. The reason? The near-unsurmountable difficulties of verification. Smoking habits seem to me to offer almost the same practical problem.

There is no practical penalty or recourse available to the company in the face of deliberate misrepresentation by the applicant. If at some date after issue misrepresentation is uncovered, I just cannot imagine a company instituting legal action to deny a claim on the grounds that the policyholder's father (or some other ancestor) did not, in fact, die at 95—he lived to a mere 83! So with the problem of misrepresentations of smoking habits. It is doubtful if any company would be comfortable or successful in entering a legal contest based on such misrepresentations.

While we are certainly interested spectators in all the continuing research and new information that is becoming available on this most inter-

esting and important problem, it is not yet clear when or how the factor of smoking habits may play a role in insurance company underwriting practices.

MR. L. JEFFERSON STULCE: The hypothesis that higher mortality of smokers is not associated with smoking but results from intrinsically different psychological or emotional constitutions asks one to ignore vast statistical evidence and common sense. Smokers produce significantly higher mortality as long as they smoke, but this excess mortality drains off after a smoker quits smoking. Does Dr. Hockett maintain that a smoker changes his very nature when he puts down that last cigarette?

No, he says this is because former smokers also are differently constructed: nonsmokers and former smokers are more safety-conscious, while smokers are perhaps bolder or more high-strung. But statistics and sense are again devastating. Death rates for smokers are considerably higher than for nonsmokers, both in total and from many specific causes, but the death rate from *accident, violence, and suicide* is actually *lower* for these bold, reckless smokers than for "timid" nonsmokers!

A recent article in *Forbes* deplored the tobacco industry's slowness to acknowledge its moral responsibilities. It also criticized our life insurance industry for its passive attitude. Perhaps it was unfair. It is surely unfair to say that life companies are indifferent to the cigarette hazard or that we prefer to leave the burden of mortality and statistical studies to medical people, to the American Cancer Society, or to government. Yet how many have inquired about cigarette use on insurance applications or asked inspection companies to obtain such information? We inquire about alcohol use, narcotics, etc., but not about daily use of cigarettes. Thus the burden of compiling mortality statistics for smokers is indeed left for others.

The public needs to know the truth, and it will not be entirely convinced by *Reader's Digest* or even by the American Cancer Society. But it would listen to a highly respected business with no ax to grind—and no political anxieties—and with expert analyses covering millions of insured persons! This is what we might have offered the public some time ago by obtaining data (e.g., with premium notices or applications) for purely statistical purposes.

Our reputation and influence are such that unintended significance has been inferred, instead, from our inaction. Dr. Steinsrohn's widely read book, *Your Life To Enjoy*, advises readers that insurance firms do not differentiate in their premiums according to cigarette usage and hence that smokers need not be concerned about smoking if they feel well. Other

smokers similarly base hopes for normal health and longevity on the premise that insurance firms with vast mortality data do not consider smoking detrimental to health or life.

So long as this controversy continues to confuse—and results in waste of human lives—we yet can render great public service in helping to find, evaluate, and communicate to the public the statistical relationship between cigarette use and extramortality. But, if altruistic reasons are not compelling, then who has more to gain in actual dollars and cents than our own industry?

We can do more than accumulate and interpret statistics and help in their dissemination. We can begin now to make use of information already available in making more equitable assignments of premium rates. For several years the argument has been advanced that taking smoking into account in our underwriting is not really practical. Most of the reasons cited have no more validity as they apply to smoking than they do, for example, to either intoxicants, narcotics, immoral conduct, reckless driving, or social class. It is futile to quibble over whether we yet have precise measures of smokers' extramortality or have isolated the precise causal ingredient. Underwriters have never been able to enjoy the luxuries of purists.

Statistical evidence of smoking hazards was first published ten years ago. We have not yet approached this with the resourcefulness and forthrightness that characterize our efforts in other areas. Perhaps the Surgeon-General's study may ease the way for us yet to make a contribution in this vital but controversial matter.

**MR. ROBERT E. SHALEN:** Dr. Hockett, considering the present state of our knowledge about the association and the lack of knowledge about causation and also considering the changes that have taken place in cigarette manufacture, what would you say to a friend of yours about the desirability of trying to give up cigarette smoking for health reasons?

**DR. HOCKETT:** This is one of the questions that I rather expected to be asked, that is, whether the present state of knowledge does not justify the public health people in trying to bring information on tobacco use and health to the attention of the public. Our reply is that we can raise no objection to such activity. We do think, however, that public health organizations are under a strong obligation to tell the whole truth and nothing but the truth in any kind of educational campaign. Information used in educational campaigns must not be weighted. It must not create panic. It must not make an emotional appeal.

People sometimes ask me if they should give up smoking. I have to tell

them that I do not know. I do not know what smoking means to them individually and how much help or pleasure it gives them. I do not know how it would affect them to give it up, whether they would be unfit to live with or whether their families would be tempted to consider murder. I can only say that I do smoke myself, mostly a pipe. I smoke cigars now and then, and I smoke cigarettes when I do not have time for a pipe or a cigar. This smoking produces no unpleasant symptoms of any kind for me, and it gives me a great deal of satisfaction.

MR. JOHN H. TUROFF: I would like to put a question to Dr. Hammond concerning the mortality of former smokers. It is my impression that persons who stop smoking usually start to put on weight. We are interested in weight gains from an underwriting standpoint, since mortality of overweights increases with age. Our question is, therefore, whether a group of former smokers is the start of another group of overweights which will experience increased mortality from causes associated with overweight.

DR. HAMMOND: Yes, I have looked into this. The majority of people, as you say, who have given up smoking say they put on weight as a result. Now, we are faced with the following set of facts. Taking the population as a whole, overweight, as shown by your own actuarial studies, is associated with excess mortality. Cigarette smoking is associated with excess mortality, and so we have two factors.

The only question is: What is the net result of these two factors—giving up smoking and putting on weight—what is the net effect on mortality? There have now been no less than five studies on this question. All of them show that the net result is a decrease in mortality. I think it not unlikely that if the former smoker also restricted his diet there would be an even greater decrease in mortality.

DR. HOCKETT: We sponsored one of these studies ourselves. It was found that men who gave up smoking without making any other specific change in living habits gained an average of nine pounds in weight. Perhaps actuaries would find it interesting to try to calculate, from epidemiological data, the relative "risk" of the additional weight to middle-aged men as compared to the "risk," mathematically, of smoking.

The main point I would like to make about people who stop smoking, however, is this: A strong element of selection is certainly involved. Those people who can and will stop smoking are likely to be quite different constitutionally from those who cannot or will not or do not choose to do so. I am sure that this selection factor must complicate the statistical picture concerning former smokers.

