

STATEMENT OF POSITION

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(c) Is it the Smoker or the Smoking? [Smokers and non-smokers have been found to differ in many ways other than their smoking habits. Many eminent persons believe that the reported statistical association between smoking and disease may simply indicate that some of the people who smoke tend also to be people who, for various

physical or psychological or other reasons, are more susceptible to disease than those persons who choose not to smoke. Stress, for example, may well "cause" a person to smoke and also predispose such person to heart attack. Smoking, however, would not be a cause but would only be statistically associated.]

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- (d) The "Missing" Ingredient: Neither the existence nor the amount of nicotine, "tar" or any ingredient claimed to be in cigarette smoke has been proved significant to human health. [The Surgeon General's Committee concluded that nicotine "probably does not represent an important health hazard". The very term "tar" is inaccurate and misleading (since there is no "tar" in cigarette smoke), animal experimentation with "tar" has produced only erratic and questionable results (which may be compared with the generally negative findings when whole smoke inhalation experiments are done) and the problem for exploration remains, in the words of the Surgeon General's Committee, "gigantic".]

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- III. Mandatory "tar" and nicotine labeling would be misleading.

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- IV. Congress should not delegate authority to require labeling of cigarettes. [This was the position taken by Congress in 1965 and it appears amply justified in view of the Federal Trade Commission's abrupt reversal in early 1966 of its 1965 (and earlier) stand on tar and nicotine (which, according to Chairman Dixon in 1965 "could result in some other kind of a misrepresentation or something misleading").] Page

STATEMENT OF POSITION

I.

Congress, the Federal Trade Commission, the Public Health Service and the Departments of Commerce and Agriculture concluded in 1965 that the labeling of tar and nicotine yields should not be required. There has been no new scientific proof justifying any change in this position.

For many years the Federal Trade Commission, supported by the Public Health Service, consistently took the position that any statement of tar and nicotine content in cigarette labeling and advertising would not be meaningful. This viewpoint was presented to Congress during the 1965 Hearings on Cigarette Labeling and Advertising.¹ The Report of the Senate Commerce Committee took cognizance of these views:

"TAR AND NICOTINE

Several witnesses urged that cigarette manufacturers be required to state tar and nicotine yields on the package as originally provided by S.559. Nevertheless the committee is satisfied, for the reasons discussed below, that such provision should not be retained in the bill.

The Chairman of the Federal Trade Commission expressed opposition at the hearing to a statute requiring tar and nicotine labeling.

With respect to nicotine, the report of the Surgeon General's Advisory Committee states that 'there is no

acceptable evidence that prolonged exposure to nicotine creates either dangerous functional change of an objective nature or degenerative disease.' The report concludes that various studies 'indicate that the chronic toxicity of nicotine in quantities absorbed in smoking and other methods of tobacco use is very low and probably does not represent a significant health problem'.

As to tar, the Surgeon General testified before the committee that, 'While it seems at least plausible that cigarettes with lower tar and nicotine may present lesser health hazards, there is presently no proof that this is so.' He further stated that 'further study' was necessary before he could recommend that particular ingredients be singled out for labeling.*²

Since the time of that Report, there has been no new scientific proof. There still is "no proof that this is so." There is no new scientific evidence justifying any change in position or warranting tar and nicotine labeling. There has been continued, perhaps louder, repetition by the same people of the same arguments made to Congress and there rejected by the Public Health Service, the Federal Trade Commission, the Departments of Commerce and Agriculture and Congress itself.

Thus, the tar and nicotine labeling proposal presents the latest example — albeit one of the worst — of a long string of conclusions and proposals relating to smoking and health that have no adequate scientific basis.

II.

Cigarette smoking has not been established as a cause of human disease.

During the course of the 1965 Congressional Hearings on cigarette labeling, a large number of eminent doctors and scientists came forward to point out that it has not been established that cigarette smoking causes human disease. Their reasons, based upon substantial experience and research, remain as valid today as when presented. While there have been many claims of causal relationships between smoking and disease, extensive research efforts in recent years have failed to prove that smoking is a health hazard.

(a) The "evidence" is statistical and statistics cannot establish the cause of any disease.

Most of the "evidence" relied upon by those who believe that smoking causes disease is statistical.³ But, as has been shown time and again, statistics alone cannot establish the cause of any disease.*

* History records strong statistical associations between pellagra and corn consumption; cholera and high places; and malaria and night air. All of these diseases proved ultimately to be caused by third factors unknown at the time (respectively, a vitamin deficiency, a bacillus and a microbe).

While this fact was accepted by the qualified statisticians who testified at the 1965 Congressional Hearings,⁴ it has been ignored by those who have sought to use the Surgeon General's Advisory Committee Report as a basis for the assertion that smoking causes hundreds of thousands of deaths each year. The Surgeon General's Committee itself stated that "Statistical methods cannot establish proof of a causal relationship in an association",⁵ and while the Committee observed that several studies showed an "association" between smoking and death rates from nearly all diseases, it refused to accept smoking as the proven cause in most cases.⁶ Notwithstanding the Committee's recognition that statistical association does not prove causation, those who claim smoking causes so many deaths cite statistical association as "proof".

Two very significant facts appear in the principal data on death rates considered by the Committee:

(i) substantially more than 90% of the cigarette smokers whose deaths were studied died from diseases which not even the Committee thought were causally related to their smoking;⁷ and

(ii) the death rates for even the heavy smokers were lower than the death rates for the

entire U. S. population (which, of course, includes all non-smokers).⁸

These facts should be kept in mind in considering any possible relationship of smoking to particular causes of death.

(b) Zealots have vastly overstated their case against cigarette smoking.

(1) Smoking and Cardiovascular Disease.

The Surgeon General's Advisory Committee did not find sufficient evidence to conclude that smoking causes cardiovascular diseases.⁹ That lack of evidence has been blandly ignored by those zealots who include cardiovascular diseases in their claims that smoking causes hundreds of thousands of deaths.

The Surgeon General's Advisory Committee said in 1964 that the basic causes of coronary heart disease were obscure.¹⁰ The nicotine in cigarettes, a traditional whipping boy, was said by the Committee not to cause degenerative disease nor to be an important health hazard.¹¹ Those statements are as true now as they were then. Then, as now, certain factors other than smoking were thought to predispose to that disease. Stress, familial background individual personality traits (a "coronary-prone personality" has

been described), occupation, city life, obesity, diet (both general overnutrition and high fat) and lack of exercise have all been mentioned.¹² Whether smoking is one of these many factors that may be related to coronary disease remains to be determined.

Congress was told, at the 1965 Hearings, that there is a lack of experimental evidence from the laboratory to implicate smoking in cardiovascular diseases;¹³ that there is a strong possibility that such factors as stress are important in the development of those diseases;¹⁴ that there is a strong possibility that smoking is merely a reflection of a kind of person who is likely to suffer from cardiovascular diseases, whether or not he smokes.¹⁵

Research has, of course, been going forward since the Report of the Surgeon General's Committee and since the Congressional Hearings in 1965. But none of that research has produced any substantial evidence to implicate smoking as a cause of cardiovascular diseases.

(2) Smoking and Emphysema and Bronchitis.

As the Surgeon General's Advisory Committee observed, bronchitis and emphysema are the chronic broncho-pulmonary diseases of greatest public health importance in the United States.¹⁶

Nevertheless, their definition and diagnosis is admittedly inexact and the relationship between them at best confusing.¹⁷

The two diseases may coexist, either may exist before the other, or they may exist independently of each other.¹⁸ There are conflicting views as to whether either causes the other or, indeed, whether either is a necessary or even possible link in the chain of causation of the other.¹⁹ The two things about which there seem to be no question are (i) that cigarette smoking did not exist when chronic bronchitis and emphysema were first recognized,²⁰ and (ii) that both diseases occur in non-smokers.²¹

The Surgeon General's Advisory Committee correctly concluded that cigarette smoking is not established as a cause of emphysema.²² Thus, those who assume that smoking causes emphysema in order to claim that smoking is killing large segments of the population have no basis for that assumption, even in the Committee's Report.

The Committee concluded that smoking is a cause of chronic bronchitis.²³ But this conclusion was reached despite the statistical association between smoking and bronchitis seemed weaker (as near as can be determined from the meager data) than that between smoking and emphysema,²⁴ where causation was not found.

Since the most important evidence said to relate cigarette smoking to either disease was stated by the Committee to be the epidemiological (i.e., statistical) evidence,²⁵ the failure to find causation where that evidence was stronger and the finding of causation where that evidence was weaker only adds to the confusion in an already confused and ill-defined field.

Unquestionably, what is needed here is more research and less speculation. This was the substance of what the experts told Congress when they pointed out that the particular disease entities should be defined in clinically recognizable ways and their various symptoms put into meaningful categories. Until this is done, they said, it is impossible to speak of causes — or to attempt by experimental or other means to discover meaningful factors in the environment — or to determine whether or not those factors include cigarette smoking.²⁶

(3) Smoking and Lung Cancer.

The possible relationship of smoking to lung cancer has received wide attention to recent years. This is true even though the number of deaths caused by lung cancer specified as primary is but a small fraction — about 1% — of the number of deaths from all causes.²⁷ The main reason for this focus of attention on smoking

may well be that it was one of the first to be suspected among the many factors²⁸ now suspected as possible causes of lung cancer. In contrast, many other factors have long been suspected as contributing to cardiovascular diseases²⁹ and emphysema.³⁰ Whatever the reason, the fact remains that the still unanswered question of a possible relationship of cigarette smoking to lung cancer has been a subject marked by spectacular charges and widespread controversy.

Not surprisingly, this situation has led to many unfounded claims and premature conclusions. Indeed, the strongest indictment of cigarettes contained in the Surgeon General's Advisory Committee Report is that they are claimed to be the main cause of cancer of the lung.

But the evidence will not support a conviction based upon this indictment. That evidence was meticulously examined by medical and scientific experts during the course of the Congressional Hearings on Cigarette Labeling in 1964 and 1965. It was clearly insufficient to establish causation.

Most of the "evidence" is found in the statistics. And, of course, statistics cannot themselves prove cause and effect. Additionally, as the statisticians told Congress, the statistics with respect to smoking and lung cancer are replete with confusing

and contradictory findings; which have not been explained and reconciled.³¹ For example:

Many countries have lower per capita consumption of cigarettes than the United States, but higher mortality from lung cancer.³²

Despite the rapidly increasing cigarette smoking among women, the ratio of male to female deaths from lung cancer has increased four-fold in the last thirty-five years.³³

Lung cancer is more than twice as common among low-income males than among high-income males.³⁴

Benzpyrene is a compound which has experimentally induced cancer in some test animals, but which is found only in minute quantities in cigarette smoke. Pipe smoke contains more than ten times as much of this compound as cigarette smoke. Yet pipe smokers, including those who inhale, have the same death rates as non-smokers, while former pipe smokers have higher death rates than either smokers or non-smokers.³⁵ As one of the witnesses told Congress:

"In brief, if these statistics can be believed, it is quite safe to smoke a pipe, but highly dangerous to discontinue the practice." ³⁶

There are, of course, many more such curious findings in the statistical data.³⁷ But even if all these were reconciled and explained, there would still be no more than a statistical association. It would then be necessary to determine what, if anything, that association meant. In this connection, properly conducted laboratory and clinical investigations can be of great importance.³⁸

Several researchers told Congress that such evidence as exists in the field of animal experimentation is either negative or insignificant.³⁹ They pointed out that the material cited by the Surgeon General's Advisory Committee supported this conclusion. As stated by one eminent pathologist:

"To date, no one has produced cancer of the lung in an experimental animal with tobacco smoke or with condensates extracted from tobacco smoke. Thus, laboratory confirmation of the statistical association is still lacking.

"It is true that cancers have been produced on the skins of animals by various condensates of tobacco smoke, but skin cancer in experimental animals can also be produced by a number of innocuous substances, such as sugar, beef, etc. I do not think one can attach any great significance to this work."⁴⁰

In contrast to the negative results of experiments involving inhalation of tobacco smoke, lung cancers have been produced experimentally by inhalation of other suspected agents.⁴¹

So much for the laboratory evidence.

The evidence relating to humans also is open to substantial question.

One doctor showed slides and claimed to see at autopsy more changes in the lung tissue of smokers than non-smokers.⁴² These changes, he speculated, might have gone on to be cancer.⁴³ But another doctor presented the results of a nationwide investigation on the same subject conducted by 12 pathologists and concluded under the sponsorship of the United States Public Health Service. These results, he said, fell short of confirming the claims made by the first doctor.⁴⁴ Other pathologists who testified also disagreed with the claims of the first doctor;⁴⁵ and one showed slides demonstrating the same lung tissue changes in non-smokers, and even in infants. He said he could not agree that these changes were meaningful so far as cancer is concerned.⁴⁶

Additionally, Congress heard from several eminent thoracic surgeons whose wide experience encompassed well more than 10,000 cases of cancer of the lung.⁴⁷ In contrast, all seven of the

prospective statistical studies relied upon by the Surgeon General's Committee encompassed a total of only 1,833 cases of lung cancer.⁴⁸

Based on their broad clinical experience, those thoracic surgeons told Congress that they could not accept the conclusion of the Surgeon General's Committee that cigarette smoking was causally related to lung cancer.⁴⁹ They pointed out that while the concentration of tobacco smoke is greatest in the trachea, or windpipe, cancer of the trachea is a medical rarity.⁵⁰ They questioned why, in the lung itself, cancer occurs more frequently toward the periphery, where the concentration of smoke is less.⁵¹

They also pointed out that cancer of the lung differs from other cancers in that the age of peak incidence is about 57 to 60, and that this peak persists regardless of whether, how long, or how much people smoke.⁵² This human experience does not support the claim that there is a direct dose-response relationship between cigarette smoking and lung cancer, i.e., that lung cancer will occur in direct proportion to the amount of smoking. If this claim were true, long-time heavy smokers should get the disease earlier than non-smokers. They don't. Despite the apparent statistical association between smoking and lung cancer, smokers do

not get the disease any earlier than non-smokers, regardless of how long or how heavily they have smoked.⁵³ Once again, the major limitation of statistics is shown: when tested against the actual clinical data, predictions based on statistics often prove inaccurate.

Doubtless, explanations and reconciliations of these apparent conflicts in the data have been and will continue to be sought. But they cannot come from armchair speculation; rather, they must come from the result of hard, extensive and painstaking research in the laboratory and in the continued analyses of medical experience.⁵⁴ Such research has gone on continuously before and after the Surgeon General's Advisory Committee Report and since the Congressional Hearings of 1965. To date, however, it has not provided sufficient scientific evidence to determine these questions and permit reliable conclusions to be drawn. Despite the impatience of some to solve the lung cancer problem by convicting cigarettes, there is still no scientific basis for determining whether or not smoking causes lung cancer.

(4) Smoking and Cancer of the Larynx.

The only cancer other than lung to which the Surgeon

General's Advisory Committee linked cigarette smoking as cancer of the larynx in the male. Here, the link is not said to be that smoking causes laryngeal cancer but only that it is "a significant factor in the causation" of such cancer.⁵⁵ The evidence does not support even that limited conclusion.

No otolaryngologist or other specialist dealing primarily with cancer of the larynx was on the Surgeon General's Advisory Committee.⁵⁶ Several such men, of unquestioned prominence in their profession, did appear before Congress in 1965 and disagreed with the Committee's conclusion.⁵⁷

They pointed out that the Committee's conclusion was based essentially on interpretation of data showing a statistical association between cigarette smoking and cancer of the larynx. They said that other facts, known to them from their experience with the disease, indicated that any such association does not prove cause.⁵⁸

One of the most eminent of the world's otolaryngologists testified that, in the course of some 32 years of experience, he had observed over 4,000 cases of cancer of the larynx and throat and had treated over 1,800 cases by surgical means.⁵⁹ His experience encompasses twice as many cases as were included in all of the

statistical studies relied upon by the Surgeon General's Advisory Committee.⁶⁰ The combined experience of the other four otolaryngologists who testified comprised some 3,000 cases of cancer of the larynx and adjacent areas⁶¹ — more than one and a half times the total number of cases involved in the studies relied upon by the Surgeon General's Advisory Committee.⁶²

Drawing from their wide experience, these otolaryngologists told Congress that they could not accept the conclusion that smoking of cigarettes was established as a causal factor in cancer of the larynx.⁶³ They assigned many reasons for their views, including the following:

(a) There has been no increase in the incidence of cancer of the larynx to parallel the rise in cigarette consumption in recent decades. If cigarette smoking were a significant factor in the causation of laryngeal cancer, a large increase in mortality or frequency of the disease corresponding to the increase in cigarette consumption should have occurred.⁶⁴

(b) There is no experimental evidence whatever demonstrating the production of laryngeal cancer by tobacco smoke or condensates.⁶⁵

(c) The ratio of males to females with cancer of the larynx was approximately 6 to 1 thirty years ago, when comparatively few women smoked. Now, when many more women smoke, the ratio has widened to 10 to 1. This is directly opposite to what should have occurred if smoking were a significant causal factor. No explanation has been advanced for this; and there is no known sex difference in tissue response.⁶⁶

(d) Curiously, cancer occurs more frequently in women than in men in the lower throat, at the base of the tongue and on the tonsils — structures very near the larynx.⁶⁷

(e) Cancer of the trachea or windpipe is extremely rare even though this structure, immediately below the larynx, is also directly in the path of inhaled smoke and should be similarly affected.⁶⁸

(f) Statistical studies of laryngeal cancer seldom define precisely the location of the cancers included in the study. The location is important because the clinical behavior of cancers at different sites in and near the larynx differs greatly. And,

as would be expected, the degree of association may vary greatly depending upon the types of cases which are included or excluded. Present knowledge does not provide explanations for these behavioral differences among cancers originating in virtually adjacent locations.⁶⁹

(g) There are unexplained geographical puzzles. Cancer of the upper throat — nasopharynx — is more than ten times as common in China or Formosa than elsewhere in the world. Cancer in the lower throat — hypopharynx — is more common in Great Britain, France and the Scandinavian countries than in the United States.⁷⁰

(h) Other factors, such as alcohol and malnutrition, are suspects in cancer in this area. At present, there is no way of knowing which, if any, of these factors are of causal significance.⁷¹

To date, the data necessary to resolve these questions and the many other unanswered ones in this field have not yet appeared. At least until such data are available, the hasty jumping to conclusions should be avoided. It is obviously premature at this time to come to any conclusion as to what role cigarette

smoking may or may not play in the causation of cancer of the larynx.

(c) Is it the Smoker or the Smoking?

If, as several studies have indicated, there is a statistical association between the smoking of cigarettes and a wide variety of diseases, then the need for scientific inquiry has not ended but has only begun. This is particularly so because laboratory and clinical findings to date have failed to support suggestions that the reported statistical association should be interpreted as showing that smoking causes some or all of these diseases.

Several prominent statisticians told Congress in 1965 that the reported statistical association between smoking and various diseases — wholly apart from all the inconsistencies and question marks — could well mean that many of the people who are going to get these diseases are the kind of people who also are going to smoke.⁷²

Thus, the reported statistical association, far from demonstrating that smoking is a health hazard, may reflect significant differences in the physical, constitutional, psychological and

genetic makeup of many smokers and non-smokers. These differences, not caused by smoking, may account for the observed association.

As the statisticians told Congress, this interpretation of the association has never been ruled out, and is at least as likely an explanation as one that assigns causal roles to smoking. The analysis begins with the statistics. For example, if the statistics had shown that smokers and non-smokers had the same history with respect to all diseases, except that smokers had a very much higher incidence of one particular disease, then it would have been appropriate to focus investigation upon the question of whether smoking caused that disease.

Such, however, is not the case. Rather, the statistics indicate that smokers and non-smokers differ with respect to their overall disease history and not just with respect to one disease. The statistics, of course, deal only with people who have been included in surveys and not with the whole U. S. population. They also deal with human beings whose decisions to smoke or not may well reflect different personal characteristics. This raises the question (assuming that the reported statistical association is valid for the entire population) of whether the observed difference in disease history came about because of smoking or because

of differing personal characteristics of smokers and non-smokers. In this latter event, smoking would be just one of the differences between the two groups and not the cause of the difference in disease history.⁷⁵

A threshold question, then, is whether there are significant differences between smokers and non-smokers, in addition to differences in smoking habits and disease history. This is a broad and wide-ranging question, requiring for its answer broad and wide-ranging research into a myriad of human characteristics.

According to the information presently available, it does appear that smokers as a class are in many ways different from non-smokers. For example, cigarette smokers as a class marry and change jobs more often, are more athletic, are more often hospitalized, drink more alcohol and black coffee, are more often neurotic, are more likely to have parents with heart disease or hypertension, and have shorter lived parents and grandparents.⁷⁶ So there is evidence that smokers are often of a different personality type from non-smokers. And, interestingly, many of the characteristics of this personality type have also been related to stress⁷⁷ which is believed by many to play an important role in the production of coronary heart disease and other ailments.⁷⁸

There is strong support for the interpretation of the statistics that smoking is a reflection of a type of person who, whether or not he smokes, is more likely to get certain diseases, and is not itself a cause of those diseases.

- (d) The "Missing" Ingredient: Neither the existence nor the amount of nicotine, "tar" or any ingredient claimed to be in cigarette smoke has been proved significant to human health.

For several years — indeed, since cigarette smoke became a suspect — intensive laboratory experimentation and analysis have been directed to the identification and isolation of the ingredient or ingredients in cigarette smoke which could cause the diseases for which smoking is claimed to be responsible. During this period of years, advances in scientific techniques have permitted the identification in given substances of fantastically minute quantities of particular ingredients. Not surprisingly, therefore, from time to time there are reports of the identification of some ingredient in cigarette smoke coupled with claims that it may have some possible connection with disease causation. To this date, however, all of the advanced technology and all of the intensive work have added up to one clear result:

There has been a complete failure to identify any ingredient or group of ingredients in cigarette smoke that is specifically responsible for causing any of the human diseases with which smoking may be statistically associated.⁷⁹

Nicotine

The presence of nicotine in tobacco has given rise to much fruitless speculation over its possible role in causing human disease. Such speculation all too often follows a demagogic line, beginning with the assertion that pure nicotine is a poison and ending with the conclusion that in cigarettes it is very harmful indeed.

In the face of this line of "reasoning", a broad consensus has been achieved in the scientific world with respect to nicotine. In contrast to the controversy over the question of cigarette smoking and health, there is practically no controversy over nicotine. This consensus was well expressed in the Surgeon General's Advisory Committee Report:

"There is no acceptable evidence that prolonged exposure to nicotine creates either dangerous functional change of an objective nature or degenerative disease."⁸⁰

"... the chronic toxicity of nicotine in quantities absorbed from smoking and other methods of tobacco use is very low and probably does not represent an important health hazard."⁸¹

The more important reasons for this conclusion are as follows:

(a) It has never been seriously suggested that nicotine has any cancer causing properties.⁸²

(b) Nicotine has no known chronic or cumulative effects. It is rapidly absorbed and rapidly changed by the human body into other simpler substances which have low pharmacological activity and are speedily excreted. There is no evidence that any of these substances, into which nicotine is rapidly changed, has any toxic effects.⁸³

(c) The chronic toxicity of small doses of nicotine is low in experimental animals,⁸⁴ and the quantities of nicotine obtained by humans in ordinary forms of tobacco use is very low.⁸⁵

(d) Pipe and cigar smoke, in general, contain considerably more nicotine than does cigarette smoke.⁸⁶ Several studies suggest that, regardless of inhalation habits, pipe and cigar smokers absorb amounts of nicotine at least

comparable to the amounts absorbed by cigarette smokers. If they do inhale, they must often absorb even greater quantities. But smokers of pipes and cigars do not show significantly higher mortality than non-smokers.⁸⁷

These generally accepted conclusions render any requirement of a statement as to nicotine content on cigarette packages pointless at best. At worst, they render any such requirement misleading. Stripped to its essentials, the argument favoring nicotine content labeling is that a smoker will be able to compare brands with respect to relative nicotine content and to choose the one having less nicotine. The suggestion is that less nicotine is somehow "safer"; and this is the message that unavoidably will be conveyed to the smoker by any such required labeling.*

But there is no acceptable evidence that the amounts of nicotine absorbed by the smoker are harmful. To the contrary, the consensus is that these amounts are not harmful. Therefore, there is no scientific basis for assuring the smoker that cigarettes with less nicotine are somehow "safer".

* The suggestion that less nicotine is "safer" is not avoided by the warning now on cigarette packages. The warning would only remind the smoker that the lower nicotine cigarette is not necessarily completely safe. Obviously, the warning would not stop him from concluding that it is "safer".

Tar

There is, of course, no "tar" as such in cigarette smoke.⁸⁸ "Tar" refers to condensate collected from smoke by laboratory methods.

The tar question was raised when, some years ago, it was reported that smoke condensate painted on the skins of a susceptible strain of mice produced skin cancers on some of those mice.⁸⁹ Those experiments, of course, did not prove that cigarette smoking was a cause of cancer in humans. It is universally acknowledged that one cannot automatically translate into human terms and conclusions the results of animal experimentation, and that is particularly true in the case of mouse skin painting with tobacco smoke condensate.⁹⁰ The concentration of so-called "tar" painted on the skins of mice was incredible, estimated by some scientists to be equivalent to human smoking of more than 100,000 cigarettes per day.⁹¹

In brief, the experiments involving the painting of mouse skin with tobacco smoke condensates were subject to the same problems as that type of experimentation generally. Even in the same animal, different tissues respond differently to a given substance, which may cause cancer in one tissue and be harmless to another.⁹²

Different species of animals respond differently to a given substance. A substance which may cause cancer in one species may not in another.⁹³ Indeed, different strains or variations of the same animal may respond differently to the same substance.⁹⁴ And, of course, experiments have shown that skin cancer can be produced in animals by a number of common substances, such as sugar and beef.⁹⁵

The lack of correlation between the almost infinitesimal amounts of suspected ingredients found to be in cigarette smoke condensates and some of the experimental results led the Surgeon General's Advisory Committee to describe the situation as presenting a "puzzling anomaly".⁹⁶ The Committee referred to the possibilities of interactions of ingredients as a "gigantic area for exploration".⁹⁷ Today, despite continued work, no one has solved the puzzle; and the area remaining for exploration is virtually as "gigantic" as ever.

This, of course, raises the question of validity of the causal hypothesis in which skin painting experiments are considered as possibly relevant. This theory is that cigarette smoke contains an ingredient or ingredients capable of causing cancer. If so, this should be demonstrable in animals. Then, in humans, sufficiently prolonged exposure to enough of the smoke containing the cancer

causing ingredient should, if the animal results are right, ultimately result in the production of lung cancer.

And, indeed, some earlier experimental results seemed to lend some support and credence to this theory. Smoke condensates did cause skin cancer in some mice.⁹⁸ Additionally, condensates were found by extremely sensitive chemical procedures to contain minute quantities of some compounds which had been found to be experimentally capable of producing cancer in animals.⁹⁹ And, some studies showed that these very compounds were being deposited in the central portion of smokers' lungs, where lung cancer was thought to originate.¹⁰⁰

At this point, the theory began to fall apart. Studies showed that lung cancers did not originate where these compounds were being deposited, but rather originated further out in the lung, where there was not any relatively large concentration of cigarette smoke.¹⁰¹ Further skin painting experiments and similar techniques began to produce equivocal and inconsistent results.¹⁰² The "puzzling anomalies" and "gigantic areas for exploration" began to appear in the experimental work. Further, scientists began to explore experimentally whether human-type cancer could be induced in animals by prolonged inhalation of cigarette smoke.¹⁰³ This

would appear to be somewhat more pertinent, since humans do inhale cigarette smoke and do not paint their skin with condensate. The results of such work, however, added up to what one investigator described as "a striking negative result".¹⁰⁴

The experimental work, therefore, failed to support the hypothesis of a simple, direct contact causation of cancer by cigarette smoke. Not only were the dosages necessary to produce cancer of the skin in animals of incredible concentration, so far as any human equivalent could be considered, but also they did not correlate with the dose-response characteristics thought to be indicated by statistical studies of human lung cancer.¹⁰⁵ Thus, many investigators abandoned the direct contact causation theory and turned to examination of other possible theories of cigarette smoke activity.

At present, about all that remains of the direct causation hypothesis is the assertion that, if the amount of cigarette smoke condensate is reduced, the smoker's chance of getting cancer of the lung may correspondingly be reduced. This suggestion has outlived the scientific theory on which it was based.

When the Surgeon General's Advisory Committee Report was released, the members of that Committee — and, indeed, the Surgeon

General himself -- recognized the lack of scientific basis for this direct causation theory. They therefore refused to accept suggestions that cigarettes could be said to be "safer" if there were lesser amounts of condensate extractable from the smoke. The Surgeon General was joined by the Chairman of the Federal Trade Commission, among others, in presenting this viewpoint to Congress in 1965.¹⁰⁷

Since the investigation of other possible modes of causal activity of cigarette smoke has proven fruitless to date, the sense of frustration with which the Surgeon General, the Chairman of the Federal Trade Commission and others must now approach this problem is readily understandable. Their belief that cigarette smoking was "guilty as charged" doubtless gave rise to expectation that the proof would shortly be forthcoming to show how this was so. That such proof does not today exist may well explain their frustration. It does not, however, excuse resurrection of the long since discredited assertion that reduced tar means added safety, when there is not a trace of new proof to support either the assertion or the rejected hypothesis upon which it was originally based.

Nor can this resurrection be justified by the thought that measures leading to reduction of tar will certainly do no harm.

even if they do no good. An example of the danger of this fallacy was provided some years ago by a doctor who has long been noted for his anti-cigarette fervor. He observed that there were some substances coating the tobacco leaf which, upon burning, produced some suspect ingredients. These, he said, could be in large part removed by subjecting the tobacco leaf to a particular chemical "bath". He suggested that if this were done it would make cigarette smoking "safer". It was even suggested that this should be done without waiting for proof that the substances were in fact harmful, because their removal could do no harm. Of course, if causation were later established, the "bath's" value would thereby also be established.¹⁰⁸

This superficially attractive argument — markedly similar to the tar labeling argument advanced today — was eventually demolished by its own author. He subsequently reported that the very portion of the tobacco leaf that would be removed by the bath contained so-called anticarcinogens, i.e., substances which inhibit cancer-causing activity in animals.

The point is that present scientific knowledge is not sufficient to permit action aimed at "tar" reduction to be taken with assurance that it is scientifically accurate and valid. Nor, even

if the value of a reduction were assumed, would it be possible to say how much of a reduction would be meaningful. There is no scientific method to determine whether a difference between "tar" content of any two brands would be meaningful — or how much of a difference might be significant.

Analyses of cigarette smoke condensates have shown that their chemical compositions can and do vary widely. Among the ingredients chemically identified as present in varying minute amounts are some which are said to inhibit cancer formation, some which are said on the basis of animal experiments to cause cancer, some which are said to promote cancer formation and some which are said to permit cancer formation by other substances.¹⁰⁹ No one knows how these interact with each other and with other factors in the human environment. No one knows whether they in fact harm human beings. No experiments have demonstrated the mechanism by which cancer develops. Yet, without this vital information, it is just not possible to determine whether these substances harm humans or, if they do, whether they do so in the form and quantity found in cigarette smoke.

"Tar" or condensate is derived in a laboratory by a method that is totally unlike human smoking.¹¹⁰ It is condensed in a chemical

solution at extremely low temperatures. Measurements vary depending on the exact method used, number and frequency of the times the cigarette is puffed in the smoking machine, how short the cigarette is smoked and other factors. This emphasizes a fundamental point which is often lost sight of: People inhale smoke. They do not inhale "tar" or condensate. Neither do they apply "tar" or condensate to their skin. There is no reason to suppose that any biological activity of whole smoke can be accurately assayed by studying "tar" or condensate (extracted from smoke in laboratories) or by studying anything but the smoke itself in the form and substance it is smoked by people. To the contrary, there are good reasons to suggest that the chemical and physical changes necessarily brought about in condensing the smoke and applying it to animals may well produce biological results completely different from any that may occur in smoke inhalation.*

The evidence thus far accumulated indicates that this is so. In contrast to even the limited activity shown by condensate

* One example of the difficulties here: Condensate is applied to mouse skin in acetone solution. Benzpyrene, a suspected ingredient found in minute quantities in smoke condensate, is capable of inducing cancer on mouse skin, as shown by experiments where relatively large doses of pure benzpyrene were applied in acetone. When injected in a water solution, benzpyrene does not produce tumors. There is no acetone in cigarette smoke; the main substance in the smoke is water.

experimentation, experiments involving inhalation of tobacco smoke have failed to produce any human-type cancers at all. This work¹¹¹ has failed to reveal any correlation with the results obtained by studies of condensates.

Obviously, studies involving inhalation of smoke by experimental animals are closer to human experience than skin painting. However, it is no easy task to develop techniques for inhalation experiments which will reasonably approximate human smoking, both in method and in the amount of smoke taken into the respiratory system of the test animals. Techniques and methodology are being developed and refined, but the complexities of dealing with a volatile and changeable substance such as smoke and of producing a situation in experimental animals comparable to human smoking requires much careful planning and extensive trial testing.¹¹²

This has been developing for several years and hopefully will ultimately result in a large-scale program of inhalation studies. Such a program is presently being considered by the Council for Tobacco Research-U.S.A. Such a program would be aimed at developing human-type diseases such as cancer of the lung, various cardiovascular diseases and emphysema in test animals. There may then be studies to determine the biological mechanisms by which

the diseases are brought about in the test animals and whether any factor or combination of factors involved in inhalation brings about these human-type diseases in the animals.

By the development of such experiments it may be possible to determine what scientific knowledge the laboratory can contribute to the relationship, if any, of cigarette smoking to these diseases.*

The need to replace speculation with solid scientific evidence has been amply demonstrated in this area. The past several years have seen a succession of unfounded claims that a disease causing ingredient has been discovered in tobacco smoke. At first, proponents of benzpyrene as the culprit vied with proponents of cigarette paper.¹¹³ Enthusiasm for these waned, however, as laboratory data failed to support the claims. For a time, arsenic¹¹⁴ attracted enthusiastic supporters. This was followed in turn by

* One recent report demonstrates dramatically the need to proceed scientifically and to avoid impatient jumping to conclusions. Dogs were subjected to heavy continuous doses of cigarette smoke not by inhalation in any ordinary fashion, but rather by surgical operation to permit introduction of cigarette smoke in concentrated quantities forced directly into the dogs' windpipes under pressure. Some dogs, not surprisingly, died promptly. Others, after a year of such abuse, in some instances developed pathologic changes which, it was said, looked something like human emphysema. Of course, the conditions under which the smoke was projected into the lungs of the dogs scarcely resembled any human form of smoking; and it is hard to give any credence to the "results".¹¹⁵

nickel.¹¹⁶ Then phenols.¹¹⁷ And then polonium.¹¹⁸ And there have been many others. As theory has been replaced by evidence, however, most of these have been ruled out — just as nicotine was ruled out by the Surgeon General's Advisory Committee.

Indeed, there has been a growing realization of the complexity of the problems here involved and a growing awareness that the answers would not come quickly and simply. It is, therefore, particularly distressing to see the current efforts, born of frustration and impatience, to claim a quick and ready solution to the problem by labeling tar and nicotine as the culprits and thus to ignore the present lack of scientific knowledge necessary for a real solution.

III.

Mandatory "tar" and nicotine labeling would be misleading.

Congress, in the Cigarette Labeling and Advertising Act of 1965, required a warning label on cigarette packages but rejected all proposals for the labeling of tar and nicotine or other ingredients. In so doing, Congress was reflecting the views expressed by, among others, the Federal Trade Commission and the

Public Health Service. Both had supported the proposed legislation; and both had made plain that scientific evidence did not exist to justify labeling cigarette packages with tar and nicotine content. Their position was based on the uncontroverted facts that there was (i) no proof that cigarettes with lower tar and nicotine content were "safer", (ii) no proof of any tar and nicotine level above which there was hazard and below which there was "safety" and (iii) no evidence on which to base a determination that any difference in tar or nicotine content between two cigarettes was or was not significant.¹¹⁹ These facts remain as true today as they were then.

The Chairman of the Federal Trade Commission, in his 1965 testimony before the Senate Commerce Committee endorsed the Surgeon General's statement that tar and nicotine labeling proposals should be rejected. He said:

"Basically this study has never arrived at what is a safe tar and nicotine content, and they have not arrived at what in smoking is the agent as such that is causing cancer. . . . They don't know whether to blame it on nicotine, tar, or many other defined and undefined hydrocarbons and chemicals that take place, chemical reactions that take place when tobacco burns." 120

As he told the House Commerce Committee:

". . . there was no certainty as to which if any of these substances could cause or even may cause

cancer and . . . there was no established moderation, you might say, of what would be a safe number of cigarettes for one to smoke, or level of content of any substance in any cigarette." 121

The Chairman left no doubt as to the unfortunate consequences which could result from tar and nicotine labeling:

"It could result in some other kind of a misrepresentation or something misleading if one cigarette came out and said it had 1.5 in tar, and so much nicotine in it, and another came out and said it had only 1." 122

The Surgeon General confirmed the lack of scientific evidence. He told the Senate Commerce Committee:

"While it seems at least plausible that cigarettes with lower tar and nicotine may present lesser health hazards, there is presently no proof that this is so." 123

Congress rejected suggestions that it require tar and nicotine labeling. It did, however, require that every package of cigarettes carry the warning:

**"Caution: Cigarette Smoking May
Be Hazardous To Your Health."**

Today, therefore, every cigarette smoker is reminded on every package of possible hazards of cigarette smoking. Now, however, the Federal Trade Commission says that this is not enough and that cigarette packages should be required to carry statements of tar and nicotine content.

The Commission's change in position was announced in March 1966. The only reason given was that the information "may be material and desired by the consuming public." ¹²⁴ No new scientific evidence or proof was described, nor was the slightest suggestion made that any had come into existence since the Chairman of the Commission had appeared before Congress and taken a contrary view.

Following announcement of the new position of the Federal Trade Commission the Public Health Service held a one-day meeting of a small group of investigators and others in June 1966. No new evidence was announced following that meeting. The group did not demonstrate that tar or any specific ingredient was harmful, nor did it venture to explain how the Surgeon General's Advisory Committee was wrong in exonerating nicotine. Instead, the group merely stated that the "preponderance" of existing evidence "strongly suggests" that the lower the tar and nicotine in cigarette smoke, the less harmful the effects.

The bald pronouncement of this group has thus transformed "no proof" into "strong suggestion". Of course, the Federal Trade Commission did not have even this group's statement to support it when it summarily changed position. If this pronouncement should

satisfy the Surgeon General that he is now warranted in giving at least tacit support to the Commission, it certainly should not satisfy Congress that it is warranted in changing its prior determination. This is particularly true because that determination was and still is solidly based on the state of scientific knowledge and not on the mere fiat of a small group.

Additionally, if it were assumed that smoking causes disease, any tar and nicotine labeling requirement might be not only misleading but dangerously so. For example:

(i) A smoker would assume — as Congress is asked to determine as an established fact in order to require labeling — that lower "tar" and nicotine content means "safer" cigarettes. But, as the Report of the Surgeon General's Advisory Committee pointed out, the particulate phase which contains the "tar" and nicotine accounts for only 40% of cigarette smoke, the other 60% being gaseous phase.¹²⁵ That other 60% is not necessarily reduced with "tar" and nicotine reduction. Yet, for all that is now known, it may well contain "harmful" ingredients. Accordingly, a smoker could be lulled into a false sense of added safety by

labeling which indicates reduced tar and nicotine content, when "harmful" ingredients have not been reduced at all.

(ii) A smoker may compare two packages of cigarettes and choose one on the ground that its tar and nicotine content is less than the other. Presumably, he would be doing so because of his belief that reduced tar and nicotine content makes the cigarette "safer". Here again, he is receiving a possibly false assurance of safety, because there is no way of knowing whether the difference between the two cigarettes is at all significant. Even if one were to assume that reduced tar and nicotine content made cigarettes safer, no one knows how much reduction would be meaningful.

In both examples, the result may well be to persuade a smoker either to continue a given level of smoking or to increase that level of smoking because the cigarettes that he has chosen are somehow "safer". And he would have an Act of Congress to back him up.

But Congress should not take the responsibility of "backing him up" without solid scientific proof to support doing

so. The smoker already is warned of potential hazards of smoking. Therefore, there is no need for Congress to take the risk, necessarily inherent in a tar and nicotine content labeling requirement, of misleading the smoker.

It has been said that there is no risk of misleading and perhaps endangering the smoker by requiring tar and nicotine labeling, because of the warning of potential hazard. True, the smoker may be reminded that the cigarette is not absolutely safe. But he may well conclude that the cigarette is substantially safer. Indeed, that conclusion is virtually inescapable, since the very reason for the proposed labeling of tar and nicotine content is to encourage reduction of that content, thereby purportedly making the cigarette "safer". Otherwise, there is no point to the requirement. Having concluded that the cigarette is safer, the smoker may well be less restrained in his smoking habits than he would be if all he had before him was the warning on the package of potential hazard. Thus, the risk of misleading and perhaps endangering the smoker is not avoided by the warning.

IV.

Congress should not delegate authority to require labeling of cigarettes.

The circumstances of the present proposal for tar and

nicotine content labeling demonstrate the wisdom of Congress' 1965 determination (in the Cigarette Labeling and Advertising Act) to retain control in this area. At that time, there were some who urged Congress either to require tar and nicotine content labeling or to delegate power to act in that area to the Public Health Service, the Federal Trade Commission or some other agency.¹²⁶ But the hearings revealed that there was no scientifically valid proof which would warrant a requirement of tar and nicotine content labeling.¹²⁷ And, in large part because the state of scientific knowledge did not permit establishment of intelligent guidelines for delegation, Congress chose instead to require reports to it at the end of specified periods on pertinent matters. Thus, Congress assured that, should the situation change so as to require further action, it would be fully informed so that it could take whatever action was necessary in the public interest.¹²⁸

At the hearings, the Federal Trade Commission had opposed tar and nicotine labeling. The Chairman had urged that "it would be better to leave the situation alone."¹²⁹ He pointed out that labeling "could result in some other kind of a misrepresentation or something misleading."¹³⁰ This, of course, was entirely consistent with the position theretofore taken by the Commission, which had regarded

a statement of tar or nicotine content, not substantiated as significant in health terms, as an unfair or deceptive trade practice.

The views expressed by the Commission to Congress were supported in substance by the Departments of Commerce¹³¹ and Agriculture,¹³² and the lack of scientific knowledge was confirmed by the Surgeon General.¹³³ Thus, the basis of the Congressional decision in 1965 is clear.

What is not clear, however, is the basis on which the Commission has sought to circumvent that decision. In March 1966, the Commission suddenly announced that tar and nicotine labeling would be permitted, giving as its reason only that the information "may be material and desired by the consuming public".¹³⁴ There had been no new scientific proof to justify the Commission's change in position. Completely lacking was any showing of health significance or of any way in which any differences in tar or nicotine content could be shown to have health significance. The sole "scientific" support that the Commission later used was a batch of opinion letters, merely repeating the same points that had been made to Congress and relying upon the same inadequate information.¹³⁵ Indeed, the letters were written by the same people and presented the

same viewpoint that had been rejected by Congress in 1965.

Nor was the Public Health Service able to support the Commission. When, in June 1966, it asked a small group to consider the matter at a one-day session, the most the group was able to say was that the evidence "strongly suggests" that a reduction in tar and nicotine would lead to less harmful effects. Again, no new evidence was forthcoming; there was only repetition of the same inadequate data.

But inadequate data, or unsupported opinions, or the louder repetition of those opinions, do not demonstrate health significance. What has not been demonstrated is any way of answering the critical questions: Do "tar" and nicotine have any effect on health at all? If so, how much of a tar or nicotine reduction has health significance? If so, how much lower in tar or nicotine content must one cigarette be than another to have "less harmful effects"?

The answers to these questions should be based on solid scientific evidence. To the contrary, the apparently overwhelming desire to "do something", based upon the lofty motive of "doing good" has been successfully urged upon the Federal Trade Commission in lieu of that evidence.

A lofty motive will in no way justify misleading the consumer. Conspicuous by its absence is any explanation from the Commission as to how tar and nicotine labeling is "misleading" and "unfair and deceptive" in mid-1965 but "material and desired" in early 1966.

Until scientific evidence establishes whether smoking in fact is harmful and, if so, what ingredient or ingredients are responsible for the harmful effects, Congress should continue to retain control under the Cigarette Labeling and Advertising Act of 1965. There is no factual basis upon which it can predicate a sensible delegation. Furthermore, the susceptibility of potential delegates to the exhortation to "do something", even though there is no basis for meaningful action, demonstrates the continued wisdom of Congress' determination to keep control. Congress can best protect the public interest by taking no further action until it has sound scientific evidence to assure that the action it takes will inform and not mislead.

STATEMENT OF POSITION
(Bibliography)

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KEY

Herein the Hearings on Cigarette Labeling and Advertising Before the Senate Committee on Commerce, 89th Cong., 1st Sess. (1965) will be designated the "1965 Senate Hearings"; S. Rep. No. 195, 89th Cong., 1st Sess. (1965) will be designated the "Senate Report"; the Hearings on Cigarette Labeling and Advertising Before the House Committee on Interstate and Foreign Commerce, 89th Cong., 1st Sess. (1965) will be designated the "1965 House Hearings"; the Hearings on Cigarette Labeling and Advertising Before the House Committee on Interstate and Foreign Commerce, 88th Cong., 2d Sess. (1964) will be designated the "1964 House Hearings"; the Hearings on False and Misleading Advertising (Filter-Tip Cigarettes) Before a Subcommittee of the House Committee on Government Operations will be designated the "1957 House Hearings"; the Report of the Advisory Committee to the Surgeon General of the Public Health Service on Smoking and Health, Public Health Service Publication No. 1103 will be designated "Smoking and Health."

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	6	Smoking and Health 37-9
	7	Smoking and Health 102 (Table 19). Of the 24 diseases specifically listed therein, the Surgeon General's Advisory Committee found causation in only three instances: lung cancer, chronic bronchitis laryngeal cancer.

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