LUNG CANCER, CORONARY HEART DISEASE AND SMOKING

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Statement of Professor Hans J. Eysenck

I am Hans J. Eysenck, professor of psychology at the Institute of Psychiatry, University of London and psychologist to the Maudsley and Bethlem Royal hospitals in London.

I received my Ph.D. in 1940 and my D.Sc. in 1964, both from the University of London. I was Senior Research Psychologist at Mill Hill Emergency Hospital from 1942 through 1946. In 1949 and 1950 I was a visiting professor at the University of Pennsylvania in Philadelphia. Between 1950 and 1954, I was a Reader in Psychology at the University of London's Institute of Psychiatry. In 1954 I was a visiting professor at the University of California at Berkeley.

I am a Fellow of both the British Psychological Society and of the American Psychological Association.

I have founded and edited three psychological journals, and I am on the editorial boards of some 15 other international psychological journals. I have written or edited for publication approximately 35 technical books and over 600 articles dealing with various aspects of the psychological field, particularly with respect to personality, intelligence,
behaviour therapy and behavioural genetics. I have conducted research in the area of smoking for over 20 years and have authored two books, the most recent of which is entitled *The Causes and Effects of Smoking*, as well as numerous articles on this subject.

A widely accepted theory asserts that cigarette smoking causes lung cancer, coronary heart disease, and many other diseases with which it is statistically linked. It is not always realized that (a) such a theory is far from proven, and is beset by many anomalies and doubts, and that (b) there is an alternative theory which is based on undeniable facts which are not explained by the causal theory. The present position seems to be that either theory may explain the tragic incidence of lung cancer and coronary heart disease (to which this brief account will be restricted), or that both may be needed to complement each other.

There is agreement that smoking is neither a necessary nor a sufficient cause of lung cancer. Of 100 heavy smokers, less than 10 will develop lung cancer; hence smoking is not a sufficient cause. And of 100 people who develop lung cancer, approximately 10 will be non-smokers: hence smoking is not a necessary cause. This simple fact (the precise numbers differ of course from country to country, but indicate
the correct order of magnitude) suggests that the scientific proof for any particular theory will be difficult to arrive at, and that any such theory will almost certainly be complex and multi-faceted.

Much of the evidence cited in favour of the causal theory is statistical, but many statisticians have severely criticized the evidence on statistical grounds. Such suggested proofs as the correlation between smoking and lung cancer within a given country, or between lung cancer and number of cigarettes smoked between countries, are evidence of correlation, not of causation; one of the first lessons the budding statistician learns is that correlation does not imply causation. (There is a very high correlation between countries linking meat eating and cancer of the large intestine, yet we do not conclude that eating meat causes cancer of the large intestine!). Hence this method of demonstration, while suggestive, is far from compelling. This would be so even if the figures usually quoted could be taken seriously; however, there are good reasons for doubting their accuracy.

The figures quoted are based on clinical diagnosis of lung cancer, but these are very unreliable and imprecise. If we take as our criterion autopsy data, and compare these
with routine diagnosis, we find that prior to World War I, out of 100 people found on autopsy to have died of lung cancer, only 3 were so diagnosed. This is typical of the very obvious under-diagnosis of lung cancer then prevalent. In recent years, exactly the opposite has been found, namely an over-diagnosis of lung cancer of up to 200% and more!

Whether these changes in diagnostic preference are completely responsible for the alleged tremendous increase in lung cancer over the years or not, and whether it may in part account for the observed correlation between lung cancer and smoking, it is impossible to say; all we can say is that with the basic data so completely unreliable, the statistics based on them are suspect.

Another important point concerns the isolation of smoking from other, correlated habits, such as drinking, living it up, staying out late, wenching, etc., i.e. a certain style of life the totality of which may increase the "rate of living", so that smokers are biologically older than non-smokers at a given age, for reasons only partly involved with smoking. Non-smokers are different types of persons from smokers, are generally more self-protective, and the personality traits and habits thus linked with non-smoking may be more relevant to the longevity of non-smokers than their refusal to smoke.
It is often suggested that sex differences, with males showing more lung cancer, are the product of the tendency of males in the past 50 years or so to smoke more. However, as several authorities whom I quote in my book have pointed out, similar sex ratios to those observed now were found before cigarette smoking became popular. Again, it is found that changes in the rate of increase of lung cancer diagnosis occurred simultaneously for men and women, although the women, who took up smoking much later than men, should have shown these changes at a much later date than men.

If the causal theory is true, then we would expect a definite dose-response relationship; in other words, the heavy smoker should be stricken with cancer earlier than the light smoker. Yet the amount smoked makes no appreciable difference to the mean age at which the person is reported first to the clinic. Again, inhalation should make lung cancer much more likely than smoking without inhaling, yet the figures show if anything an opposite trend. These two observations are difficult to reconcile with the causal theory of smoking.

The most impressive evidence for the causal theory has been the report that physicians who gave up smoking showed less lung cancer than members of the general public who
continued to smoke. Thus, it might appear that giving up smoking has saved the lives of those who did so. But this proof is only acceptable if those who continue to smoke, and those who later on give up smoking, are essentially identical with respect to their health before some of them gave up smoking. Clearly, if those who later on give up smoking are already much healthier than those who later on continue to smoke, then the final differences in health may be due to the already existing differences before anyone gave up smoking, rather than to the cessation of this habit! But there is good evidence to show that smokers and ex-smokers already differed with respect to their health record before the ex-smokers gave up smoking. Similarly, there is evidence that from the point of view of personality ex-smokers are different from continuing smokers. Thus this alleged proof is based on an erroneous assumption.

These objections to the causal theory, and others made in my book, do not prove the theory to be wrong; they simply argue that it is still only a theory, not a scientific law. More convincing proof is required before the theory can be accorded a more advanced status. But further than that, there are numerous facts suggesting an alternative theory, and these facts cannot easily be integrated with the causal theory. Yet a proper theory demands that attention be paid to all relevant facts, and thus again the causal theory is found wanting.
The alternative theory, first suggested by the eminent geneticist and statistician Sir Ronald Fisher, suggests that genetic factors are important in causing lung cancer; that genetic factors are active in causing people to maintain the smoking habit; and that possibly the same genetic factors may be involved in both these trends, thus producing the observed correlation between smoking and cancer (insofar as such a correlation is real). There is evidence that genetic factors do play a part in the causation of lung cancer; this is not in doubt. I have brought forward evidence (in addition to already very convincing evidence produced by many other people) to show that genetic factors are relevant to the maintenance of the smoking habit. Thus there is evidence for both the assumptions on which Fisher's argument was based.

The origin of the smoking habit, on the other hand, is hardly at all influenced by genetic factors. It appears from our genetic analysis and from the direct study of the problem by Professor Spielberger that the origin of the smoking habit is due to peer pressure; parental influences play a much smaller part, and advertising almost none.

My own contribution has been to suggest that the mediating factor between cancer and smoking may be the personality of the people involved. Thus it is assumed that people of
a certain personality are more likely than others to die of lung cancer irrespective of smoking. It is also assumed that people of a certain personality are more likely to smoke than others. There is evidence for both these propositions. My original work with Dr. Kissen, an eminent British oncologist, showed very marked personality differences between lung cancer patients and patients suffering from non-malignant tumours, with the personality assessment made before diagnosis. Since then, a large-scale study in East Germany has replicated our findings (themselves replicated in another study by Kissen), and has found similar personality traits to those characteristic of lung cancer patients in women with cancer of the breast. Other studies, also indicating a relation between lung cancer and personality, are cited in my book.

In a similar way, my early work with Tarrant and Woolf established a correlation between personality and smoking, and many studies in different countries have since confirmed our findings, and added new ones. We may thus say that the fundamental assumption of Fisher's genetic theory have found empirical support, and we may add that there is also some modest support for my own attempt to integrate these two major fields. Unfortunately there has been too little work along these unusual and somewhat unorthodox lines to say that
the results are anything more than suggestive, and the theory linking them is still in a very elementary stage; nevertheless, as far as the findings go they support the genetic rather than the causal theory, although they do not necessarily contradict the latter. It seems unfortunate that the premature crystallization of spurious orthodoxies has prevented the genetic theory from attracting sufficient research grants to work it out in sufficient detail, and to carry out the research necessary to put it on a more acceptable footing.

Recently some progress has been made on the theoretical development of the genetic hypothesis by linking it with research on stress, in particular the differential effects of chronic and acute stress, and the "inoculation" theory of stress. However, in the absence of large-scale research into the refinements of this theory, and more widespread familiarity with and criticisms of its details, not too much should be claimed for it other than it presents a viable alternative to the causal theory.

In relation to the causal theories of coronary heart disease (CHD), similar criticisms apply as do in the case of lung cancer. There are considerable unreliabilities in diagnosis; there are large numbers of factors other than smoking which have been associated and which are not usually
controlled for in studies of the effects of smoking; inhalers do not on the whole differ from non-inhalers in disease proneness; the statistical relation between cigarette smoking and CHD disappears in many countries, e.g. Finland, Holland, Yugoslavia, Italy, Greece and Japan; there is an absence of dose-response relationship, i.e. there is little or no relation between duration of heavy cigarette smoking and risk of myocardial infarction; and the correlation between number of cigarettes smoked and CHD is not linear; ex-smokers in some studies appear to be safer than non-smokers; some types of CHD, such as angina pectoris (which comprises some 20% of CHD in men) fail to show even a statistical correlation with cigarette smoking; some types of smoking (cigar, pipe) fail to show even a statistical correlation with CHD; etc. These are anomalies or failures of the causal theory which demand an explanation before the causal theory can be accepted.

Some of these facts are much more readily explained in terms of a genetic-personality theory; thus the differential effects of cigarette vs. pipe/cigar smoking may find an explanation in terms of the known differences in personality type associated with these different smoking patterns.

The general conclusion would seem to be that in the case of CHD, as in the case of lung cancer, proof for the causal influence of smoking is still lacking and is by no
means as clear-cut and decisive as is often alleged. There is evidence in the case of CHD of genetic factors, and there are published correlations with personality; here too there appears an important element of stress determining the appearance of CHD, and stress is intimately linked with personality. No formal theory of genetic determination of CHD has yet been put forward, but it seems likely that such a theory is needed as an alternative (or perhaps as complementary) to the causal theory for an explanation of the many gaps and anomalies in the latter.

One important function of the genetic theory has been that of explaining the reasons why people smoke, and to link these reasons with their differential personality patterns. Work along these lines has had the important effect of suggesting new and improved ways of teaching people to give up smoking. The causal theory of smoking causing disease has nothing to say on this topic. Another important function of the genetic theory has been to suggest better designs for research in this complex field; a good example is the use of the discordant twin method by Cederlof, Lundman and others, i.e. the investigation of the illness patterns of identical twins of whom one smokes, the other not. If this type of research had been carried out on the large and international scale required, instead of investing in the redundant and
scientifically not very valuable replication of correlational studies, we would know far more about the relation between smoking and disease than we do now. Such studies allow us to look at environmental factors, including those of smoking, while controlling for genetic factors; this is essential if any convincing results are to be achieved.

In summary I would like to state that the causal theory of smoking as being responsible for lung cancer and coronary heart disease, while it has found strong support, is far from being established, and has many gaps, anomalies and contrary findings to contend with; these are too frequently glossed over and dismissed as unimportant, when in reality they may be found to discredit the causal theory in whole or in part. An alternative theory, based on genetics and implicating personality factors, is much less well developed, more complex, and at present not too well known to oncologists; nevertheless there are many well-established facts which suggest that in part if not in whole it can account for the major findings. At the very least, this alternative theory suggests novel research methodologies which would serve to overcome the difficulties of the older methods and remedy their lack of proper controls. The possibility has also been raised that the two theories may be complementary, rather than opposed to each other; this possibility too
should be looked into from the experimental point of view. What is certain is that at the moment no final decision can be made about whether or the degree to which cigarette smoking may cause lung cancer or coronary heart disease, how it interacts with other factors (stress; personality), or how we can best protect the health of our citizens in relation to these diseases. "In ignorance, absten!" warned the famous French scientist, Claude Bernard; hasty action on the basis of partial knowledge is unlikely to be in the best interests of those most concerned, namely the prospective victims of lung cancer and coronary heart disease.

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