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SMOKING AND HEALTH

Paper by Dr. D.C.I. Felton

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THE CHARGES.

The first allegations that there were health problems associated with the habit of smoking arose from a study of mortality rates from various diseases. Following the development of effective therapy against tuberculosis, doctors had expected deaths from respiratory disease to decrease. Instead, they observed an increasing incidence of deaths from lung cancer.

The study of the incidence and distribution of disease in populations is known as epidemiology and there are basically two ways in which this can be carried out. Using the simpler, cheaper technique of retrospective survey, doctors found that those who had died from lung cancer were much more likely to have been cigarette smokers. Prospective studies are to be preferred and a number of these have tended to show that the risk of death from lung cancer is greater among heavy smokers. One of the best known studies is that of British doctors by Doll and Peto, which was interpreted as showing increased risk of a number of diseases, particularly lung cancer and coronary heart disease (CHD). Other prospective studies have come to similar conclusions resulting in a list of so-called smoking-associated diseases.

This all seems pretty clear cut and has been used by antismokers to influence opinion formers,

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such as journalists, to spread the antismoking message and so bring pressure on legislators.

But is it all so clear cut ?

THE DOUBTS.

Epidemiology is based on statistics and this can be a difficult science to understand. It has been said that

"Statistics pose questions, they don't answer them".

Firstly, statistics cannot prove (or imply) a cause and effect relationship. So the existence of an undeniable statistical association between smoking and a given disease does NOT mean that smoking is a cause of the disease.

Secondly, epidemiology is a very inexact science, subject to several potential sources of error.

(a) Inaccurate death certification has been acknowledged to be substantial in a number of papers; perhaps the most noteworthy being a report from the Royal College of General Practitioners.

(b) Bias in samples is unavoidable if smokers and non-smokers are basically different sorts of persons. The A.C.S. one million persons study was not typical of the U.S. population as a whole in terms of age, racial type, residence or social status.

(c) The existence of a genetic component is revealed by studies of twin pairs. Identical twins generally have similar smoking habits (if they are smokers) and it is very difficult to find pairs of

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identical twins with different smoking habits. So, if the genetic make-up of the samples is different there can be differences in susceptibility to diseases. These differences in disease susceptibility may be linked to different personality types. In particular, to anticipate briefly, there are two main personality types :

Type A - the hard-driving, outgoing person

Type B - the placid, relaxed, easy person

It is the Type A personality which tends to have a higher incidence of CHD, irrespective of his smoking habits. The fact that Type A people tend to smoke more than do Type B is a personality factor which, unless care is taken, could become confounded with disease incidence.

Finally, the results for one kind of population cannot be extrapolated to a different population. There is no reason why results found for an American population should apply to Kenya.

Ideally, the way to overcome the genetic factor is by the study of identical twins who differ in smoking habits, difficult though these are to find. The results of such studies are generally too small for statistical significance, but they tend to show no differences in disease incidence as a result of smoking habits.

MECHANISMS.

The causal interpretation of the statistical

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association between smoking and disease would be strengthened if a plausible mechanism were demonstrated whereby smoke could be shown to cause a particular disease. Despite some 30-35 years of scientific effort, this has not been possible for any of the so-called smoking-associated diseases.

(a) Cancer.

Painting huge doses of smoke condensate on the backs of mice does produce tumours. But condensate, however fresh, is not smoke and mouse skin is not lung tissue. Attempts to produce lung tumours by exposing animal lungs to fresh smoke has only given the odd tumour in specially sensitive animals. In any case, these models have failed to reveal a mechanism. No-one has been able to show how cancer is caused.

Despite this, authorities have advocated the development of low-tar products, believing these might be "less hazardous". The industry, world-wide, has responded to the demands of consumers, stimulated by this advice, and products today are very different from those existing in 1964, the date of the first report of the U.S. Surgeon-General.

Some people have claimed that the development of low delivery products has led to a reduction in disease risk, particularly that of lung cancer or in the rate of incidence of cellular abnormalities in the lung as found at post-mortem examination.

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At a well publicised conference, many of the most celebrated anti-smokers agreed among themselves that a "less hazardous" cigarette was feasible. They even provided data for its design.

This seemed very plausible, provided one accepted the underlying assumptions, but a detailed analysis of mortality rates showed that for most people, other than the oldest age group, the reduction in death rates began before low-tar cigarettes were widely available. Add to this that mortality rates from stroke (one of the smoking associated diseases) had been steadily decreasing since the 1920's and the more recent reduction in deaths from CHD, which is not linked to a change in smoking habits, in USA and Australia - but not in U.K. - and the plausibility becomes more questionable.

Around this time, reports began to appear which indicated that diet might play an important role in cancer incidence. Thus, fat intake was shown to be associated with cancer incidence and Vitamin A and its precursors appeared to be protective against cancer, particularly lung cancer.

So diet, ignored in the past, may be a key factor and should be added to the other factors responsible for potential bias in epidemiological samples.

(b) Coronary Heart Disease

The incidence of CHD is very different from that of lung cancer. It affects non-smokers

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nearly as much as it does smokers; it has roughly the same incidence rate for all social classes and many factors, besides cigarette smoking, have been indicted as "causes". Personality types (Type A vs Type B) have been mentioned as showing different rates of CHD, irrespective of smoking habit. A number of studies have shown a significant correlation between stress and CHD and studies with identical twins have demonstrated a strong genetic influence. Death rates vary greatly between countries and do not correlate with cigarette consumption data.

Once again, no plausible mechanism has been advanced as to how tobacco smoke might cause CHD. No acceptable animal inhalation studies using tobacco smoke have been developed for CHD. Both carbon monoxide and nicotine have come under suspicion (perhaps because they are among the major components of tobacco smoke) as causing CHD; but, since smokers of pipes and cigars, who are exposed to both substances, are said to have no increased risk of CHD, both substances appear to be exonerated from blame.

Despite these failures to show how cigarette smoking might lead to CHD, a number of intervention trials have been undertaken in recent years, some at very great expense. In these a group of people are divided, somewhat arbitrarily into two, or more, groups, some of whom are exposed to persuasion to stop smoking, in the expectation that those who gave

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up smoking would show an improvement in their health, particularly in terms of CHD. Other attempts were made to influence blood pressure or blood lipid levels.

The results from these trials have now been collected and have confirmed that factors other than smoking are involved in the incidence of CHD. Some of those who gave up smoking had a worse health record than those who continued to smoke.

Four of the trials, including the very expensive MRFIT in USA, failed to produce statistically significant reductions in CHD mortality.

A typical conclusion is that from the Oslo study:

"In the seven-country study there was no significant association between CHD incidence and smoking in the different countries, and in the prospective necropsy series of the Oslo study a significant correlation between prevalence of coronary raised atherosclerotic lesions and smoking could not be shown."

(c) Chronic Obstructive Lung Disease (COLD)

This recently adopted terminology conceals the real difficulties that exist over the definitions of chronic bronchitis and emphysema. While emphysema can be diagnosed post mortem or by using samples of excised lung, most diagnosis on living subjects is dependent on the interpretation of various tests of respiratory function.

The latest report of the US Surgeon-General, published in 1984, considered COLD purely in relation to cigarette smoking and ignored a number of pathological mechanisms and factors suspected of being related to COLD.

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It is possible to produce emphysema in animals by exposing them to known chemical pollutants such as nitrogen dioxide, sulphur dioxide and smog, but similar experiments using cigarette smoke have been unsuccessful.

Distribution of Diseases.

If cigarette smoke were the most important cause of the so-called smoking-associated diseases, it might be expected that the geographical distribution of the diseases would be similar - perhaps related to consumer patterns or population densities.

Where such geographical distributions are known, in the USA for example, this is clearly not the case. In the U.K., the incidence of CHD appears to be related to the hardness of the water supply, rather than to any other factor.

Passive Smoking.

Passive (or involuntary) smoking is a different sort of problem. Up to this point, we have been considering the allegations that smokers may be putting their own health at risk by indulging in the habit. Passive smoking means the involuntary inhalation of tobacco smoke by a non-smoker (or a smoker) exposed to smoky atmospheres. If a risk is thereby incurred, it is not a voluntary one.

Before 1979, the allegations against passive smoking tended to be somewhat marginal. Passive

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smoking was only viewed as either a social problem or as an annoyance. One or two militant anti-smokers tried to make it an issue, but most medical authorities concluded that involuntary exposure to smoke was without risk.

This view was questioned by the publication of a paper by White and Froeb, which alleged that prolonged exposure to smoky atmospheres led to impairment of respiratory function in non-smokers. There were many things about the White and Froeb study which were questionable - the selection of the sample, the reliability of the measurements, the significance of the alleged impairment. But the paper was not challenged publicly by non-industry sources for some years. Even though it is now not accepted generally, it is still cited by anti-smoking activists.

It was soon followed by a retrospective case-control study in Athens, which alleged that the non-smoking wives of smokers had an elevated risk of lung cancer, but it dealt with relatively few cases.

Soon after this, another well-known epidemiologist Hirayama, published results from his large prospective study in Japan. This also claimed to show that non-smoking wives of smokers had a greater incidence of lung cancer than wives of non-smokers. A later analysis of his data has led to a further claim of increased risk of brain cancer from passive smoking.

These allegations were counterbalanced soon

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afterwards by a publication by Garfinkel, based on an analysis of data from the ACS million person study. This showed no significant increased incidence of lung cancer in non-smoking wives of smokers. Similarly, the Greek study was balanced by a study in Hong Kong which failed to show an effect of passive smoking. These papers were all reviewed in a German Medical journal by an independent authority who concluded that the case against passive smoking had not been demonstrated.

Since these publications, there have been a number of similar claims and counterclaims. In evaluating these contradictory studies, one must remember the potential sources of error in epidemiology. Moreover, the plausibility of the allegations must be considered. Is it possible that merely breathing air containing tobacco smoke can expose a non-smoker to a risk similar (or a little less) than that alleged to be the risk of an active smoker ?

What are the levels of smoke to which passive smokers may be exposed ? These have been measured in various natural situations, in terms of nicotine. How do they compare with what is absorbed by active smokers ? This can be determined by measuring the urinary levels of nicotine.

The measurement of levels of other smoke components in atmospheres is possible, but very difficult. But the demonstration that these are very low does not stop misrepresentation by anti-smokers, whose aim

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is to sow seeds of doubt and worry in the minds of smokers and non-smokers alike. Who could continue with his habit if he believed he was harming those he held dearest ?

Some years ago, the U.K. Laboratory of the Government Chemist published data on the mainstream and sidestream yields of cigarettes of various tar categories. This is how a regional medical adviser interpreted the data. Such ignorant (or wilful) misinterpretation by opinion formers, rather than proven and demonstrable danger, can affect the perception of risk by non-smokers. It is for these reasons that the passive smoking issue may be seen as the biggest threat to the industry.

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