THE RELATION OF SOCIAL TO PATHOPHYSIOLOGICAL PROCESSES: 
EVIDENCE FROM EPIDEMIOLOGICAL STUDIES

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ABSTRACT

Epidemiological strategies have proved appropriate for studying aetiology of chronic diseases. Sound application of epidemiological techniques allows the testing of hypotheses. Such techniques have been applied only to a limited extent in studying psycho-social factors. This is, in part, owing to the difficulty of adapting both concepts and measures for study in different social and cultural groups. Studies of Type-A behaviour, occupational stress and social supports provide examples where this problem must be tackled.

The demands of large population studies mean that relatively simple measures are used in epidemiological studies. Therefore results from these studies must be taken together with other smaller, but more in depth, investigations.

EPIDEMIOLOGICAL APPROACH TO CAUSATION

Who "discovered" that smoking causes lung cancer? Was it a clinician who noticed that his last 3 cases of lung cancer had all been heavy smokers and said "I wonder if...?". Was it the molecular biologist, studying the action of constituents of tobacco smoke on cells? Was it the experimenter who taught beagle dogs to smoke? Was it the epidemiologist who, in a variety of ways, linked smoking with increased risk of lung cancer?

There is not a single answer. The elaboration of a hypothesis is not, by itself, a discovery of causation - evidence is required to support it; nor is the astute clinical observation - it requires the systematic estimation of lung cancer incidence in exposed and 'unexposed'; nor is the demonstration of a plausible biological mechanism - it tells us what can happen, not what does happen; nor the animal experiment - does it apply to humans? There were many hypotheses about the cause of lung cancer, cigarette smoking among them. The contribution of the epidemiologists was to take one of these hypotheses and test it. The epidemiologists showed smoking to be the likeliest cause: by showing that smokers were at increased risk, that the increased risk was
unlikely to be due to factors other than smoking, that there was a
dose-response relationship, that the distribution of lung cancer in
the population followed the distribution of smoking, that trends over
time in lung cancer were correlated with trends in cigarette smoking,
that ex-smokers had a lower risk than continuing smokers, and so on.

What possible insights does this hold for the study of social
and psychological factors? Epidemiological study must form one of
the strategies for demonstrating causation. But where psycho-social
factors have been included in epidemiological studies, not all the
strategies available have been utilised fully.

For example, the association between type A behaviour and coronary
heart disease (CHD) is among the strongest of any psycho-social factor.
The most compelling evidence for this association comes from the
Western Collaborative Group Study - a study of predominantly middle
class white American men (Rosenman et al 1976). Our confidence in
the Type-A:CHD association is increased by its demonstration in women
(Haynes et al 1980a), and in Europe as well as in the U.S.A. (French-
Belgian Collaborative Group 1982). What has not been shown is that
Type-A behaviour follows the distribution in the population of CHD.
Or to put it another way, very little work has been done on the extent
to which variations in the frequency of Type-A behaviour may explain
variations in CHD over time, geographically and between subgroups of
the populations. Could an increase in Type A behaviour have accounted
for the 20th century epidemic of CHD? and a decrease in Type-A
contributed to CHD decline in several countries? We have no information.
To what extent could differences in Type A behaviour account for
international differences in CHD occurrence? We have little information.

Such information as we have, within populations, is not
encouraging. In our Whitehall study of British civil servants, for
example, men in the lower grades of employment had more than three
times the risk of CHD death (Marmot et al 1978), but fewer were Type-A's
than men in the upper grades (Marmot 1982). In Framingham, working
women have less heart disease than men but equal prevalence of Type-A
behaviour (Haynes et al 1980b).

These conflicting data suggest either that differences in CHD
between subgroups of the population are not due to differences in
Type-A behaviour, or that the measurement (and perhaps the concept)