Citation:

The Environment and Disease: Association or Causation?

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Amongst the objects of this newly-founded Section of Occupational Medicine are firstly 'to provide a speach, not readily attended elsewhere, whereby physicians and surgeons with a special knowledge of the relationship between sickness and injury and conditions of work may discuss their problems, not only with each other, but also with colleagues in other fields, by holding joint meetings with other Sections of the Society'; and, secondly, 'to make available information about the physical, chemical and psychological hazards of occupation, and in particular about those that are not so easily recognised'.

At this first meeting of the Section and before, with however loudspeaker intentions, we set about impressing our colleagues in other fields, it will be proper to consider a problem fundamental to our own. How in the first place do we detect these relationships between sickness, injury and conditions of work? How do we determine what are physical, chemical and psychological hazards of occupation, and in particular those that are rare and not easily recognized?

There are, of course, instances in which we can reasonably answer these questions from the general body of medical knowledge. A particular and perhaps extreme, physical environment cannot fail to be harmful, a particular dimessal is known to be toxic to man and therefore suspect on the factory floor. Sometimes, alternatively, we may be able to consider what might be a particular environment do to man, and then see whether such consequences are indeed to be found. But more often than not we have no such guidance, no such means of proceeding: more often, than not we are dependent on our observation and enumeration of defined events for which we then seek an explanation. In other words we see that the event E is associated with the environmental feature A. Thus, to take a simple example, some form of respiratory illness is associated with a dust in the environment. In what circumstances can we pass from this

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President's Address

observed association to a verdict of causation? Upon what basis should we proceed to do so?

I have no wish, nor the skill, to embark upon a philosophical discussion of the meaning of 'causation'. The 'cause' of illness may be immediate and direct, it may be remote and indirect underlyng the observed association. But with the aims of occupational, and almost synonymous preventive, medicine in mind the decisive question is whether the frequency of the undesirable event B will be influenced by a change in the environmental feature A. How such a change in its influence may call for a great deal of research. However, before dedicating 'causation' and taking action we shall not invariably have to sit around waiting the results of that research. The whole chain may have to be unravelled or a few links may suffice. It will depend upon circumstances.

Disregarding then any such problem in semantics we have this situation. Our observations reveal an association between two variables, presently clear-cut and beyond what we would have to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?

I (strength). First upon my list I would put the strength of the association. To take a very obvious example, by comparing the occupations of patients with respiratory cancer with the occupations of patients presenting with other disease, Persico Pont could reach a correct conclusion because of the curency increase of scrotal cancer in the chimney sweeps. Even as late as the second decade of the twentieth century, writes Richard Doll (1960), 'the mortality of chimney sweeps from scrotal cancer was some 200 times that of workers who were not specially exposed to tar or mineral oils and in the eighteenth century the relative difference is likely to have been much greater.'

To take a more modern and more general example upon which I have now referred for over fifteen years, prospective inquiries into smoking have shown that the death rate from cancer of the lung in cigarette smokers is nine to ten times the rate in non-smokers and the rate in heavy cigarette smokers a twenty to thirty times

Note: (D) Rev. J. Dyer, M.A. (Associate 1923) Public Health: Advisory Health Advisory Committee.
as great. On the other hand the death rate from coronary thrombosis in smokers is no more than twice, possibly less, the death rate in non-smokers. Though there is good evidence to support causation it is merely much easier in this case to think of some features of life that may go hand-in-hand with smoking - features that might conceivably be the real underlying cause or, at the least, an important contributory, whether it be lack of exercise, nature of diet or other factors. But to explain the pronounced excess in cancer of the lung in any other environmental terms requires some feature of life as intimately linked with cigarette smoking and with the amount of smoking that such a feature should be easily attributable. If we cannot demonstrate or casually infer a specific one, then in such circumstances I think we are reasonably entitled to reject the vague contention of the amoeboid sort you can't prove it, there may be such a feature.

Certainly in this situation I would reject the argument sometimes advanced that what matters is the absolute difference between the death rates of our various groups and not the ratio of one to the other. That applies upon what we want to know. If we want to know how many extra deaths from cancer of the lung will take place through smoking (i.e. presuming abjuration), then obviously we must use the absolute differences between the death rates. 0.951 per 1,000 per year in non-smoking doctors, 0.857 in those smoking 1-14 cigarettes daily, 1.399 for 15-24 cigarettes daily and 2.907 for 25 or more daily. But it does not follow here, or in more specifically occupational groups. The best we can do with the available mortality is also the best measure in relation to etiology. In this respect the states of 6.20 and 32 to 1 are far more informative. It does not, of course, follow that the differences revealed by radio are of any practical importance. Maybe they are, maybe they are not; but that is another point altogether.

We may recall John Snow's classic analysis of the deaths from the cholera outbreak in Soho (Snow 1855). The death rate that he recorded in the customers supplied with the grossly polluted water of the Southwark and Vauxhall Company was in truth quite low - 71 deaths in each of 70,000 houses. What stands out strikingly is the fact that the small rate is 14 times the figure of 5 deaths per 10,000 houses supplied with the unsanitary water of the rival Lambeth Company.

In thus putting emphasis upon the strength of an association we must, nevertheless, look at the observers. Indeed, we must not be ready to dismiss a cause-and-effect hypothesis merely on the grounds that the observed association appears to be slight. There are many examples in medicine when this is in truth so. Relatively few persons occupationally exposed to rat's urine contract Weil's disease.

(2) Causality: Next on my list of features to be specially considered I would place the consistency of the observed association. It has been repeatedly observed by different persons, in different places, circumstances and times.

This requirement may be of special importance for those rare hazards singled out in the Section II terms of industry today. Many an environmental association may be thrown up. Some of them on the customary tests of statistical significance will appear to be unlikely or too close to chance. Nevertheless whether chance is the exclusion of whether a true hazard has been revealed may sometimes be answered only by a replication of the circumstances and the observations.

Returning to my more general example, the Advisory Committee to the Surgeon-General of the United States Public Health Service found the association of smoking with cancer of the lung in 29 retrospective and 7 prospective inquiries (US Department of Health, Education & Welfare 1964). The journals here is that broadly the same answer has been reached in quite a wide variety of situation and techniques. In other words the curves indicating the percentages fall sick are not due to some constant error or fallacy that permeates every inquiry. And we have indeed to be on our guard against that.

Tak, for instance, an example given by Heady (1948). Patients admitted to hospital for operation for peptic ulcer are questioned about recent domestic anxieties or crises that may have precipitated the acute illness. As controls, patients admitted for operation for a simple hernia are similarly queried. But, as Heady points out, the two groups may not be in pari materia. If your wife ran off with the lodger last week you will have to take your perforated ulcer to hospital without delay. But with a hernia you might prefer to stay at home for a while - to mourn (or celebrate) the event. No number of postoperative wounds would remove or necessarily reveal that fallacy.

We have, therefore, the somewhat paradoxical positions that the different results of a different inquiry certainly cannot be held to refute the original evidence; yet the same falsely the same form of inquiry is greatly strengthens the objection possibly to the real result reached in quite different.

Once again looking at the other there will be occasions when rij or impossible and yet we should draw conclusions. The expanded report of South Wales is an example. I quote from the Memorial Lecture that I gave Institution of Actuaries:

'The population at risks, worker numbered about one thousand. D 1929 to 1936, account of 1,000 about the lungs, eleven of them had died, assaults. The age range England and Wales at this time ascultated one case from amongst the 1,000, and a third cause of the three (to compute the other body sites cancer had any certainty and one would do so at 1-in-1 times. There had been other cases of mortality and the period 72 would have been expect. The final division of the in relation to their jobs showed cause of the lung and now had the workers employed in the plant.

'Now recently my colleague, a 1920 had a year's or two, he was in control of the long and 73 months. He recorded numbers of workers deaths, respectively.

'In 1932, long before any specific attempts to alter changes in these, no case of cause of the number is in any man who has been reared, and in this case there has not been case of the lung, the readings from the area is uniquely a feature in it deliciously, roughly, the last 23 years.

'No causal agent of there is identified. Until recently no one had given any clue or any of the pathological evidence. Yet I would hesitate to accept it as proof of anything' (Healy 1942).

In relation to my present di or parallel investigation we both to make up our minds and there is no difficulty in do
original evidence; yet the same results from pre-
cisely the same form of inquiry will not inevitably greatly
strengthen the original evidence. I would myself
put a good deal of weight upon similar
results reached in quite different ways e.g. pros-
evocatively and retro-evocatively.

Once again looking at the obverse of the coin,
there will be occasions when repetition is absent
or impossible and yet we should not hesitate to
draw conclusions. The experience of the nickel
poison of South Wales is an outstanding example. I quote from the Allied Wallsen
Memorial Lecture that I gave in 1962 to the
Institute of Architects:
The population at risk, workers and policemen,
numbered about one thousand. During the ten
years 1932 to 1935, twelve of them had died from removal of
the lung, eleven of them had died from cancer of the
rectal tissues. At the age specific death rate of
England and Wales at that time, one might have
expected one death from cancer of the lung (to
c ompare with the 12), and a fraction of a death from
cancer of the nose (to compare with the 1). In all
other bodily sites cancer had appeared on the death
certificate 31 times and one would have expected to
have 10-11 times. There had been 27 deaths from all
other causes of mortality and over the ten years' period
22 house had been expected at the national
c death rates. Finally division of the population at risk
in relation to their age showed that the forces of
cancer of the lung and nose had fallen wholly upon
the workers employed in the chemical processes.

More recently my colleague, Dr. Richard Doll, has
brought home this story a stage further. In the nine
years 1946 to 1956 there had been, he found, 48 deaths
from cancer of the lung and 12 deaths from cancer of
the nose. He showed the numbers expected at normal
d rates of mortality to be respectively 39 and 1.

In 1929, long before any special hazard had been
recognized, certain changes in the refinery took
place. The cause of cancer of the nose has been thought
in any way that affected the workers after that date,
and in these cases there has been no increase in
cancer of the lung in other respects, no excess in both
in uniquely a feature in men who entered the
refinery, is, roughly, the first 27 years of the present
century.

No special analysis of those affected was identified. And recently no animal observation
had given any clue or any support to this wholly
observation. Yet I wonder if any of us would
hesitate to accept it as a proof of a grave industrial
hazard? (Doll 1965).

In relation to my present discussion I know of no
animal observation. We have (or certainly
had) to make up our minds on a unique event
and there is no difficulty in doing so.

(3) Specificity: One reason, needless to say, is the
specificity of the association, the third character-
istic which inevitably we must consider. If, as
here, the association is limited to specific workers
and to particular sites and types of disease and
more is no association between the work and other
modes of dying, then clearly that is a strong
argument in favour of causation.

We must not, however, over-emphasize the
importance of the characteristic. Even in the
present example there is a cause and effect rela-
tion with two different sites of cancer - the
lung and the nose. Milk as a carrier of infection and,
in that sense, the cause of disease can pro-
duce such a disparate galaxy as scarlet fever,
diphtheria, tuberculosis, urinary fever, sore
throat, diphtheria and typhoid fever. Before the
discovery of the underlying factor, the bacterial
origin of disease, beef would have been done by
pushing too firmly the need for specificity as a
necessary feature before convicting the dairy.

Coming to modern times the prospective
investigations of smoking and cancer of the lung
have been criticized for not showing specifically -
in other words the death rates of smokers is
higher than the death rate of non-smokers from
many causes of death (though in the results of
Poll & Hill 1964, do not show this). But here
surely one must return to my first characteristic,
the strength of the association. If other causes of
death are raised 10, 30 or even 50% in smokers
whereas cancer of the lung is raised 200-1,000%
we have specificity, a specificity in the magnitudes
of the association.

We must also keep in mind that diseases may
have more than one cause. It has always been
possible to acquire a cancer of the sarcoma
without smoking, clamydiae or smoking to mule
spinning in Lancashire. One-to-one relationships
are not frequent. Indeed I believe that multi-
causation is generally more likely than single
causation. Thus possibly if we attempt to
the answers we might get back to a single factor.

In short, if specificity exists we may be able to
draw conclusions without hesitation; if it is not
apparent, we are not thereby necessarily left
standing in vacuity on the issue.

(4) Temporality: My fourth characteristic is the
temporal relationship of the association - which
is the start and which the end? This is a question
which might be personally relevant with scenes
of evolution. Does a particular cigarette lead to
cancer or do the early stages of the disease
lead to those particular dietary habits? Does a


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lead to those particular dietary habits? Does a
particular occupation or occupational environment promote infection by the tubercle bacillus or are the men and women who select that kind of work more liable to contract tuberculosis whatever the environment – or, indeed, have they already contracted it? This temporal problem may not arise often but it certainly needs to be remembered, particularly with selective factors at work in industry.

(5) Biological gradient: Fifthly, if the association is one which can reveal a biological gradient, or dose-response curve, then we should look most carefully for such evidence. For instance, the fact that the death rate from cancer of the lung rises linearly with the number of cigarettes smoked daily, adds a very great deal to the simpler evidence that cigarette smokers have a higher death rate than non-smokers. That comparison would be weakened, though not necessarily destroyed, if it depended upon, say, a much heavier death rate in light smokers and a lower rate in heavier smokers. We should then need to envisage some much more complex relationship to satisfy the cause-and-effect hypothesis. The clear dose-response curve admits of a simple explanation and obviously puts the case in a clearer light.

The same would clearly be true of an alleged dust hazard in industry. The dossier the environment the greater the incidence of disease we would expect to see. Often the difficulty is to secure some satisfactory quantitative measure of the environment which will permit us to explore this dose-response. But we should invariably seek it.

(6) Plausibility: It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day.

To quote again from my Alfred Watson Memorial Lecture (1962), there was...

...no biological knowledge to support (or to refute) Pott's observation in the 18th century of the excess of scrotal cancer in chimney sweeps. It was lack of biological knowledge in the 19th that led a prime assize writer on the value and the fallacy of statistics to conclude, amongst other "absurd" associations, that "it could be no more strikes for the sweeper who passed the night in the storage of an eminent ship to ascribe the typhus, which he then contracted, to the vermin with which bodies of the sick might be infested."

And coming to nearer times, in the 20th century there was no biological knowledge to support the evidence against rubella.

In short, the association we observe may be new to science or medicine and we must not dismiss it too lightly because it just too odd. As Sherlock Holmes advised Dr Watson, "when you have eliminated the impossible, whatever remains, however improbable, must be the truth."

(5) Causality: On the other hand the cause-and-effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease – in the expression of the Advisory Committee to the Surgeon-General it should have coherence.

Thus in the discussion of lung cancer the Committee finds its association with cigarette smoking coherent with the temporal rise that has taken place in the two variables over the last generation and with the sex difference in mortality – features that might well apply in an occupational problem. The known aetiological ratio of lung cancer mortality does not depart from coherence, nor the restriction of the effect to the lung.

Personally, I regard as greatly contributing to coherence the histopathological evidence from the bronchial epithelium of smokers and the isolation from cigarette smoke of factors carcinogenic for the skin of laboratory animals. Nevertheless, while such laboratory evidence can enormously strengthen the hypothesis and, indeed, may determine the actual causative agent, the lack of such evidence cannot nullify the epidemiological observations in man. Animal can undoubtedly cause cancer of the skin in man but it has never been possible to demonstrate such an effect on any other animal. In a wider field John Snow's epidemiological observations on the conveyance of cholera by the water from the Broad Street pump would have been put almost beyond dispute if Robert Koch had been there to isolate the vibrio from the baby's日记, the well itself, and the gentleman in delinquent health from Brighton. Yet the fact that Koch's work was to be awaited another thirty years did not really weaken the epidemiological case though it made it more satisfying. Similarly public health authorities had to withstand the criticism of the day – both just and unjust.

(9) Experiment: Occasionally it is possible to appeal to experimental, or semi-experimental, evidence. For example, because of an observed association some preventive action is taken. Does it in fact prevent? The dust in the workshop is reduced, lubricating oils are changed, persons stop smoking cigarettes. Is the frequency of the associated events affected? Here the strongest

support for the causation is revealed.

(9) Analogy: In some circumstances fair to judge by analogy, the former is naturally merely be ready to accept evidence with another drug disease in pregnancy.

Here then are nine different of which we should study a every causation. What I do in has been suggested is that some down some hard-and-fast must be obeyed before we effect. None of mine v indisputable evidence for a and-effect hypothesis and n in a slow run. What they or less strength, is to help minds on the fundamental a other way of explaining the is there any other proof e cause and effect.

Tests of Significance

No formal tests of significance. Such tests are, in the effects that the pity and they will instruct us in of those effects. Beyond nothing to the "proof" of our

Nearly forty years ago, a occupational health that I am Health and Safety Council was one that cor the cotton-spinning mills 1930). The question that I use of the National Health that time, was this: Do the room of the spinning mill, that clean the raw cotton is ence in any way differ operative in the same m to the dust features of the bedroom unqualified "Yes." From other workers suffer much from respiratory or from whether or not different from that. This pronounced difference was derived not periods of sickness but a number of repeated site childhood workers.
support for the causation hypothesis may be
found in the following.

Analyze: In some circumstances it would be
fair to judge by analogy. With the effects of
salubrity and salubrity alone before us we
might well be ready to suspect that similar
effects are caused by another drug or another
cause in pregnancy.

There are at least nine different viewpoints
from which we might study association between
events. What do we not believe? - that we can
modify the course, some hard-and-fast rules of evidence that
must be observed before we accepts facts and
effects. None of the six viewpoints can bring
distinguishing evidence for or against the cause-
effects hypothesis and none can be required as a
evidence. What can they do, with greater or less
strength, to help us to make up our minds on
the fundamental question: Is there any other
way of explaining the set of facts before us,
if there any other answer equally, or more, likely
than cause and effect?

Tests of Significance
No formal tests of significance can answer the
questions that arise. Such tests can, and should, remind
us of the effects the play of chance can cause, and
they will instruct us in the likely magnitude
of such effects. Beyond that they contribute
nothing to the proof of our hypothesis.

Nearly forty years ago, among the studies of
comparisons health that I made for the Industrial
Health Research Board of the Medical Research
Council was one that concerned the workers in
the cotton-spinning mills of Lancashire (1980).
The question that I had to answer, by the
establishment of the Health Insurance records of
that time, was this: Do the workers in the card-
ning-milling or the spinning mill, who tend to result
in child incidence, those cotton mills, have a lower
incidence of respiratory illnesses including illnesses
which are relatively unnoticeable to the dust and fibre
that were features of the question? The answer was no.

Qualitative. Yet, from age 30 to age 60, the
respiratory worker suffer from respiratory illnesses in
those who do not, and this effect is greatest
in the early years of life. However, when we look at the
experience of the respiratory illnesses and the
differences in the nickel workers, we find that
there is a difference between the groups that
is not due to chance. The evidence is therefore
stronger than chance and may be more likely
than cause and effect.

The most important consideration is that
we should be able to see whether the
results are statistically significant. If they
are not, then we cannot be sure that the
difference is real. If they are, then we can be
more confident that the difference is real.

I would wonder whether the pendulum has not swung
too far - not only with the attentive pupils but
even with the statisticians themselves. To
draw conclusions without statistical analysis is
certainly a bad idea. Fortunately, I believe that
we have not yet gone so far as to say that
the study of the respiratory illnesses in
the cotton-spinning mills is not important
because it is negligible, or because it is
not statistically significant. Therefore, we
must be careful not to overgeneralize.

The situation is that we have
not enough information to make a
definite conclusion. However, it is
reasonable to say that the
results are statistically significant
and that the difference is real.

Of course I exaggerate. Yet too often I
suggest that we waste a day that we group the shadow and
The Case for Action

Finally, in passing from association to causation I believe in 'real life' we shall have to consider what flows from that decision. On scientific grounds we should do no such thing. The evidence is there to be judged on its merits and the judgments (in that sense) should be utterly independent of what hangs upon it – or who hangs because of it. But in another and more practical sense we may surely ask what is involved in our decision. In occupational medicine our object is usually to take action. If this be operative cause and that be deleterious effect, then we shall wish to intervene to abolish or reduce death or disease.

While that is a commendable ambition it almost inevitably leads us to introduce differential standards before we convict. Thus on relatively slight evidence we might decide to restrict the use of a drug for early-morning sickness in pregnant women. If we are wrong in deducing causation from association no great harm will be done. The good lady and the pharmaceutical industry will doubtless survive. On fair evidence we might take action on what appears to be an occupational hazard, e.g., we might change from a probably carcinogenic oil to a non-carcinogenic oil in a limited environment and without too much injustice if we are wrong. But we should need very strong evidence before we made people burn a fuel in their homes that they do not like or stop smoking the cigarettes and eating the fish and sugar that they do like. In asking for very strong evidence I would, however, request emphatically that this does not imply crossing every 't' and swords with every cite, before we act.

All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the day that it appears to demand at a given time.

Who knows, asked Robert Browning, but the world may end tonight? True, but on available evidence most of us make ready to commute on the 8.30 next day.

REFERENCES


Dr John Sturrock
(Simpson Memoir Royal Infirmary)

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