Cause of occupational disease

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Abstract

The concept of causality is reviewed with special emphasis on occupational diseases. Separate approaches from the philosophical, scientific, and legal points of view are identified. There is controversy over the methodology of logical causality; inductive and deductive methods are described and reference is made to the verification or refutation approach. Application of the methods to epidemiology are reviewed.

It is likely that many diseases have multiple causes and that only a component of occupational causality can be identified in each patient. Methods of assigning such a component are discussed. The difficulties of developing an equitable compensation policy in such circumstances are reviewed. The possible benefits of proportional compensation are noted.

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Identifying the cause of disease in a population or in an individual person may be difficult and the criteria for recognition of causality have been considered by several authors. Causality is particularly important in the case of occupational diseases because prevention is usually more important than therapeutic intervention and also because many countries have developed policies for the financial support of victims of occupational diseases that are more generous than those provided for other disabled people. The purpose of this article is to review the concept of causality as it was developed in classical philosophy, developments in epidemiological thought, and the impact of legal analysis. The emphasis is on causality of occupational diseases and related policy issues.

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The nature of causality

The philosophers of early civilisations observed the natural world and proposed various explanations for what they saw. Francis Bacon in 1599 considered these to be superstitions that should be distinguished from the methodical approach of the true scientist.[1] The true scientist in his view, collects empirical observations in an unbiased way and, with an open mind, infers the cause of events from the evidence. The terms inductive and deductive inference are exemplified as follows. Very extensive epidemiological surveys on the health of cigarette smokers have been carried out. The observations induce the inference that smoking is a major cause of lung cancer. This argument in inductive logic is from particular examples to a general rule. On the other hand, if it is once accepted as a general law or rule that cigarette smoking is a cause of lung cancer, then a physician may argue from the general to the particular and make the deductive (or hypotheticodeductive) inference that smoking was the most important cause of lung cancer in his particular patient. This example illustrates deductive logic from the general to the particular.

The Scottish philosopher Hume in 1739 questioned the validity of inductive logic from repeated observation to general laws of causality.[2] In a formal logical analysis, he claimed that repeated and consistent observations of two associated events could not be used as evidence that one caused the other. This was the start of a major philosophical dispute. One school (the positivists or verificationists) claim that sufficient and increasing numbers of observations do provide a persuasive body of evidence of causality that works on a practical day to day basis. A certain framework must be used to avoid nonsensical associations. With that reservation, the positivists maintain that the inductive method offers sensible approaches to public health policy.

An opposite approach was favoured by Karl Popper who was directly opposed to all attempts to operate with the idea of inductive logic.[3] in his view, simply collecting further observations that confirmed an association between two events could not contribute in any way to the debate as to whether one caused the other. He suggested that an explanatory theory connecting two events was first developed or invented in the mind of a creative scientist. Certain deductions or predictions could be made from the theory and these could be tested. If the conclusions turned out to be acceptable then the theory held for the time being, but this did not establish the truth of the theory or even its probable truth. If the conclusions were falsified, however, then the theory itself must be false. The application of Popper's ideas to human epidemiology was first proposed by Buck.[4] This was followed by a vigorous debate in which other epidemiologists[5] emphasised the value,

from a practical and policy point of view, of careful and repeated observations on defined populations with reasoned inductive inferences as to causality.

Criteria for causality in epidemiology were proposed by Hammond in 1955,[6] Yerushalmy and Palmer in 1959,[7] and in the landmark Report of the Surgeon General's Advisory Committee on Smoking and Health in 1964.[8] A framework for drawing inductive conclusions was formulated and was used to identify cigarette smoking as the major cause of lung cancer. The framework was developed more concisely by Bradford Hill whose name is firmly associated with inferences of causality.[9] His suggestions for considering causality are widely taught and are as follows:

the strength of association
consistency specificity
the relation in time
the biological gradient
biological plausibility
coherence of the evidence
the experimental evidence
reasoning by analogy.

Hill wrote that none of these nine viewpoints could bring indisputable evidence for or against a cause and effect hypothesis and equally none could be required as essential (a *sine qua non*). What they could do, with greater or less strength, was to answer the fundamental question—is there any other way of explaining the set of facts? Was there any other answer that was more likely than cause and effect?

In a formal sense Hill's proposals are subject to the same underlying weakness of inductive logic described by Hume so many years ago. Unease about the lack of distinction between association and causation in the case of smoking and lung cancer was expressed by such a formidable statistician as R A Fisher.[10] In a series of essays, supporters of Bradford Hill's inductive method or, conversely, of Popper's deductive refutation, presented their arguments and counter arguments.[11] For the general reader it is of interest to note that opinion is divided. Neither Bradford Hill's nor Popper's method receive universal support. The idea of using the Bradford Hill criteria in a Popperian sense was suggested by MacClure[12] whereby a causal hypothesis based on inductive logic generates deductive predictions that could then be tested. If the Bradford Hill criteria failed to reject the hypothesis, then it could continue to be used for making day to day practical decisions, although fulfilment of the criteria did not, of itself, constitute formal proof.

The above analysis of causality refers essentially to single causes of disease. This is becoming uncommon in clinical medicine and the next section considers the consequences of multiple causality.

The nature of multiple causality

Occupational epidemiology examines the relation between disease in populations and concomitant toxic exposures. Clinical occupational medicine and the legal framework are more concerned with attribution of causality in the individual worker. It is increasingly clear, however, that many illnesses are the culmination of multiple causes. In a population of workers exposed to dust and to cigarette smoke, certain people will develop chronic obstructive airway disease. In some, the major cause of airway damage may be cigarette smoking and in others, it may be dust exposure. The relative importance of each will depend on the duration and concentration of exposure to dust or to cigarette smoke. It is not appropriate to treat either agent as if it were the single effective cause.

Attribution can be achieved by applying data from group epidemiological studies to the individual case. The increased risk of lung cancer in groups of workers who smoke 40 cigarettes a day is known. If a patient smokes that number, then it is not unreasonable to make the assumption that the same risk is born by that patient. When more than one cause is to be evaluated in the patient it is possible to assign the proportion of harm from each source by the use of epidemiological data including cohort or case control studies.[13] This method has been used in deriving tables of proportional risk of malignant disease caused by exposure to radiation.[14] The approach has been criticised on statistical grounds and the unmeasurable issue of individual susceptibility. No alternative method, however, seems to be available.

When two or more factors cause a disease the question may be raised as to whether one or other component made a significant contribution. A physician can reasonably hazard an informed guess whether some event such as an infection made any clinically significant difference to the inevitable train of events in a patient with untreatable malignant disease. The same value judgement of significance cannot be used to evaluate the clinical importance of a proportional estimate of causality. If cigarette smoking is estimated to account for 15% of the damage in a patient with obstructive airways disease, then this point estimate cannot be further evaluated in terms of clinical significance.

As physicians have struggled to analyse the cause of disease in their patients a parallel body of scholarship has been developed within the legal framework. To an extent the professions have remained separate and there has been relatively little common exchange. The following paragraphs summarise the idea of causality in law.

The legal framework

An extensive review of the development of legal concepts of causation, starting from the days of early Roman law was prepared by Honoré (1986).[15] A distinction is drawn between scientific theories that attempt to give an account of the way in which events happen, and legal theories of causation and remoteness of damage that have the purpose

of defining how the limits of responsibility should be fixed. Some believe that rough common sense should suffice to define legal causation but this is generally thought to be too vague or imprecise. A difference in the professional approach is provided by the example of the physician who is concerned whether coal dust caused lung disease in a miner. A lawyer traces the sequence further back to see whether some wrongful act (failure to guard against dust exposure) can be regarded as the cause. The lawyer provides a further explanation of a different type. Honoré traces the development of the philosophy of causation through Marx and Hegel and Mill.[16] Although there are many publications on the theory of legal causation Honoré proposes that the ideas around which the decisions revolve are limited to necessity, explanation, probability, and legal policy.

Necessity

This has given rise to the *equivalence theory* or essential condition (*conditio sine qua non*) theory. The crucial question is whether the event was *necessary* for the production of the harm. Each condition is necessary to the production of harm and is in that sense equivalent (equally necessary). In most cases it is impossible to apportion any particular part of the harm to any given condition. The rule, common in many countries, that independent tortfeasors, each of whom contributed to the harm, is wholely liable (*in solidum*) points to the equivalence theory. The theory was more popular in criminal cases and has fallen out of use in modern times.

Explanation

Causes of an event are those conditions that explain its occurrence. This extralegal idea is used in a negative way so that a tortfeasor is not liable for harm that is adequately explained by some other cause. This idea generates several theories. In the *efficiency* theory, various conditions can be thought of as making a quantifiable contribution to the harm. Those whose contributions come above a certain percentage are causes of the harm, and the quantification can be used as a basis for the apportionment of damage between tortfeasors.

Probability

The adequate theory—In its 19th century form, an essential condition (*conditio sine qua non*) is the adequate cause of harm if it is of the type that significantly increases the objective probability of harm, of the type actually suffered. Harm can have two or more adequate causes, and they need not be of equal probability of causing harm.

Legal policy

The theories are not strictly causal but refer to codes of law, responsibility, and a sense of equity between parties.

The very brief summary of legal theory shows clearly that the law is concerned with evaluating human conduct and not with inanimate objects. Whether asbestos causes cancer is an issue of scientific fact. Whether the industry that sells asbestos is involved in causing cancer is an issue of legal causality. A key question in decisions rests on the "but for" test. If harm would not have occurred "but for" the defendant's actions, then those actions were a cause, not necessarily the only cause of the harm.

The boundary between legal and scientific causation

The distinction between scientific and legal causation or responsibility has been described, but there are many situations where the two are almost inextricably linked. The most important is the problem of multiple causation and the questions of what constitutes a significant cause. The difficulty of applying legal concepts to scientific analysis is illustrated by the case of the asbestos worker who smokes. Would he have developed lung cancer but for the exposure to asbestos? Obviously the answer can only be given in probabilistic terms. The risk of developing lung cancer may have been much less in the absence of asbestos, but was certainly not negligible if he was a heavy smoker.

Two much quoted judgements have influenced legal thought. In Bonnington Castings *v* Wardlaw (1956),[17] a worker developed silicosis as a result of exposure to silica from a pneumatic hammer at which he worked and also silica from swing grinders at the same workplace. No dust extraction equipment was known or practicable for use with the hammer and the employers were not liable, in law, for the consequences, the health effects on the worker being covered by the regulations governing pneumoconiosis in the United Kingdom at that time. Although the swing grinders were fitted with dust extraction equipment this was not kept free from obstruction and, in this respect, the factory owners were in breach of their statutory duty. The evidence suggested that most of the dust came from the hammer but the legal argument centred on whether the dust from the grinders could have been the cause of the disease. In the House of Lords, judgement was given that the cause of the disease could have been from either source. No attempt was made to take account of contributions from two separate sources. It was held that the plaintiff did not have to show that the dust from the swing grinder was the sole or even the most substantial cause of his disease, if he could show, on balance of probabilities, that dust from that source had materially contributed to the disease. Anything that did not fall within the negligible (*de minimus*) principle would add a material contribution. The plaintiff won the case. It is evident that this is tantamount to treating the pulmonary disease as a single indivisible harm. Scientifically, however, the degree of lung damage in silicosis is closely related to the amount of dust exposure and, when two sources can be identified, then the corresponding degree of harm can be

apportioned. The worker might indeed have developed a degree of silicosis from exposure only to dust from the swing grinder but the amount of disability is likely to have been much less. A further issue is that terms such as material or significant contributions have no defined medical meaning. Courts seem to be willing to infer that a given cause makes a material contribution. This may respond to questions of legal policy but there seems to be no valid scientific support for the proposition.

A somewhat different situation occurred in McGhee *v* the National Coal Board.[18] A worker developed dermatitis due to dust from two sources. One was an unavoidable component of his employment in a brick works and was encompassed by appropriate legislation. The other was due to dust remaining on his clothing as he cycled home, and was held to be the responsibility of the employer who should have provided adequate shower facilities. The physicians who gave expert testimony did not seem to be able to quantify the harm from the two sources of dust and the damage could not be apportioned. In the absence of any such evaluation it was evident that the whole cause of the dermatitis could, in principal, have been due to dust on the workers clothing and the plaintiff won. This case is better known because of the nature of the legal tests of proof that were involved, but this will not be discussed here.

Causation and public policy

The early literature contains many reports that concluded that there was insufficient proof that asbestos exposure caused cancer or even that smoking caused cancer. Suggestive evidence, but that more research is needed, seemed to be the view of many writers. Perhaps lack of evidence was not the real issue. The ill effects of smoking are no longer seriously disputed, but the overall world sales of tobacco are rising. The certainty of causality required in making public policy depends on the potential for harm and the number of people affected. Risks and benefits must be weighed. Rothman and Poole point out that practical policy making is really a political process and, in their view scientists should not allow themselves to be drawn into this during working hours.[19] Their estimates and interpretations of causality should be as unbiased as is possible. If a given agent is thought to be a possible cause of human disease on weak evidence, it may be reasonable to prevent it being used in the first place. Removing an agent, which has been present for many years, might require much more evidence of causality before huge costs are incurred.

Causation and toxic tort litigation

The cause of occupational or environmental disease becomes an issue to be decided, not by scientists, but by a non-medical jury in the many cases that have come to be called toxic tort litigation in the United States. In a review, Foster *et al* note that the court must assess the evidence and also the credibility of expert witnesses who present the evidence. The doubtful reliability of some of the expert witnesses has created what one author has called junk science.[21] As far as the evidence itself is concerned the plaintiff in these cases must prove that the exposure, more likely than not, caused the injury. Admissible evidence, based originally on the Frye (1923)[22] rule now accepts federal rules of evidence,[23] which, among other criteria, evaluates evidence of causation after taking account of the testability of the theory, peer review, and sources of potential error.

Significant references exist in the legal literature on the use of epidemiological evidence for attribution of causality.[24] Some think that complex issues of risk assessment should not be left to trial lawyers, judges, and juries.[25]

Discussion

The central theme of this review is that identification of the cause of diseases related to occupation is difficult. Unambiguous proof is rarely obtained and is not demanded by physicians familiar with statistical and epidemiological concepts. The nature of causality is complex and the scientific and legal approaches can be quite different. Many diseases, both occupationally related and otherwise, have multiple contributing causes. The Pearson Commission thought that multiple causality would be an increasing problem in the future.[26] Causality has a very special place in occupationally related diseases because of the issues of prevention and compensation. As far as prevention is concerned it does not matter whether lung cancer in asbestos workers is due both to smoking and to asbestos. Reduction or removal of both is important. Compensation is more difficult, however, because of the practice of awarding full compensation even though only a part of the cause in an individual person is due to occupational factors. The percentage point at which an occupational component is considered sufficient to result in full compensation varies remarkably with a range from 50% in some jurisdictions to 5% in others. This has substantial economic effects on the individual people and communities concerned. Whatever the cut off point it leaves certain workers without financial consideration even though a portion of their illness or disability was occupational in origin. Other workers may be overcompensated in the sense that only a portion of their illness or disability was due to occupation. The origin of the difficulty is the imposition of a binary decision process (full compensation or no compensation) on a continuous distribution of causality. One solution might be to compensate on a sliding scale in proportion to the amount of occupational causality.[27] If disease in a worker, whether obstructive airways disease, lung cancer, or any other condition can be estimated to have an occupational component of causality of (say) 30% then that is the proportion of the disability that is compensated. Whatever the administrative difficulties of such an approach it has the merit of consistency and logic.

A more radical criticism of occupational compensation is that financial support to a disabled patient depends on causation rather than on need.[28] This generates a privileged class of disabled within the community[29] that is the more illogical when occupational causality is only part of the origin of disability in an individual subject.

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2 Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *N Engl J Med* 1976;**294**:687–90.
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