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Causation in epidemiology

Determinism versus stochasticism: in support of long coffee breaks

C C Tam, B A Lopman

Is there a recipe for the causal pie?

In his recent article describing what characterises a useful concept of causation in epidemiology,[1] Olsen provides a useful overview of the now popular component-cause model and its relevance for epidemiological research, and renews the call for discussion on how best to conceptualise causation. As he rightly points out, this is not merely of academic interest—how we view causation influences (whether consciously or not) the way in which we frame research questions and analyse and interpret epidemiological data. In recent decades, the component-cause model has been the predominant causal framework on which epidemiological research has been based, and it has been of great use for the identification of individual risk factors associated with disease and the development of the advanced statistical techniques that are now widely used for this purpose. Based on these successes, Olsen argues for the usefulness of the component-cause model over more recently propounded frameworks based on a probabilistic view of causality.[2 3] In doing so, however, we feel he is rather hasty in accepting a deterministic future for epidemiology.

The great value of the component-cause model lies in its heuristic power. A person, through exposure to various risk factors, eventually accumulates a combination of contributing exposures that constitute a "sufficient cause" and that, under identical conditions, invariably lead to disease. As visualised by Rothman and Greenland,[4] these contributing exposures, or "component causes", form the slices of a "causal pie" that, when complete, constitute a "sufficient cause". This deterministic model provides a useful framework with which to conceptualise causation in a chronological manner, from first exposure to a component cause all the way to the completion of the "causal pie" and subsequent disease. It is here, however, that the component-cause model faces its greatest problem. Epidemiology is a population science and, while it may have the ability to explain differences in disease risk or exposure distribution between groups of individuals, it cannot provide causal explanations for any single one of those individuals. That a person smokes, drinks, has a diet rich in saturated fats, and subsequently develops coronary heart disease does not demonstrably mean that any of these factors or their combination was the "cause" of their illness. Thus, the "black box" that Olsen attributes to models based on probabilistic thinking applies equally to the deterministic approach.

Olsen describes probabilistic models as minimalist and purely statistical, in which causes merely increase the probability of disease and preventable factors decrease it—"what you see is what you get". We disagree that a probabilistic model need necessarily be entirely descriptive. The causal criteria of effect magnitude, consistency, temporality, biological plausibility, and dose response are equally applicable to probabilistic frameworks, and Olsen himself acknowledges that the latter is not easily incorporated into the component-cause model. Nor do we think that the component-cause model has greater explanatory power. The fact that exposures are termed "component causes" is an admission that they only serve to increase the probability of disease and need not be a "cause" unless they are in themselves sufficient. This merely obscures the probabilistic view, as held by Parascandola and Weed, of causes as factors that increase the probability of disease and where "a sufficient cause is . . . one that raises the probability of its effect occurring to 1, and a necessary cause raises the probability from 0."[3]

In addition, Olsen perceives problems for probabilistic models in terms of risk communication:

> "At present we tell a smoker that he will increase his risk of getting lung cancer 10-fold by smoking. If he gets lung cancer from smoking, it will take decades to develop, and he may even get lung cancer, should he decide not to smoke at all."

His view that this statement "is in conflict with a common sense understanding of causation, and it is apparently not very convincing" may be true, but the statement is also realistic. To pretend that we can make more elaborate predictions of disease at the individual level is to make rather immodest claims of our understanding of causation.

Olsen is right to emphasise that our view of causation influences the way in which we conduct research. In this respect, the component-cause model has tended to individualise epidemiology, an opinion that has been extensively commented on by others.[5–8] The component-cause model was borne out of an era in which infectious diseases, with their singular causal pathways (a single infectious agent as a "necessary" factor for disease), ceased to be (for a notable minority and rather prematurely) the major concern and novel causal frameworks were necessary for the new challenges of non-infectious diseases. The realisation that this second group of diseases could be linked to a whole plethora of exposures meant that a new way of thinking was needed in which all these could be investigated and incorporated into a theory of causation. In attempting to determine why some people become diseased while others do not, epidemiological inquiries were directed at differences in lifestyle between individuals, in the belief that changes in personal behaviour would lead to a decrease in exposure and disease risk, and with the assumption that such behavioural change was possible. The premise was simple: through

their behaviour, people become exposed to a certain combination of factors that act independently or synergistically, or both, to cause disease. This framework is essentially mechanistic—it entails identifying the steps, the serial accumulation of component causes, that eventually lead to illness. We therefore contest Olsen's assertion that "[t]he component-cause model attempts to explain why" disease occurs. The model does not answer the question of *why* people become diseased, but rather *how*—what processes are involved in "causing" their illness. In fact, we argue that much of what is commonly referred to as "risk factor epidemiology" is a discipline in which the question "why" has conveniently been replaced by the more readily accessible "how". If epidemiology is to be explanatory rather than descriptive in its inquiry and proactive rather than reactive in its application, causal frameworks that will provide insights into the underlying factors that influence these biological processes are needed.

This is not to decry the successes of such epidemiological research. We now know a great deal about individual factors associated with non-infectious diseases, most notably cancer and coronary heart disease. The continued emphasis on this kind of individualistic inquiry is much disputed, however, as illustrated by a recent series of commentaries on the search for risk factors for coronary heart disease.[9–14] Moreover, the component-cause view has spawned an era of epidemiology characterised by drug dependency and the promotion of expensive therapies that will most likely be inaccessible to those in lower income countries who, by current projections, will increasingly bear the brunt of the non-infectious disease epidemics in the coming decades.[15] The contribution of risk factor epidemiology to the victimisation of individuals has also been commented on, particularly by Farmer, who speaks of the "exaggeration of personal agency"[16]—the assumption that behavioural change alone is a realistic intervention given the strong cultural, social and economic forces that are exerted on individuals.

The answer to the question of *why* some individuals become diseased while others do not then, lies further upstream and requires investigation of the factors affecting a person's daily life choices (or lack thereof). This concept was recognised by Rose, who differentiated between "the causes of cases and the causes of incidence".[7 8 17 18] Krieger has expanded upon this notion, arguing that differences in disease distribution are "mutable and embodied biological expressions"[19] of social inequity and injustice. The component-cause model is based on the premise of a sufficient cause that, all else being equal, will invariably lead to disease in individuals. The fact is that all else is hardly ever equal. Simply looking at differences between decontextualised groups of individuals can lead to what could be termed "outcome bias", a failure to recognise that disease distributions in different populations can be affected by a whole host of social factors that influence individual risk. The growing epidemic of childhood obesity provides a good example of this. Although linked to an imbalance between energy intake and expenditure in both higher and lower income countries, a full understanding of why opposite socioeconomic groups are predominantly affected in these two settings is not possible without taking into consideration factors such as local food production, global food trade, marketing of foodstuffs, and social changes leading to decreased physical activity, all of which affect choice, accessibility, and individual risk.

In arguing for a broader scope of epidemiological inquiry we do not intend to favour stochasticism over determinism. We consider this issue to be part (perhaps a small part) of a wider discussion on what epidemiologists regard as being causal and mutable and, therefore, within the scope of epidemiology. It is probable that both views will encounter problems in incorporating the multiple levels required to understand disease causation, and ultimately neither may fully succeed. We thus agree with McPherson's view that "[i]t is high time that public health stopped behaving as if one single dominant paradigm was good enough." Restricting our perspective of causation based on the past successes of a model grounded largely in a biomedical view of health will prevent us from exploring alternative frameworks and will not suffice to further our understanding of disease determinants. Developing new frameworks of causation will be crucial for expanding the boundaries of epidemiology and liberating the field from the confines of individualism. As Karhausen's Epimenides eloquently concludes, "the cause of a disease 'is the handle, so to speak, by which human beings can manipulate it.'"[2] The question now is what we view as having handles that can be manipulated. This discussion will run for as long as epidemiologists roam the Earth and so we welcome Olsen's call for a more open debate. Olsen himself has been quoted as saying that "[t]he view [of causation] we adopt has consequences which reach far beyond informal discussion during coffee breaks."[3] We can only hope for longer coffee breaks.

ACKNOWLEDGEMENTS

The authors thank Dr Laura C Rodrigues for helpful comments on the manuscript.

J Epidemiol Community Health 2003;**57**:477–478

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Accepted for publication 18 March 2003

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