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*Commentary*

# Causes in epidemiology: the jewels in the public health crown

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In this issue, McDowell pinpoints the central challenge in public health sciences—the extraction of causes from the morass of statistical, anecdotal or clinically observed associations. Understanding causes paves the way to success, whether it be cure in clinical work, or prevention and control in public health. Hippocrates told us that.[1] McDowell reminds us of the historical continuity of this central challenge and emphasizes its increasing importance in the modern era where the diseases and health problem are (or seem) more complex than those tackled by our predecessors (infectious, nutritional, occupational and environmental problems). This apparent complexity may, however, be a mirage. In the era before the germ theory of disease and an understanding of vitamins, the cause of cholera and scurvy, for example, was truly a mystery. Our predecessors still found causes and solutions. In contrast, the causes of some of the modern problems are staring us in the face (e.g. obesity), but we still cannot find or implement the solution, often because we cannot agree on causation (e.g. the deflection of interest in obesity research towards genes, away from caloric intake and expenditure).

There is a great deal of material for reflection in McDowell's article. I disagree with only one of his fundamental concepts, i.e. that risk factors are useful in explaining disease at the individual case level and may have less value at the population level. I propose the exact opposite is true. Herein, in my view, is the cause of a simmering controversy that needs to be resolved. Epidemiology is the population health science that generates, summarizes and interprets associations between exposure and outcome variables. Epidemiological risk factors are a subset of associations. Epidemiology is a population science and, therefore, can only produce knowledge about populations. The knowledge about populations produced is, therefore, sound. Interpreting that knowledge for an individual is not epidemiological science but clinical or public health judgement. Such judgements are extrapolating from the population data—no more. Moreover, they require extrapolating beyond the data.

Presently, epidemiology is often the best we have for individuals, but in future, the sciences of individuals (toxicology, pathology, microbiology, genetics, etc.) will surely rise to the occasion and produce an understanding of cause and a prediction that is not merely the aggregate probability based on a population, which is mostly seriously inaccurate, and this is possibly inevitable. In a population, the risk of disease lies between 0% and 100% and rarely will it be either 0% or 100%. In an individual risk of disease is either 0% or 100%, never in between. It is, therefore, virtually never possible for population data to be extrapolated to an individual accurately. In contrast, causal findings (and even risk factors that are not agreed to be causal) can usually be generalized globally, with only modest qualification, e.g. hypertension is a risk factor for stroke, essentially in all human populations.

Once we understand how and why a risk factor impacts on disease incidence our terminology changes, or should change, from risk factor to cause. Although this causal understanding may be applied to individuals, it rests solely on populations and is demonstrable only in population data on incidence, mortality, etc. and derived relative risks. Individual cases for chronic disease, unlike infections and other diseases where the cause is integral to the definition of the condition, seldom have demonstrable causes that are risk factors, e.g. we cannot say that the cause of a stroke in an individual with hypertension was hypertension—it may well have been something unrelated. Hypertension could even have been protective in relation to stroke in that individual.

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Causal models are necessary but insufficient (to echo an epidemiological refrain) in the art and science of causality. Models are a simplification of reality—they help us conceptualize cause, but no causal answers pop out. The counterfactual (model) has no reality (by definition); the reliable trial design that mimics the counterfactual model so yielding causality has not yet been achieved—which is why systematic reviews, meta-analyses and numerous checklists on quality control and assessment of trials are available or in development; and we now know the causal interpretation problems of the other study designs that only conform with difficulty to the counterfactual model, but will always comprise the vast bulk of public health evidence.

Causal thinking is hard and the interaction of causal factors makes it harder still. The metaphorical models of causes (lines, chains, multiple chains reflected in INUS and pies, wheels, webs—even as elaborated by Bhopal, and given prominence by McDowell—and neural networks) help us to think about causes, but do not provide answers.[1] Our statistical and mathematical modelling techniques help us quantify, but only to a limited degree interpret, associations.

Ultimately, the judgement of health scientists (not exclusively population ones) must decide: is the association an error/bias, chance, confounding or cause (including reverse cause).[1] If other explanations seem unlikely, and causality is plausible, despite their weaknesses, in my view, no approach to causal reasoning has yet improved on the guidelines as elaborated and refined by a succession of philosophers (e.g. Mills' canons) and population scientists (e.g. Alfred Evans' postulates and Bradford Hill's principles). A synthesis of these ideas is in *Concepts of Epidemiology*.[1]

As I have written before,[2] looking for causality in associations—and there is nowhere else to look within the population sciences—is like mining for gold. We mostly turn up dirt, but sometimes we find flecks of the precious substance. Occasionally, there is a nugget. Rarely, we may discover a new seam or mine. Watch out for fool's gold. Keep mining, but take care for it is a dangerous occupation, but one with potentially huge rewards.

## References

1 Bhopal RS. Cause and effect: the epidemiological approach. Ch 5. *Concepts of Epidemiology*. Oxford: OUP, 2002. (Second edition with elaborations on causal reasoning scheduled for September 2008).

2 Bhopal RS. Smoking and suicide. *Lancet* 1992;**340**:1095.