Commentary
Explanations adequate for public health

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Ian McDowell argues that public health would be well served by a deep conception of causation that helps us to understand the ‘why’ of ill-health as well as the ‘how’. I do not think anyone would disagree, but there is bound to be a variety of views on the best route to acquire such understanding.

The title of the paper implies that we need to leave the traditional risk factor model behind if we are to make progress on ‘causes, explanations and understanding’. There are plenty of examples in the public health literature of primitive analysis that has distracted rather than informed. But, does that require the classical methods of epidemiology should entirely be put on one side? I think not, but of course, as every school report says, there is room for improvement.

It is helpful to be clear about the subject of the inquisition. What do we mean by ‘risk factors’? The simplest reading would be any quantitative research that attempts to relate exposures, broadly defined, to particular health outcomes. ‘Exposures’ may be located at any level of complexity, from genome to geo-politics; the independent variables might be upstream, downstream or side-stream. Health outcomes may be measured in tissues, or individuals or populations, and amidst this variety, it is only measurement that the investigations have in common.

I doubt anyone would advocate abandoning measurement, but the proposal to move ‘from risk factors to explanation’ is more attractive if we adopt another, narrower, construction of ‘risk factor’ research. This is research that takes to heart Popper’s description of positivism as ‘the world is all surface’ and holds no brief for phenomena that cannot be measured. It is research that equates validity with reductionism—the closer a measurement is made to the molecular, in this view, the more meaningful it is. And to complete the picture, we might define risk factor research as research without lateral vision, inquiry that makes no connections with other disciplines. This is caricature, but not entirely unrepresentative, unfortunately. Research of this kind, I would agree, is unlikely to significantly enlarge our understanding of the causes of disease, and it builds a thin and unreliable foundation for public health action.

Ian McDowell’s paper draws a useful distinction between understanding (an internal alignment of information and organizing frameworks) and explanation (the external representation of understanding). The latter is more accessible than the former, so his paper rightly focuses on the question of what kinds of explanations will assist the public health endeavour. He points out that explanations are always provisional, since information accumulates and what is ‘right’ or ‘best’ at one point in time is always superseded. So, I am drawn to the standard of adequacy—what will do the job, even if it is less than ideal? If we were moving on from risk factors along the road to explanation, what would be the first, acceptable, stopping point?

In the empirical domain, explanations are tested with new data sets and alternative study designs, and an explanation that does not withstand refutation is discarded as unsatisfactory. However, this should be treated as just the start of the ‘Warrant of Fitness’ for explanations in public health. Inter-disciplinary validity is another check. This is an aspect of coherence (‘spanning several layers of causal processes’ in Ian McDowell’s words), where one would hope to see a reasonable match between the results obtained by qualitatively different research processes.

Comprehensiveness may be another test: the paper suggests that one of the criteria for judging an explanation is the ability to cover individual cases as well as patterns in populations. Is this a fair requirement for adequacy? Would we reject an explanation that satisfactorily describes the reasons for differences between populations because it fails to

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predict disease in individuals? A clinician might say yes, but I expect that most public health practitioners would demur.

Explanations may also be checked on their theoretical pedigree. Does the explanation fit within an internally consistent, multi-level, information-rich theory of causation? As with the previous ‘criterion’, this is an ideal, but perhaps too stringent a test of adequacy of explanation. For example, Doll and Hill’s explanation for the post-World War 2 epidemic of lung cancer in the UK had no theoretical basis, but formed the basis for effective intervention.

What about a utilitarian basis for judging explanations? History, by my reading, does not support the view that epidemiological thinking succeeded with infectious disease but struggled to gain traction against non-infectious disease. Classical risk factor studies on smoking, salt and high blood pressure have provided substrate for spectacularly successful public health interventions against chronic disease. There is a wealth of examples elsewhere. For instance, successes in injury (e.g. road crash deaths) and nutritional diseases (e.g. iodine deficiency) followed from old-style agent-host-environment-type analyses. But, risk factor explanations, like other kinds of explanations, tend to be adequate up to a point. Inevitably, there are aspects of public health problems that require a new analysis, a different mindset and a fresh set of disciplinary spectacles. The social patterning of injury, for example, is not adequately explained by the same types of investigations as those that showed very satisfactorily the relation between vehicle speed and injury severity.

Perhaps, the most important guide to the adequacy of an explanation is the question that prompted the investigation in the first place. If the question is whether lower speed limits will reduce the frequency of severe injuries due to road crashes, then a risk factor-type explanation will probably do the job very well. If the question is how to reduce social and ethnic inequalities in the incidence of injury, then different kinds of explanation will be required.

Finally, to argue, as Ian McDowell does, that epidemiology now struggles because present-day problems have no specific causes is to overlook the primacy of disease definition. Specific causes are a product of nosology, which is a moving feast: miners’ phthisis gave way to silicosis and tuberculosis, and likewise, the commonest lethal malignancy world-wide—cigarette cancer—will, in due course, I am sure, gain a place in the International Classification of Diseases.