Towards a realistic and relevant public health: the challenges of useful simplification

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Ian McDowell discusses many of the limitations of the risk factor paradigm in public health and epidemiology, building and amplifying on prior work.1–3 As discussed by McDowell, an important and perhaps unintended consequence of the focus on risk factors is that it has shifted epidemiologic inquiry from understanding causes to estimating ‘independent’ associations. This has impoverished epidemiology and has constrained not only the methods that we use but also the way in which we formulate research questions and even the research questions that we ask. For example, many epidemiologic investigations routinely formulate research questions in terms of identifying the presence of an ‘independent association’ of a given factor with an outcome, rather than on understanding the causes of an outcome or explaining (in McDowell’s sense) differences in health between people or between groups. In many ways, the methods that we use (in terms of both study designs and analytical approaches) which largely focus on enhancing our ability to estimate ‘independent effects’ constrain the questions that we ask and the answers that we obtain. The task is to develop an alternative approach that is rigorous, feasible and informative for public health. Unfortunately, as is often the case, this is easier to say than do. Three challenges that I will briefly discuss in turn are the challenge of multiple levels, the challenge of systems and the tension between full explanation and identification of intervention points.

McDowell notes that ‘explaining public health matters involves many levels’. The need to consider multiple level of organization, from genes to society, when studying the causes of ill health, has been repeatedly noted by epidemiologists and public health researchers in recent years.4–6 But, of course, it would be impossible to study all possible levels all the time. The art of research is the ability to determine which levels are likely to be the most relevant in answering a particular research question. And then features of these levels need to be operationalized so that they can be studied empirically. This means that grandiose multilevel conceptual schemes need to be simplified so that they are tractable. The trick is to make them simple enough so that they can be studied empirically but not so simple that we get the wrong answer. Ultimately, this is what scientific inquiry is all about.

An important aspect is understanding the connections between levels, or more specifically, how factors at one level may affect variations at another level.7 For example, even when investigating the causes of ill-health in individuals, factors defined at higher levels of organization may need to be considered. This is because higher level factors may be causally related to the health of individuals or may interact with individual-level factors. For example, living in an unequal society may operate as a stressor for a given individual that may in turn cause health problems, or inequality may interact with genes causing disease in susceptible individuals. Similarly, the group-level health outcome, or the disease rate, results from the joint and interacting effects of both individual and higher level factors. For this reason, I would quibble with McDowell’s statement that ‘causes of cases are typically drawn from lower levels of investigation while causes of patterns of incidence typically invoke higher level phenomena’. Both lower and higher level factors may be important for understanding the causes of disease in individuals and the causes of higher rates of disease in a particular population. Geoffrey Rose8 is often cited to support the notion that ‘the causes of cases’ may be
different from the ‘causes of incidence’. But a careful reading of Rose reveals that what Rose means by the ‘causes of cases’ is actually the causes of differences between individuals within a population (for whom the higher or population-level factor is invariant). In this case, it is fair to say that the individual-level differences are caused exclusively by individual level factors because all individuals are exposed to the same higher level factors so these cannot explain variation among them. But in the more general sense, both disease in an individual and rates in a population result from the combined effects of lower and higher level factors.

McDowell discusses common metaphors used in public health and epidemiology to characterize causation and proposes an interesting alternative. It is notable that several of the features of the concentric circle model he proposes are characteristics of systems. These include, for example, the presence of dynamic interactions and feedback loops within and between levels, the possibility that alternative pathways may lead to the same outcome, and the role of chance and stochastic processes. Although calls for systems approaches to public health problems are by no means new,9 the growth of complex systems methodologies in biology and the social sciences has stimulated new thinking not only into theoretical aspects of applications of systems approaches to population health but also concrete applications of these methods to answer specific research questions in population health.10–13 Moving beyond theoretical discussions to concrete applications remains an important challenge.

McDowell devotes a considerable part of his piece to discuss the meaning of explanation and differentiating explanation from understanding and from the mere quantification of associations in the data. I found this discussion particularly insightful and useful. It is worth noting, however, that full explanation in the sense of fully specifying the complex causal processes that lead to adverse health may not always be the objective in public health or epidemiologic research. In fact, many times we may be perfectly satisfied with identifying effective points of intervention, i.e. factors that we can intervene on to improve health, even if we do not fully understand the complex processes involved in causation. In defense of the much maligned risk factor or black box epidemiology,14 the desire to identify intervention points has been an important force behind the focus on identifying ‘independent’ associations of factors with disease. And indeed the risk factor approach has led to the identification of factors on which we can intervene on to improve health. Of course, one could argue that ignoring the complex causation may result in identification of ineffective interventions or may highlight more proximal antecedents rather than the more distal (and often social and politically contentious) determinants. These are valid critiques and they highlight the need for system approaches to public health in which the effects of interventions at different levels can be evaluated in the context of more realistic dynamic interactions between levels and factors. Ultimately, because the goal of public health is to improve health, the key challenge is figuring out how to identify the interventions or policies that are most likely to improve the health of populations in the absence of complete and full understanding of all the complex processes involved. But this ultimately is what makes our work as public health researchers both intellectually interesting and (hopefully) relevant to society. McDowell’s thoughtful commentary stimulates us to continue our work towards this goal.

References