Commentary: Modelling multiple pathways to explain social inequalities in health and mortality

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Investigations into socioeconomic inequalities in health and mortality have primarily been carried out in the US and in Europe,1–3 therefore reports from other parts of the world are a welcome addition to the effort to explicate pathways linking social structure to health and mortality. Research on social inequalities in Korea is particularly interesting as it has undergone rapid industrialization, with the GDP per head increasing 100-fold in <40 years.4 In this issue of the International Journal of Epidemiology, Khang and colleagues show socioeconomic differentials in mortality in South Korea to be better explained by early life exposures compared with biological risk factors, health behaviours, and psychosocial factors.5 The authors point out that it is the latter that explain more of the social gradient in mortality in the western countries. They argue that this discrepancy in the relative importance of different pathways might lie in the differential cause-specific structure of mortality in Korea. The authors recognize that this hypothesis needs to be further tested in age-specific analyses in order to control for cohort effects, before firm conclusions about the relative importance of different pathways can be drawn. In this commentary, I suggest that another aspect that requires further effort is attention to the modelling of interrelationships between variables.

The examination of pathways explaining socioeconomic differentials in mortality is not straightforward. Consider a simple example with three variables: an exposure (X), here socioeconomic position; an outcome (Y), here mortality, and a third related variable (A). These variables could be associated with each other in at least four different ways (Figure 1), described below; each of which would require a different statistical treatment to assess the impact of X and A on Y.

1. The relationship between X and Y could be mediated by an intervening causal variable, A. This could either be a single variable (A) or a series of variables (A1, A2, A3, etc). Thus the exposure would have a ‘direct’ effect (pathway XY) on the outcome and (or) an ‘indirect’ effect (pathway XA + AY), mediated through its effect on A.

2. The relationship between X and Y could be moderated by a variable, A. This implies that the strength of the relationship between the exposure (X) and the outcome (Y) would depend upon the moderator (or effect modifier), as this variable specifies the conditions under which X will produce Y.

3. The third variable, A could be a confounder. This happens when A is itself associated with both the exposure (X) and the outcome (Y). Inadequate modelling of a confounder will bias the estimate of the association between the exposure and the outcome. This is particularly true when there is no real association between X and Y.

4. Finally, the third variable could be an antecedent variable, in that it occurs earlier in time than either the exposure or the outcome. Thus A could be causally antecedent to both X, Y and other mediators being considered in the analysis. Treating an antecedent variable as a mediator is incorrect as here it is X that is mediating the effect of A on Y.

In effect, pathway variables are mediators and require the following four conditions to be met: X must be significantly associated with A, the hypothesized mediator (see Figure 1) A must be significantly associated with Y, X must be significantly
associated with Y, and the impact of X on Y must be attenuated after controlling for A. Khang and colleagues consider the following pathway variables: biological risk factors (BMI, blood pressure, cholesterol, and glucose), health behaviours (alcohol, smoking, and exercise), psychosocial factors (depression, perceived stress, and marital status) and early life exposures (education, and adult height). Tables 1 and 2 in the paper test conditions 1–3. Results show that all the biological risk factors fail condition 1 (Table 2) and the measure of cholesterol further fails condition 2 (Table 1). The more serious problem in their analyses is the attempt to test condition 2 with the early life exposure factors, height and education (Table 2). This translates to testing whether income (in adulthood) ‘causes’ education and height. Both height, as a proxy for childhood conditions, and education are antecedent to income and mortality (scenario 4, Table 1) and not mediators of the relationship (pathway variables) between income and mortality.

Education has previously been shown to have both a ‘direct’ effect and an ‘indirect’ effect, mediated through its effect on later measures of socioeconomic position, on health. Treating it as a predictor and the other pathway variables, is likely to seriously bias the results. In a recent paper, Son hypothesizes that the stronger effect of education on mortality in Korea compared with western countries could be linked to a strong effect of education on ‘occupational position and material well being’ in Korea. This ‘antecedent’ role of education, linking it to future life chances is likely to be particularly manifest in all countries undergoing rapid economic expansion. Education would thus be a measure of both early life circumstances, as the opportunities available to an individual are likely to be patterned by parental social class, and future socioeconomic trajectory.

At this stage, it is worth highlighting the important role that clearly defined conceptual models play when investigating mechanisms leading to socioeconomic differentials in mortality. Attention to the temporal order in a sequential causal framework is essential in estimating the importance of different pathways. Furthermore, the relative importance of different pathways linking social position to health can only be assessed if these pathways are modelled simultaneously. Papers dealing with multiple pathways at the same time are rare. This, despite the fact that most conceptual models of the social determinants of health are both multilevel (hierarchic) and multiprocess, in that multiple pathways are hypothesized.

Future work on pathways would also benefit from closer attention to the interrelationships among the pathway variables themselves. For example, it is probable that psychosocial factors have both a direct and an indirect effect, mediated through health behaviours in particular, on mortality. There is considerable research evidence on the clustering of poor health behaviours and psychosocial characteristics in socially disadvantaged groups. It would be interesting to test whether the psychosocial variables examined here (depression, perceived stress, and marital status) also have an association with the health behaviours examined (alcohol, smoking, and exercise). Thus, some of the effect of depression, for instance, on mortality could be mediated through its effect on health behaviours.

Notwithstanding the limitations, the present study is of importance to social epidemiology. Similar results on social inequalities in different cultural contexts hint at universal processes, whereas differences provide further insight into causal mechanisms; in both cases international research is crucial to the understanding of mechanisms underlying social inequalities.

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References

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