Is the study of genetic propensities within the remit of health inequalities research?

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In his viewpoint, Bann claims that social epidemiologists and others working in health inequalities research should concern themselves with cognition and genetic factors.[1] But do differences in health between groups defined on the basis of cognition or genes constitute health inequity?

There are many definitions of health (in)equity, including that of Whitehead (cited in the viewpoint [1]), which, based on a review of the literature, concluded that “health differences determined by [biological variation] would not normally be classified as inequities in health”.[2] It is arguable whether this definition needs to be updated in view of more recent research.

Based on the Braveman and Ruskin definition of equity in health as “the absence of systematic disparities in health (or in the major social determinants of health) between social groups who have different levels of underlying social advantage/disadvantage”[3], Bann argues that this is applicable to genetic propensity for disease because the disease may lead to social disadvantage. This is an argument of reverse causation: social position is determined by health rather than the other way round. Traditionally, social epidemiology has been thought of as the study of the social determinants of the distribution of health across populations[4], but it seems reasonable to extend this to improving our understanding of the social patterning of disease and, as such, would include reverse causation.

The idea that genetic predisposition might influence health, and this in turn impact on social position, differs from the suggestion by Mackenbach that socioeconomic differences in health may be confounded by cognitive or genetic factors.[5] As potential confounders these factors should be important to social epidemiologists; accurate estimation of the effect of social determinants on health outcomes in regression models requires adjustment for all such important confounders. Unfortunately, it is rare to find datasets that include all the confounders (including genetic confounders) required. Randomisation is seen as a means of overcoming the influence of confounding variables when examining causal relationships, but not all studies are open to randomisation. It is possible that the risk, or effect, of confounding can be minimised using appropriate natural experimental designs.[6]

The Commission on the Social Determinants of Health provided another definition of health inequity as “systematic differences in health [that] are judged to be avoidable by reasonable action”.[7] While gene-environment interactions mean that environmental changes may benefit those of certain genetic predispositions, the changes involved (such as removing or reducing access or exposure to harmful substances) will generally improve population health and reduce socioeconomic inequalities in health. It is difficult to conceive of an intervention of this kind that would be undertaken purely to reduce differentials between genetically defined groups.

The lack of specific interventions to address differences according to cognition or a genetic predisposition to ill health makes it difficult to see these as axes of health inequity. However, their roles as potential confounders, or as causes of socioeconomic position with this relationship mediated by health (that is, reverse causation), means that they should remain of interest in health inequalities research. They are likely to form part of a much bigger picture; our understanding of the complex nature of the processes underlying the generation of health inequalities is far from complete.[8]

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