Commentary: Personality and health inequality: inconclusive evidence for an indirect hypothesis

John Gallacher

Nabi et al. explore the ‘indirect selection’ hypothesis for health inequality, linking personality to relative all cause and cardiovascular mortality using data from the GAZEL study. There have been previous studies linking personality constructs to mortality and linking quasi-personality constructs to health inequality but none linking psychological status to health inequality. Instead, the focus was on psychological characteristics that, at the inception of the study, were thought likely risk factors for vascular and neoplastic disease. The scales used were the Bortner Type A, the neurotic hostility and reactive hostility scores from the Buss-Durkhee Hostility Inventory.

The strengths of this study include that mortality was the outcome and that complete follow-up was achieved. Limitations lie in the small number of deaths in women, the range of socioeconomic position (SEP) indicators available and the type of psychological assessment used.

Psychological assessment did not cover the classic ‘big five’ personality dimensions of openness, conscientiousness, extraversion, agreeableness and neuroticism. Instead, the focus was on psychological characteristics that, at the inception of the study, were thought likely risk factors for vascular and neoplastic disease. The scales used were the Bortner Type A, the neurotic hostility and reactive hostility scores from the Buss-Durkhee Hostility Inventory.
and six scales from the Grossarth-Maticek and Eysenck Personality-Stress Inventory (GEPSE). These concepts may be more informatively described as reflecting psychological status than personality. Depression was also assessed using the CES-D, but was considered as a covariate in the analysis.

Indicators of SEP were education, occupation grade, income and father’s social class. Apart from education these are occupationally related. The equivocal findings in women may be due to the determinants of health inequalities in women being expressed more broadly than the occupationally and educationally related indicators used here.4 Evidence for sex differences in the mechanisms contributing to inequality also comes from within the study insofar as father’s social class was related to female mortality but not to male mortality. Alternatively, the null findings may be due to there being only 75 deaths occurring in women.

Nevertheless, the interesting finding of this study is that in men crude measures of how we typically think, feel and act are related to inequalities in mortality. To object to this conclusion on the grounds that not all the psychological scores were related to mortality, that many of the decrements in relative mortality were evidently negligible and that no formal tests of significance for larger effects of personality were presented would be overly critical. Where there was an association with mortality, a consistent pattern of inequality was demonstrated and some moderate effects were found, the largest being a 44% difference in male relative cardiovascular mortality for income associated GEPSE coronary prone behaviour.

The public health impact of these moderate effects may not be large but the point is whether the indirect selection hypothesis is supported. That various negative cognitive and emotional states such as hostility are associated with mortality and with SEP does not explain why. Using mortality as the outcome and an implied 100% follow-up of those providing psychological data is important as it removes objections of reverse causality and of selection bias in detection and diagnosis of the outcome.5 However, the extent to which psychological status is the cause or the result of SEP cannot be resolved in these data. Given this caveat, the data are consistent with the indirect selection hypothesis but the evidence is far from conclusive. Given the complexity of the many weak associations likely to underlie these findings, it is difficult to avoid the conclusion that progress lies in ‘the genes’. Not that a complete explanation will be bio-molecular, but relating genetic information on personal characteristics, such as hostility, to indices of social mobility and also to health will provide a more certain starting point for causal inference.

There are at least two ‘epigenetic’ issues also relevant here. The first is the rise of the meritocracy in a technological society. In a meritocracy the indirect selection hypothesis implies an inevitable health inequality between the elite and the remainder. This is due to the elite enjoying the rewards of their efforts (affluence, power and health) and being poorly motivated to relinquish those benefits in order to reduce inequality.6 In our technological society the elite have hitherto been understood to be the cognitively more able. To add the psychologically more stable to the criteria for elite membership provides a further challenge to the reduction of health inequality. The extent to which the indirect selection hypothesis is supported is the extent to which meritocracy is inherently unsuited to the promotion of health equality.

The second is the need for large cost-effective cohort studies.7 The prohibitive cost of traditional cohort methodology inevitably leads to few, underpowered and comparatively blunt cohorts. The result is inadequately tested hypotheses. Nabi et al.1 should be commended for making the most of their opportunity, but it remains, that for reasons of limited exposure assessment and power, data such as theirs cannot provide a definitive test of the indirect selection of personality on health inequality hypothesis. Cohort studies are the eyes and ears of epidemiology, which is the basic science of public health. More cost-efficient methodology is required that allows the full potential the cohort design to be realized. Only through these methodological developments can we anticipate real progress in addressing complex hypotheses such as indirect selection.

References