Commentary: How does socioeconomic disadvantage during childhood damage health in adulthood? Testing psychosocial pathways

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The multi-national comparative study by Power and colleagues\textsuperscript{1} provides strong evidence for a significant correlation between low socioeconomic status (SES) and health risk in adulthood. As expected, adult SES was related to obesity and smoking behaviours. However, low childhood SES was also significantly related to adult health risk, even after adjusting for contemporaneous adult SES. The evidence supports a long-term association between childhood SES and adult health that is not simply due to the life course continuity of low SES. To obtain such broadly consistent findings in seven samples from six countries is impressive, more so, because of the many factors that work against this outcome (e.g. historical, cultural, sampling, and measurement differences across studies).

The challenge is now to explain the reasons for these disparities, ideally by mapping the developmental pathways leading from childhood socioeconomic adversity to poor adult health. Currently little is known about mediating pathways, mainly because most datasets documenting health inequalities have limited data to explore possible explanations for these inequalities.\textsuperscript{2} The key limiting factor has been the lack of longitudinal birth cohort studies that contain detailed life history data from birth to adulthood and direct physiological assessments of health. To date, empirical work has been limited by several factors. First, the majority of research on the psychosocial correlates of physical health has been cross-sectional. In contrast, longitudinal methods and within-subject comparisons offer a stronger strategy for inferring developmental influences. Second, many available longitudinal studies have suffered high attrition rates, selectively losing unhealthy participants. Third, reported associations between psychosocial risk factors (e.g. psychiatric symptoms) and health outcomes (e.g. obesity) often neglect to control for confounding factors (e.g. medical illness). Fourth, and more generally, epidemiological studies with good health data tend to have weak psychosocial data and vice versa. Gold standard measurement of psychosocial variables is paramount, as are direct measures of physical health uncontaminated by self-reported biases.

Childhood SES is a distal risk factor. Distal factors are important because they set the scene for more proximal risk factors to exert their influence. There are several mechanisms by which proximal factors might act to explain the long-term relation between childhood SES and adult health. These include delivery of class-biased health care during children’s formative years; differences related to social-class in health promoting parenting practices; and stressors related to social class in childhood that might alter biological systems. The importance of the psychosocial stress pathway has been hotly debated.\textsuperscript{3,4} Here we suggest three classes of candidate ‘stress’ mediators as a way of illustrating the rationale, data, and methods required to strengthen future tests of psychosocial mediation of the gradient between childhood SES and adult health.

(i) Psychologically stressful family environments interfere with homeostatic processes and can cause health problems in children.\textsuperscript{5} At least three domains appear relevant. The first concerns the poor mental health of parents, which can compromise their ability to optimally provide for the child.

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The second domain is related to family circumstances and includes variables associated with socioeconomic conditions of the household that generate stress (e.g. parental loss, residential turnover, caregiver changes). The third domain refers to family functioning and encompasses the quality of parenting, especially emotional aspects of the parent-child relationship, conflict, and abuse in the home. Data on all three domains are necessary to test whether stressful family conditions mediate the influence of childhood SES on adult health.

(ii) Early-emerging psychiatric disorders and related personality vulnerabilities of Negative Affectivity might mediate the effect of childhood SES on adult health. Depression and hostility are two of the most important mental health contributors to physical illness in adults. Research has shown that low childhood SES is related to early onset and persistent cases of depression and conduct disorder (of which hostility is a cardinal feature). Likewise, low childhood SES is related to developing personality traits of Negative Affectivity (e.g. stress reactivity, hostility), and these traits increase adolescents’ involvement in health-risk behaviours such as smoking. Longitudinal data collected repeatedly since childhood on psychiatric symptoms and personality traits are needed to test whether these psychological vulnerabilities account for the effect of childhood SES on adult health.

(iii) Lack of access to social capital, including social support, might explain the association between childhood SES and adult health. It is widely known that social affiliation is linked to physical health. Moreover, there is some evidence that social support (both emotional and instrumental) is differentially distributed by social class. Data over the life course are needed to test whether the lack of access to resources and limited provision of social support in childhood accounts for the effect of childhood SES on adult health.

These types of mediational analyses need to control for health selection effects (e.g. the possibility that differences in newborn health contribute to greater stress exposure) as well as for other psychological factors known to be related to both SES and adult health; for example, new longitudinal evidence identifies the possibility that differences in childhood general intelligence may account, in part, for social class inequalities in health. Genetic data should also be integrated into developmental analyses wherever possible because genes can moderate sensitivity to environmental stress. However, there may be some reluctance among social epidemiologists to do this, either because information about which genes are relevant is currently limited, or because of a more general unease about genetics per se.

Life-course epidemiology is throwing up intriguing new findings about social inequalities in health. The new findings offer considerable challenges and opportunities. In particular, findings about the enduring and cumulative effects of childhood socioeconomic position on adult health invite epidemiologists; (i) to evaluate the biological plausibility of such long-lasting effects, (ii) to nominate and measure new variables that may account for these developmental links across the life course, and (iii) to test factors that may account for the enormous heterogeneity in the association between childhood socioeconomic position and adult health.

References