Is an unhealthy lifestyle more harmful for poor people?

A socioeconomic gradient has been observed in morbidity and mortality in the UK and elsewhere.[1,2] That people with socioeconomic deprivation have a higher prevalence of many unhealthy lifestyle factors such as smoking and obesity than do wealthy people is also well known. However, these standard risk factors do not fully explain the gradient.[2] The same pattern of results was observed in epigenetics research, showing that the rate of epigenetic (biological) ageing between people varies, and is faster among those with low socioeconomic positions than among those with high socioeconomic positions; however, this finding was not fully attributable to their poorer risk factor profile.[3] These findings show that important mechanisms associated with the socioeconomic gradient in health are still unknown.

In *The Lancet Public Health*, Hamish Foster and colleagues[4] explore one possible explanation. They hypothesise that deprived populations are disproportionately affected by the harmful effects of unhealthy lifestyles. It is assumed that overall increased vulnerability and the other factors associated with deprivation, such as psychosocial stress, more extreme or risky lifestyle, and poorer access to health services, can all intensify the harmful effects of the risk factors. Furthermore, the investigators introduced emerging risk factors, such as television viewing and sleep duration, which might additionally increase premature mortality and cardiovascular event risk among the socioeconomically disadvantaged groups.

A large sample size, such as that of the UK Biobank, which includes almost 330 000 participants, was necessary for this research. Reliable testing of subgroup differences requires a sufficient number of outcome events at each level of socioeconomic deprivation, indicated here by the Townsend deprivation index linked to residential addresses. Foster and colleagues divided the study population into three groups on the basis of lifestyle risk score (high, intermediate, and low), depending on how many of the risk factors (long or short sleep duration, excessive television viewing time, smoking, high alcohol consumption, a diet characterised by high red meat consumption and low oily fish consumption, and low physical activity) the participant had, all measured between 2006 and 2010. All-cause mortality was followed up until 2016.

The study showed that in the most affluent socioeconomic group, poor lifestyle score was associated with an increase in risk of all-cause mortality (adjusted hazard ratio 1·65 [95% CI 1·25–2·19]) and an increase in risk of cardiovascular disease mortality (1·93 [1·16–3·20]). The corresponding adjusted hazard ratios were much higher among individuals in the most deprived group (2·47 [95% CI 2·04–3·00] for all-cause mortality and 3·36 [2·36–4·76] for cardiovascular disease mortality). These findings are consistent with the vulnerability hypothesis, suggesting that the same risk factors are more harmful for people living in deprived circumstances than for people living in affluent areas.

Notably, when Foster and colleagues repeated the analyses with the outcome including incident cardiovascular disease, the lifestyle score associations among the socioeconomic groups did not differ. This finding might be due to, for example, ascertainment bias resulting from a higher rate of undiagnosed disease in individuals from the most deprived group than in those from the least deprived group. However, an association with mortality but not with the development of disease might also mean that the greatest impact of social deprivation is on the late stage of the disease process. Similarly, in the British Whitehall study,[5] one of the pioneering investigations of the socioeconomic gradient in health, socioeconomic deprivation had a substantial role in the progression of cardiometabolic disease, whereas clinical vascular risk factors were important predictors of the incidence in all socioeconomic groups. One candidate for a mechanism, psychosocial stress, through acute and chronic stress responses, has been shown to increase the risk of cardiovascular events in people with pre-existing atherosclerosis, but such triggering effects were not similarly harmful among those free of atherosclerotic burden.[6] Given the greater atherosclerotic burden in deprived populations than in affluent populations, these observations are consistent with the current findings from the UK Biobank.

It remains unclear how important a role the two emerging risk factors—television viewing and sleep—actually have in the current findings. Does television viewing merely mark a sedentary lifestyle or is there something particularly damaging about televisions?[7] Would sitting and reading a book be equally harmful?

One explanation for the excess risk associated with time spent watching television is increased intake of energy from snacks during this activity.[7] The associations found between long and short sleep duration and cardiovascular health are similarly unclear, because extreme sleep habits might at the same time be a proxy for socioeconomic deprivation and a consequence of an underlying disease or risky health behaviour.[8]

The Townsend deprivation index reflects socioeconomic inequalities (or socioeconomic structures), which operate outside the individual's control, as the causes of the causes behind more proximal factors such as lifestyle.[9] Residential address is already included in some summary risk scores, such as the UK QRISK3, to improve the identification of individuals at a high risk of having a heart attack or stroke within the next 10 years.[10] However, evidence of the added value of including sitting times and sleep patterns in lifestyle risk scores is still scarce.

Healthy ageing is an achievable goal for society as a whole because it is already experienced by individuals in the highest socioeconomic groups. Studies such as that of Foster and colleagues are important, because they have the potential to increase our understanding of the reasons behind these inequalities. Identification of the novel contributing factors in these studies could add to the knowledge on health-related mechanisms more generally, and thus benefit us all.

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