

Income inequality and mortality: why are they related?

Income inequality goes hand in hand with underinvestment in human resources

See pp 996, 999, 1004, 1013

The long held belief that household income is an important indicator of risk of death has recently received strong support from a series of large prospective studies.^{1,2} Income inequality within a population has also been suggested to be an important determinant of population mortality. In a cross national comparison, Rodgers found associations between income inequality and three mortality indicators—infant mortality, life expectancy at birth, and life expectancy at age 5—after taking overall gross national product into account.³ Several replications of this, across both a wide range of countries and within industrialised nations alone, using a variety of health indicators, have appeared.⁴⁻⁷ These studies have related income inequality to infant mortality,⁴ life expectancy,⁵ height,⁶ and morbidity,⁷ with a consistent finding that the less equitable the income distribution in a country, the less favourable the health outcome.

In this week's issue of the *BMJ*, two studies relate income inequality between states in the United States to mortality rates within these states. Kennedy and colleagues (p 1004) show that greater income inequality is associated with higher mortality from several broad causes of death, although taking levels of poverty and smoking prevalence into account attenuates these associations.⁸ Kaplan *et al* find associations between level of inequality and mortality in both 1980 and 1990 (p 999), with trends in mortality differences between states over this decade being inconsistently related to changes in income inequality.⁹

In Britain, reliable data on income inequality by area are not readily available. Also in this issue, Ben-Shlomo *et al* (p 1013) have used the variation in small area deprivation scores within local authority areas in Britain as their indicator of socioeconomic inequality¹⁰ and demonstrate that both overall level of deprivation and variation in deprivation contribute to an area's mortality experience, with overall level of deprivation being of somewhat greater explanatory power.

The existence of these associations seems secure, but what do they mean? They seem to show that inequality per se is bad for national health, whatever the absolute material standards of living within a country. The reasons for this have been framed by the leading proponent of the income inequality hypothesis in explicitly psychological terms: "the evidence strongly suggests that the health effects of income distribution involve comparative social and cognitive processes, rather than the direct effects of material standards."⁵

The implication is that the psychological effects of being low down the social ladder have detrimental health effects, whatever the actual material conditions of life. Biological plausibility can be sought in human and animal psychoneuroendocrinological studies, but this rarely goes beyond analogy. When

the major causes of death are considered—cardiovascular diseases and cancers—it seems odd to find apparently instantaneous changes in mortality in response to changes in income inequality,^{5,9} since these diseases are ones in which causal exposures are thought to act for many years before death. Studies based on individuals have borne out the expectation that cumulative measures of lifetime social circumstances—such as wealth,¹¹ family assets,¹² lifetime earnings,¹ and occupational careers¹³—are the crucial socioeconomic predictors of longevity. Short term changes in income inequality will have only a moderate influence on such lifetime exposure.

Changes in absolute income levels and changes in mortality over time have not been strongly associated,¹⁴ in part because material factors will produce changes in mortality with very different latency periods depending on the cause of death. This makes interpreting changes in all cause mortality problematic. Increases in permanent but not transitory income are associated with declines in relative mortality,¹⁵ in keeping with the evidence on the importance of cumulative socio-environmental insults.

Early exposures will have long lasting effects

In general, however, secular declines in mortality have been remarkably resistant to the influence of even dramatic social changes. This reflects the fact that such changes cannot retrospectively alter what has gone before, so the influence of such social assets as education, welfare coverage, and infrastructural improvement are not reversed in the short term. The well established benefits of preschool programmes in deprived areas on the social and personal functioning of the recipients of these programmes when they are adults¹⁶ would not be expected to disappear instantly when the socially progressive administrations which established them are replaced. Similarly, there are likely to be long lasting biological assets, reflected in secular increases in height together with the improvements in health during youth and early adulthood that were seen for the cohorts who are now elderly. These assets, too, are not lost during periods of increasing social polarisation.

Considering time trends in mortality provides a framework for viewing the effects of income inequality. In Britain and the United States, the continuing secular decline in mortality was not reversed by increases in income inequality seen earlier in this century^{17,18} and more recently.¹⁹ This is well illustrated by the analysis of Kaplan *et al*, in which income inequality in the United States increased from 1980 to 1990 while overall mortality fell.⁹ The only group for which the continued fall in mortality does not survive increases in inequality is the young

adults, for whom recent trends in mortality have not been favourable,⁵ particularly for residents of deprived areas.²⁰ It is, of course, the people within this age group who die of causes—in particular, accidents and violence—which are not the outcome of long term biological processes and which will plausibly respond rapidly to increasing social disruption. Indeed, homicide is the cause of death found to be most strongly related to both income inequality indices used by Kennedy *et al.*⁸ Similarly, more rapid responses to increasing inequality and social polarisation may be expected for psychological distress, general wellbeing, and morbidity than for chronic disease mortality. The finding of a relative deterioration in health status of civil servants anticipating job change and non-employment in comparison with those remaining in stable employment²¹ provides an example of this. Inequality may make people miserable long before it kills them.

The apparently overly rapid response of mortality to changes in income distribution may have various explanations. Firstly, relatively small absolute changes in mortality are involved, with increases in life expectancy of about two years being seen in the period covered by the analyses of Wilkinson⁵ and Kaplan,⁹ while 30 year increases, unrelated to any systematic change in income distribution,^{17 18} have been seen over the century. Major determinants of variations in mortality between countries or between areas within countries need not be the same as the major determinants of overall population mortality.

Secondly, those countries that are now experiencing the largest increases in income inequality are precisely those that have systematically underinvested in human resources for many years. The countries and governmental units which are currently those experiencing the greatest increases in inequality will contain the populations whose social and biological assets have been most undermined.

Increases in income inequality go hand in hand with underinvestment, which will reap poor health outcomes in the future. In the United States, poor investment in education and low expenditure on medical care is seen in the states with the most unequal income distribution.⁹ Similarly, low birth weight is commoner in the states with the greatest inequalities, with the possible long term detrimental influences on adult health that go with this. Cross nationally, higher levels of both social expenditure and taxation as a proportion of gross domestic product are associated with longer life expectancy, lower maternal mortality, and a smaller proportion of low birthweight deliveries.²² The relative and even absolute deterioration in social and biological assets that is occurring in increasingly unequal societies can be expected to produce poor health outcomes in the future.

The only coherent argument against redistributive social policies is that they hinder overall economic growth. Here it is supposed that the greater rewards offered to the entrepreneurially successful makes them even more successful and in turn drives overall economic growth, which, through the “trickle down” effect, ultimately benefits the poor. Cross national comparisons, however, show the reverse: if anything, countries with greater income inequalities have shown lower levels of economic growth.²³ The current government, however, continues to pay no heed to the growing evidence^{5 18 20 21} that increasing income inequality is bad for the economy, bad for crime rates, bad for people’s working lives, bad for infrastructural development, and bad for health—in both the short and long term.

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Potential transmission of BSE via medicinal products

Patients can be reassured that measures are in place to reduce risk

The identification of 10 cases of Creutzfeldt-Jakob disease, which seem to represent a new variant,¹ and the announcement by the Spongiform Encephalopathy Advisory Committee (SEAC) on 20 March that these cases could be linked to exposure to bovine spongiform encephalopathy, have caused great concern. Patients are worried about the risks of developing Creutzfeldt-Jakob disease not only from eating beef but also from medicinal products of bovine origin and are looking to doctors, pharmacists, and pharmaceutical companies for reassurance.

The risk of transmission of bovine spongiform encephalopathy via medicinal products depends on whether the infective agent is a human pathogen and on the level of exposure to the

agent. To date, no epidemiological link has been made between any spongiform encephalopathy that is transmissible in animals and human disease, despite exposure of humans to the scrapie agent for at least 200 years. This suggests that the risk of transmission to humans is small.²

Measures aimed at minimising exposure to transmissible spongiform encephalopathies via medicinal products were introduced soon after the report of the Southwood Committee in 1988, in guidelines for manufacturers issued by Britain's Committee on Safety of Medicines in 1989, and essentially adopted by the European Committee for Proprietary Medicinal Products in 1992.³ Materials were to be sourced from cattle aged under 6 months from countries free of bovine