

## Counting early deaths due to socioeconomic inequality



Published Online  
December 5, 2019  
[https://doi.org/10.1016/S2468-2667\(19\)30242-7](https://doi.org/10.1016/S2468-2667(19)30242-7)  
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Descriptive studies of inequality in England include classic works such as Charles Booth's poverty maps of London,<sup>1</sup> and the Black report.<sup>2</sup> The Black report powerfully linked measures of deprivation to health outcomes. Similar analyses are now widely available for many other countries.<sup>3</sup> Statistical summary measures, such as the slope index of inequality, are routinely used to inform public health practice and policy in England.<sup>4</sup> Therefore, more descriptive studies of inequality might not seem needed and the focus now might seem to be on what to do about this inequality.

An Article by Dan Lewer and colleagues<sup>5</sup> challenges that view by presenting an innovative analysis of the effect of inequality on premature mortality. Records of all deaths in England of people younger than 75 years from 2003–18 were used to quantify premature mortality attributable to socioeconomic inequality (MASI). The counterfactual was the expected mortality if the whole country had had the same mortality as the least deprived decile at the time. MASI results were calculated for all deaths but also by age and sex, by cause, and for local administrative areas, and as numbers of deaths or as proportions. A similar approach using life-table analyses was used to estimate where were possible years of life lost to inequality (YLLI).

The MASI is, in effect, the proportion of early mortality or the number of early deaths that would not have happened if inequality had somehow been eliminated. This is a standard epidemiological approach equivalent to the population attributable fraction used to assess the burden of specific risk factors, such as smoking, or air pollution.<sup>6</sup> This approach has not before been applied comprehensively to the complex set of causes that generate health inequalities.

It is no surprise that the study produced some huge numbers. Over the whole study period, 877 082 premature deaths (about a third of all such deaths) were ascribed to inequality, that is one every 10 min. The life table analyses produced similar results with an average of 1.2 YLLI over the period, out of a total measure of loss of life expectancy due to deaths before 75 years of 3.3 years.

Using the MASI statistic as a proportion shows that, for individual age and sex groups, the peak relative effect of inequality is in childhood for both sexes and in working age men: for men aged 40–44 years, as

much as 77% of premature mortality is attributable to inequality. Relative measures of MASI by condition also tell a compelling story. More than half of premature mortality was due to inequality for chronic obstructive pulmonary disease (COPD), liver diseases, and for flu and pneumonia. The absolute measures of MASI will perhaps be of most interest to policy makers, and these were greatest for cardiovascular diseases, respiratory cancers, COPD, and digestive cancers. For cancers generally, the effect of inequality was much less, except for sites such as larynx, lung, and mouth.

The pattern of results by cause strongly highlights the effect of known intermediate risk factors such as smoking, alcohol use, air pollution, and occupational risks, which we know mediate the effect of social and economic deprivation on mortality. Most striking, however, was the intensity of the effect of inequality on certain less common causes of death. The highest relative measures were seen for tuberculosis, drug use disorders including opioids, HIV, and viral hepatitis; for these causes, 69–80% of premature deaths were attributable to inequality. This pattern speaks strongly to the inclusion health agenda in which subgroups of the population have multiple disadvantages with devastating effects on their health.

Results for men and women were broadly similar. Findings for local areas are supported by an online interactive map and show the expected stark differences by geography with some whole localities in prosperous regions doing better than the least deprived national decile.

This study was not intended to be explanatory and the assumed counterfactual of levelling up the entire population to the status of the least deprived decile is hardly a realistic policy objective. The most that could be aimed for is a gradual reduction of relative inequalities while improving overall health, and even that is challenging to achieve.<sup>7</sup> Another issue with the approach as a guide to action is that it is not exactly the inequality that causes premature mortality: it is the poverty, unemployment, poor health literacy, bad environments, and other disadvantages that are the true causes.

The results<sup>5</sup> are more interesting and valuable than might have been expected given the extent of past

work. They show the utility of carefully quantifying and conveying the effect of public health threats by person, time, place, and cause. Descriptive epidemiological studies are less common in the published literature than are analytical ones (the Global Burden of Disease being a notable exception) but are more often used to support policy and practice. Unlike analytical studies, the results of descriptive studies are not by their nature generalisable to other settings, but the methods can often be replicated elsewhere.

Lewer and colleagues suggest that their results could be used directly to guide the allocation of resources. More likely is that the findings will be used to support advocacy for an epidemiologically informed approach to health inequalities. The results by cause support the need for progressive action to address unhealthy behaviours, and the need for additional targeted action for inclusion health groups. The results by local area show a clear need for universal place-based measures to tackle underlying causes as recommended by the Marmot review.<sup>8</sup>

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I declare no competing interests.

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