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Health inequalities

**Anne Case
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Executive summary

- **There are marked differences in life expectancy between the regions of England, between the countries in the UK, and between the UK and other wealthy countries.** Within England, prior to the COVID-19 pandemic, life expectancy at birth was more than two years higher in London and the South East than in the North of England. For decades, life expectancy in Scotland has been two years lower than in other constituent countries. Throughout the UK, gains in life expectancy slowed considerably after 2010, proximately attributable to a slowdown in progress against cardiovascular disease. After many years of gains in life expectancy, something has gone seriously wrong. In 2019, the UK ranked 24th in the OECD in life expectancy, behind all other English-speaking countries (with the exception of the US) and nearly all countries of Western Europe.
- **Gains in life expectancy have been stronger for men than for women in the UK over the past 50 years.** Throughout the world, women live longer than men. However, since 1970, men's life expectancy gains have outpaced those of women in most wealthy countries. The female–male gap in the UK, which stood at 6.3 years in 1970, had fallen to 3.7 years by 2018.
- **In England, where much measurement has been done, there are large differences in mortality rates between less and more educated adults, and between less and more deprived places.** Progress in closing these gaps for adults largely stalled after 2010, while gaps in early childhood between less and more deprived areas continued to close.
- **The austerity measures enacted after the financial crisis of 2008 not only harmed many people directly and unequally, through reductions in public services, but may also have played a role in the anaemic reduction in mortality rates observed for adults after 2010.** Evidence for this claim, however, is mixed. Gains in life expectancy did indeed slow in the UK after 2010, concurrent with the onset of austerity. However, mortality decline in midlife was slower in the UK than in other rich countries in the decades prior to austerity (1990–2009). Moreover, life expectancy changes across the rich countries of Europe correlate *positively* with the level of austerity incurred. Portugal, Spain, Ireland, Italy and Greece, with harsh austerity, continued to see rapid gains in life expectancy, while those in Great Britain faltered.
- **Prescriptions on policy to improve the health of those left behind will need to wait for more and better data.** It is not possible to understand the upstream mechanisms responsible for health outcomes using geographic indices of multiple deprivation. Such indices, which transform and combine social and economic health inputs at a small-area level, are not helpful in understanding the mechanisms linking health to income, class, employment and education. Release of matched census and mortality records holds out hope of real progress.

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- **The most promising approach to improving population health is to continue to focus on the health and well-being of children.** This is true, not only for the children themselves, but to weaken the intergenerational transmission of deprivation. Birth cohort studies allow documentation of why and how some children enter adulthood on lower health trajectories, which place them at disadvantage throughout their lives. It is distressing that some important early-childhood indicators changed little between 1958 and 2000, in spite of improvements in the economy and markers of population health.
- **COVID-19 has changed mortality patterns in the UK.** The epidemic severely disrupted the normal provision of healthcare, and changed the epidemiological environment and the risks associated with work. Even so, gradients in mortality by local-area deprivation did not change in the first wave of the epidemic. However, according to provisional ONS data, death rates by ethnicity changed sharply in a way that was markedly different from pre-existing patterns. These outcomes echo findings in the US, where educational gradients in mortality were largely preserved while racial and ethnic differences changed distinctly.

1. Overview on health inequalities

Place- and class-based differences in mortality in Britain have been documented, and their causes debated, for 150 years. One of the motivations for the introduction of the National Health Service in Britain in 1948 was to redress the injustice of health inequalities thought to be caused by the need to pay for healthcare. That health inequalities remained after 1948 – and exist today in all rich countries, whatever the nature of their healthcare provision – was initially a source of puzzlement, one that led researchers and policymakers to focus less on healthcare and more on 'upstream' social factors, such as education, income and income inequality, racism, the operation of the labour market, and social security, tax and transfer systems.

In this chapter, we take stock of what is known about current-day health inequalities in the UK, and their potential upstream causes. While an understanding of the role of healthcare and of upstream causes is paramount for policy purposes, our understanding is far from complete. Health evolves over the 'life course' – from the womb, through infancy and childhood, into adulthood and old age. Moreover, it evolves jointly with the upstream factors whose importance we would want to quantify: on average, less-healthy mothers have less-healthy babies; children in poor physical or mental health complete less education, and are at risk for poorer health in adulthood; adults with less education and poorer health are in a weaker position to compete for good jobs; and adults who fall into poor health are often forced by their health to leave the labour force. These are channels that run from poor physical and mental health to lower economic and social status. At the same time, those with lower incomes may not have the resources to move away from environmental hazards, or the luxury to leave jobs that grind them down, or the freedom not to worry about how the rent will get paid. These are channels that run from lower economic and social status to physical and mental health.

If we take a snapshot of the health of a population at a point in time, we find that those in poor health have less education and lower incomes. They live with more crime and pollution at their doorsteps. Their interactions with the healthcare system may be less beneficial. They die at younger ages. While these relationships have been documented many times over, they do not tell us whether lower incomes, or less education, or lower social status caused poor health, or whether these factors resulted from poor health, or – as seems likely – both sets of channels are open, with economic and social status more important in affecting health at some points in the life course, and health affecting economic and social position at other points.

Policymakers need something more concrete to guide them than an explanation that 'this is complicated', and there are many researchers who have resolved questions of causality to their own satisfaction. We could simply declare that we know social position is the most important driver determining health, and work to change social relationships. Equally simple is to declare that low income, or income inequality, is the root cause of poor health, and work to redistribute resources, or that health *in utero* is determinant, and work to reduce smoking and increase prenatal care among pregnant people. There may be social value in doing all of these things, but whether they will lead to lower health inequalities remains an open question. Just as was the case when the National Health Service was introduced.

More promising is a life-course approach that follows individuals from cradle to grave, and observes for each person the joint evolution of health status and economic and social status. This kind of life-course modelling cannot settle causality but it allows us to identify chains of reactions to, say, a step-down in health status, or in economic status, at different points in life. Some countries (e.g. Sweden and Denmark) allow researchers, behind firewalls, to combine administrative data for all residents on health at birth, schooling, labour supply, earnings, transfers, interactions with the healthcare system and mortality. This provides a rich picture of health and its relationship to education and economic status. Other countries have, over time, built (relatively) small but rich longitudinal data sets that allow researchers to track health, economic and social status from midlife through to death. Britain is fortunate to have four birth cohort studies (1946, 1958, 1970, 2000) that follow study members from birth to the present, which has facilitated important life-course analyses for Britain. We will return to the British birth cohort studies in what follows.

We begin with a discussion of health inequalities – what they are and why they matter. We draw on data and analyses provided by the Office for National Statistics (ONS) and the British literature to examine health trends and levels in the UK. We present differences between regions of England and between countries of the UK. Much can be learned by examining similarities and differences in health outcomes between wealthy countries, and we present comparisons with other rich nations. The lion's share of evidence available on health inequalities within the UK comes from England and Wales, although we will note geographical differences between the constituent countries of the UK. We then turn to the British cohort studies, and what they have taught us about the joint evolution of health status and economic and social status. Throughout, we will appeal for more and better data with which to quantify health inequalities in the service of better understanding their origins.

Several elements important to the understanding of health inequalities are not covered in this chapter, but are provided in excellent commentaries included in the IFS Deaton Review.

Propper (2022) focuses on healthcare – a factor important both for the role it plays in health outcomes and because it is something over which government has at least some control. Are there inequalities and inequities ('unfair' inequalities) in the provision or utilisation of healthcare? As Propper notes, these are difficult to measure, in no small part because people vary in their needs for care and in their preferences toward care. People of lower status (measured in a variety of ways) are less healthy on average and use more healthcare. They generally present for care at more advanced stages of illness and experience poorer healthcare outcomes. Propper and colleagues (Cookson et al., 2015), surveying what is known about inequalities in healthcare in England, find little evidence of a pro-rich bias in the supply of NHS family doctors (GPs) per capita, or in healthcare utilisation or expenditure. (An exception is in knee and hip replacements where, conditional on need, people in the most deprived areas of England are only a third as likely to have

a joint replaced.) They identify pro-rich biases in the quality of healthcare provided, including waiting times, but note that the differences between rich and poor are not large in general, with waiting times falling for many procedures in the 2000s. Large and systematic differences are found between the rich and poor in preventive procedures – dental check-ups, mammograms, cervical and colorectal cancer screenings. To the extent that poorer people lack access to preventive care, this is a clear inequity that may contribute to unequal outcomes. However, to the extent that poorer people choose to forgo screenings that could save their lives, we need to look upstream to understand their decision to do so; as with choices about lifestyle, the fact that choices are (at least in part) voluntary does not mean that they are not conditioned on factors outside of people's control and, thus, of ethical concern. Data on privately funded healthcare, which is important in elective hospital care, dental services, and preventive care, are not widely available for analysis, and would allow a more complete look at healthcare inequalities.

Currie (2022) discusses a range of economic factors that could drive the morbidity and mortality differences found between rich and poor. She highlights the role played by safety nets and public health infrastructure, and places special emphasis on the importance of safety nets in reducing inequalities in childhood health. Currie suggests that perhaps 'the main reason that life expectancy is low in the US relative to other rich countries is not because there is more income inequality per se, but because the US lacks an adequate social safety net'. We return below to the question of whether austerity measures imposed following the financial crisis of 2008 can explain health inequalities we find in Britain today.

Fancourt and Steptoe (2022) discuss the importance of early-life mental health and adult psychosocial health. These are important determinants of well-being in their own right, and act as determinants of physical health in adulthood. Positive affect, optimism and purpose in life, Fancourt and Steptoe argue, are malleable, and strategies to improve positive well-being are widely available. Such strategies may prove an antidote to poor economic and social status. Childhood experiences are important, but their effects on life-course health are far from immutable.

2. What are health inequalities and why do they matter?

History and philosophy

In any account of human well-being, health looms large. Without being alive, income and wealth mean nothing, and disabilities and morbidity restrict the capability to live the sort of lives that people have reason to value. No account of inequalities can ignore inequalities in health, and health is intertwined with the other topics in the IFS Deaton Review, most obviously in early child development, in families, in education, in the labour market, in the consideration of why inequalities matter, and in the design of policies such as transfers, benefits and taxes. At the same time, while it is impossible to think about well-being without thinking about health, we should never think about health on its own, without considering other things that matter. Health, education and material well-being all contribute to the capability to live a good life.

Amartya Sen (2002) writes:

Health equity cannot be concerned only with health, seen in isolation. Rather it must come to grips with the larger issue of fairness and justice in social arrangements, including economic allocations, paying appropriate attention to the role of health in human life and freedom. Health equity is most certainly not

just about the distribution of *health*, not to mention the even narrower focus on the distribution of *health care*. Indeed, health equity as a consideration has an enormously wide reach and relevance.

Yet the literatures on health inequality and on material inequality are usually separate, and are largely contained within different academic disciplines. They rarely rise to the challenge in Sen's quote.

Material inequality is often taken to refer to the dispersion of income, consumption and wealth across the population as a whole. Health inequality usually refers not to the dispersion of health across the population – though there is a small literature on the dispersion over people of years of life lived – but to the differences in health across subgroups of the population according to social class, sex, education, income or wealth, race or ethnicity, or geography. It is closer to studies of material inequality across groups, referred to as *horizontal* inequality as opposed to the *vertical* inequality that dominates discussions of inequality in economics, measured by indexes such as the Gini coefficient. Even so, health inequalities are often defined and measured across the same groups that are relevant for studies of horizontal material inequality, defined by geography, education or class.

Income inequalities are much better defined than are health inequalities. Income has a cardinal scale, but health does not. A famous example is Sheps's paradox of the living and the dead (Sheps (1958); see also Preston and Taubman (1994) for an excellent exposition). When, as often happens, mortality is falling over time, but more so for higher social classes, one might reasonably claim that health inequalities – measured as the ratio of mortality rates – are increasing within a context of generally improving health. But in the same circumstances, if we work with survival rates instead of mortality rates, health inequalities – the ratios of survival rates – can be improving. In general, because of the scale problem, we can never make claims that health inequalities are rising or falling, as opposed to more specific claims about ratios of or differences in mortality rates or in life expectancies. This paradox is relevant in Britain today, where Mackenbach et al. (2016) have shown that *differences* in mortality rates between the less and more educated are falling while, at the same time, *ratios* of mortality rates are rising.

An important empirical insight of the literature is that health inequalities often track material and other inequalities, so that disadvantages and deprivations tend to cluster in the same people or groups of people. People who are materially poor, or poorly educated, or who live in deprived communities tend to have poor health. As a result, studying inequality in a single measure will usually understate the extent of deprivation, or lack of capabilities, and miss the full range of benefits to the fortunate.

There is no general rule here, and the most obvious and important exception is for women, who do worse than men in many dimensions, but have longer lives than men; indeed, they have lower mortality rates than men at all ages, even before birth, a regularity that holds around the world.

It is sometimes taken as obvious that health differences in morbidity and mortality across groups are unjust, and that policy must be designed to remove them. We are used to living with the fact that goods are unequally distributed, that some people have more income or wealth than others, but many people balk at the idea that life and death should be unequally distributed. Even in the US, where most Americans accept the idea that much of healthcare is provided by the market, no one suggested early in the pandemic that the supply of vaccines or COVID-related medical care be rationed through the price system.

Whether or not an inequality is unjust depends, to a degree, on how it comes about. Beyond that, policies to address health inequalities need to understand the mechanisms that generate them, and that understanding is far from complete and remains controversial. Attaining a consensus is not helped by the current and often siloed research streams in economics, epidemiology and demography.

Again, Sen (2002) argues that health inequalities are unjust when they can be attributed to inadequate social arrangements.

If, for example, there are gross inequalities in health achievement, which arise not from irremediable health preconditions, but from a lack of economic policy or social reform or political engagement, then the fact of health inequality would be materially relevant.

If we can identify the faulty social or economic arrangements, we are on firm ethical ground in trying to eliminate them, and our understanding of the mechanisms gives us a starting point for policy. Here are two examples.

In the US, there is good evidence that some of the gap between black and white mortality can be traced to hospitals, which are currently run on something like an *apartheid* basis. Housing is largely segregated by race, much of which can be explained by explicitly racist policies such as redlining. As a result, in places where the population is largely African American, hospitals have patients who are largely black, and conversely for places with few blacks. The hospitals with a preponderance of black patients are of lower quality on multiple measures – qualifications of doctors, and the range of equipment, pharmaceuticals and procedures available. Minorities are more likely to live in poorer neighbourhoods and, among other things, run higher risks of morbidity and mortality from pollution. There is no good reason why American living arrangements should be like this, so there is a clear injustice. This chain from redlining to residential segregation to healthcare and health differences is a good example of systemic racism.

In the UK, several writers have blamed the recent stagnation of life expectancy, especially in more deprived areas, on the austerity programme of the 2010s (Marmot et al., 2020a). As a means of balancing the public purse, austerity is a policy that redistributes upwards, by asking the less well-off to bear the brunt of correction, by cutting services or transfers, and sparing the better-off people who otherwise would have to pay more tax. If the link from austerity to health were clearly established, there would be a clear line to a faulty social arrangement, and a clear line to policy. However, a reversal of austerity policy alone may not have much effect on mortality trends. There was little relationship between the degree of austerity and mortality across European countries following the financial crisis of 2007–08. In addition, as we examine below, the erosion of progress in lowering midlife mortality in England and Wales long pre-dates the financial crisis. This is not to say that the lack of progress does not have economic roots, but rather that we need to examine mechanisms that have been at work over the past several decades.

That not all health inequalities are unjust or in need of remediation is illustrated by the superior health outcomes of women, usually attributed to biological differences. We do not seek to eliminate these, for example, by giving men preference in life-saving treatment. (Note that not everyone agrees with this, and it is empirically true that the gap in health by sex is not constant; in the past half-century, the gap between men and women has been closing, at least in the rich

countries. If we understood the connections, some of those temporal changes might point to unjust arrangements, but we do not know.)

Health gaps are sometimes useful to identify targets or opportunities for improvement. The Swiss live for two years longer on average than the British (United Nations, 2019), and it is hard to see why this is an immutable fact of nature, though it is much easier to see the difficulty of expecting Kenya to make up the 15-year gap with Britain. But differences in health and in life expectancy between otherwise similar countries are of great relevance for studying health inequalities; just why are Scottish lives two years shorter than English lives, or why are American lives shorter than either one?

Health inequalities: causes

Britain has a long history of documenting and studying social differences in health; it is well laid out by Macintyre (1997), on whose account we draw.

In the first half of the 19th century, the Industrial Revolution brought workers and their families from the countryside to unsanitary cities. The immiserating material and health conditions faced by those workers were documented in numerous classic studies, most notably the work of Friedrich Engels and of Edwin Chadwick, eventually inspiring a sanitary revolution. Starting with the census of 1851, which was used to calculate populations at risk, death rates were calculated by geography and by occupation. These tabulations identified high death rates in more deprived places, and led to tabulations of mortality according to an occupation-based scale of social class.

The documentation of place- and class-based differences in mortality in Britain is therefore of long standing. So, too, are competing explanations of why they exist and what policies are needed to combat them, or whether any policies are needed at all. Are such differences the result of faulty social arrangements, or are they, in some sense, natural, just as it is 'natural' that some people earn more than others?

Macintyre (1997) shows that the main battle lines on causes and policies drawn in the 19th century have, in broad terms, remained in place. In modern terms, we might distinguish four strands of explanation. They are not mutually exclusive. They are:

- *genetics*, or at least predisposition of preferences;
- *reverse causation* (sometimes called selection), in which position is determined by health, not health by position;
- *environmental factors*, so that the first account (genetics) and the third account (environment) are versions of the nature-versus-nurture argument; and
- *health-related behaviours*.

The role of genetics was argued in the 19th century by eugenicists, that people came from distinct 'stocks', and that those from poor stock were inferior in a range of domains, including criminality (this was the age of transportation of the 'criminal stain' to Australia), fecklessness, poverty, bad habits and ill health. Eugenicist policy was to discourage such people from propagating or to transport them to Australia.

Echoes of such views are still heard today. Economists and psychologists talk about time preference or self-control, that those who are born with patience and the ability to delay gratification will be more likely to get educated, to earn higher incomes, to move up the social scale, to take care of their health, and to live to a great age. That whole groups lack such virtues is frequently argued in the conservative literature – for example, by Charles Murray, who blamed black culture for the disasters that befell the black community in the US in the 1970s and who is today arguing that the same is true of the US white working class (Murray, 1984, 2012). There is limited scope for public policy here, the victims have only themselves to blame. Indeed, providing help to this group sorely lacking in industriousness, Murray argues, is responsible for an unhealthy dependency on the state.

Reverse causation argues that the poor health of a group with some characteristic – low social status – is because poor health causes that characteristic, at least in part. That healthier people make better marriages, for example, is institutionalised in many societies. More relevant today are the effects of health on education and on earnings. Poor health in childhood and in young adulthood limits educational opportunities, though once educational qualifications have been earned they are largely impervious to illness. That is not so with earnings or income, which are often severely affected by sickness or approaching death. It is unclear to us why this channel should be controversial, often denounced by those who work on the health gradient. Of course, it does not preclude income causing health, though it is surely not a good idea to rely solely on one mechanism and ignore others.

That **environmental factors** matter for health is clearly correct. Pollution is an obvious and clear example. Position at work – or more generally status – has been argued to be important in the famous Whitehall studies. Hospitals and schools matter for health, though neither the underlying mechanisms by which they matter, nor the extent to which they matter, is settled. Conditions in early childhood, or even in the womb, can have lifelong effects on health. These conditions are nearly all amenable to policy. These issues will be discussed further below, as well as in the chapter on early childhood (Cattan et al., 2022).

Health-related behaviours are clearly important, with smoking the lead case. After the dangers of smoking became clear in the 1960s, smoking, which had previously been positively associated with social status, reversed direction, so that the health burden of smoking, including lung cancer and heart disease, is now heavily borne by poorer and less educated people. Binge drinking – although not moderate drinking – and obesity are now more prevalent among those of lower social status.

While there is no question that these behaviours affect health – and the enormous effects of smoking have been particularly well documented – that is not enough to settle the ethical or political questions. In particular, conditions of life may predispose people to these behaviours. To the extent that time preference, self-control and the ability to delay gratification are inculcated at an early age by a subgroup of parents, some children are placed on healthier trajectories (Moilanen, Shaw and Fitzpatrick, 2010; Flouri, Midouhas and Joshi, 2014; Storksen et al., 2015).

Choices themselves are conditioned by the environment, and we may not want to hold people fully responsible for them, which takes us back to the environmental story, at least in part. Tobacco may be a relatively inexpensive pleasure for those who cannot afford to go to resorts, or nice restaurants, or personal trainers. If behaviours are freely chosen, it is parallel to Milton Friedman's account of income inequalities, that they are what people want, and we should leave them alone. It can be argued that high taxes on tobacco, well in excess of those required to cover

the externalities of smoking, are paternalistic and hurt poor people in the name of helping them help themselves, while the money is used for things that rich people like, such as lower taxes. (However, this argument relies on behaviours being freely chosen; when substances are as addictive as nicotine, laissez-faire solutions lose a good deal of their appeal.)

One line of argument admits the importance of healthcare, but argues that the parts of it that are important are widely available, and attributes the rest of health inequalities to freely chosen behaviours that we collectively have no business interfering with. That we recognise such arguments does not mean we agree with them, but we think it is important that all of these strands of the argument be recognised.

Challenges for empirical analysis

Data deficits. Causal stories on health outcomes should allow for multiple mechanisms and try to disentangle them – for example, by allowing for the effect of ill health on earnings when studying the effect of earnings on health, or allowing for potential effects of childhood circumstance on adult health. Mackenbach (2019, 180) soberly notes that

after decades of research we still do not know what the relative importance is [of direct causation, reverse causation, and confounding due to selection on personal characteristics during social mobility].

But if we are far from a full account, this is in part because of a lack of data. Surveys often focus on health with rudimentary economic measures, or else focus on income and wealth but have scant information on health. Mortality tends to occur long after the conditioning events that we are interested in, and linking deaths to conditions in life is often partial, involving samples that are sometimes too small, particularly when we are interested in specific causes of death or uncommon conditions. Merging census data with death records would be close to ideal, but most countries currently do this only for a sample of the census. Raj Chetty and his collaborators have merged the complete US tax records with information on death, permitting a rich analysis relating income, place and mortality (Chetty et al., 2016). But many large administered data sets are 'skinny' in that they lack a great deal of the ancillary measures that have been shown to be important in the literature. With the tax records, for example, there is no record of either education or race, two of the central constructs in the American literature on health inequalities. These data problems become even harder when we are trying to link early-life events with adult health or mortality.

Beyond the individual records, there are studies, from the 19th century on, looking at places as the unit of analysis – US states, or local authority areas in Britain. Much has been learned from those studies, but they are not a substitute for studying individuals. Poor people do not necessarily live in poor places; indeed, if inequality is higher in richer places – such as New York or London – it is possible for most poor people to live in wealthy places. It is easy for writers to slip from one to the other, substituting 'poor people' for 'poor places', particularly when it is the former that are of interest, but the latter for which we have data. Ecological fallacies are not uncommon; for example, in the US, people for whom religion is important in their lives have better health than people for whom it is not important. Yet cities and states where religion is more important have the worst health outcomes on a range of measures.

The literature on health inequalities in Britain differs in some ways from the related literature in the United States, where a major focus is on differences in health by race and ethnicity, particularly the fact that mortality among black Americans is higher than mortality among

whites, a difference that goes back as far as data exist. By contrast, Hispanics, many of whom are immigrants to the US, or the children of immigrants, have *higher* life expectancies than whites. Immigrants as a group have better health outcomes than native-born Americans (Hendi and Ho, 2021). There is similar evidence on the health of immigrants in the UK, who, like those in the US, tend to do relatively well (Fernández-Reino, 2020). COVID-19 has widened the gap between blacks and whites in the US, but has partially closed the gap between Hispanics and whites (Andrasfay and Goldman, 2021; Masters, Aron and Woolf, 2022). In the US, death certificates report race and ethnicity, while in Britain they do not, although the Office for National Statistics (ONS) has recently merged death records into the 2011 Census, and has released preliminary estimates of all-cause and COVID-19 mortality rates by ethnicity, which we discuss below.

Much of the American literature concerns educational differences in mortality, starting with Kitigawa and Hauser (1968). This topic has been less studied in Britain, in part because until recently a much smaller fraction of the British population had advanced education – see again Macintyre (1997) – and in part because education is not recorded on British death records. The US has been recording education on death certificates since 1989 (with a few states lagging), and this information, together with information from mortality supplements to nationally representative economic and health surveys, has permitted a large body of work on differences in mortality by race, ethnicity and education.

In particular, the stagnation and reversal of *adult life expectancy* (from age 25) in the US in recent years is sharply different for those with and without a four-year college degree (a BA). Life expectancy for those without a degree has fallen for most of a decade, widening the educational gap in adult life expectancy. At the same time, gaps between blacks and whites have been shrinking, so that gaps by education conditional on race are now much larger than gaps by race conditional on education (Sasson and Hayward, 2019; Case and Deaton, 2021). The widening mortality gaps by education in the US can be traced back to suicides, alcoholic liver disease and drug overdoses, as well as a stalling of progress in cardiovascular disease mortality, all of which are worse for those without a college diploma. It is impossible to understand current adverse changes in health for the US population without looking at the widening gaps by education.

The US also has a number of mortality follow-ups to larger surveys that typically have information on a range of economic and sociodemographic characteristics, allowing an analysis of mortality in relation to both income and education. These studies typically show that education and income each predict mortality conditional on the other; see, for example, Elo and Preston (1996) and Olfson et al. (2021). None of those allows us to sort out the full mechanisms, and in particular to consider feedbacks from sickness to income or from earlier health to education. The British birth cohort studies are much better suited to this task.

Educational differences in mortality in the UK can be studied using the merge of death certificates into the 1% sample of the census, and these data have been used by Mackenbach and co-authors, who have also carried out parallel studies for many European countries (Mackenbach et al., 2016 and 2018; Mackenbach, 2019), discussed below. More could be learned from the complete merge of mortality records and the 2011 Census and, in time, the 2021 Census for England and Wales.

Multiple deprivation indexes. For more than a century, health inequalities in Britain were described according to social class, with social classes defined according to occupations. This was in sharp contrast to most of the work in the US, which focused on race, ethnicity, income and education. Though perhaps the contrast is more apparent than real. If we think of inequalities in terms of social respect and whether people are treated as equals, and not simply in terms of

income, wealth or education, the gulf between the upper and lower classes in Britain shares much with the gulf between white and black in the US.

ONS no longer uses the social class measure; many occupational codes are missing on death certificates, and there have been long-running issues with the lowest social classes depopulating, and becoming increasingly negatively selected by health. Instead, ONS tabulates health outcomes according to an index of multiple deprivation (IMD), generated at a fine level of geography by the Ministry of Housing, Communities and Local Government.² Although clearly useful for some purposes, these indexes present many challenges for studying health inequalities. For policy purposes, as we emphasised earlier, we need to identify causal mechanisms that take us back to labour markets, pollution, social support, healthcare and other aspects of the way that society is structured. For these purposes, the deprivation index – a blend of components that includes income, employment, education, crime, environment and even health – is over-aggregated. (Components of the index, such as health, can be removed, although often are not.) The components are themselves transformed versions of the original variables and as such are unlikely to match the variables that we should like to work with.

The IMDs, at a fine level of geography, also contain imputed data. To quote McLennan et al. (2019, 79), 'The technique of shrinkage estimation (in other words empirical Bayesian estimation) is used to "borrow strength" from larger areas to avoid creating unreliable small area data'. Bayesian estimation and borrowing strength are words for what might crudely be described as making up data. At the same time, the indexes suffer from the geographical aggregation that we discussed earlier so that, for example, we do not know whether the poorest people live in the poorest places or whether the least educated people live in the least educated areas. And, even with all the care in the world, it is possible to slip from what we have to what we wish we had. For example, the latest update of the Marmot Review is fastidious in the main text (Marmot et al., 2020a), but the summary on the Health Foundation's website says that life expectancy for the poorest 10% of women has declined over the last decade,³ which might be true, but it is not known. What is known is that, at least before the (one-year) rise in life expectancy in 2019, life expectancy fell for women in the areas in the bottom tenth of the deprivation distribution in the decade up to 2018. (That this is true for women and not for men reflects the multi-year superior performance of male over female life expectancy.)

With this caveat, we note that the deprivation indexes are what we currently have, and they have been the basis for much useful work, discussed below. We begin by describing health and health inequalities in the UK prior to the arrival of COVID-19. The COVID epidemic continues to unfold, and we examine the extent to which the virus has changed trends in health inequalities later in this chapter. We end by examining health over the life course, using the British cohort study data.

3. Health inequalities: mortality and morbidity

Mortality in a pre-COVID world

Mortality across countries and within the UK. There are longstanding disparities in expected years of life within the UK. Figure 1 shows life expectancy at birth for men (left panel) and women

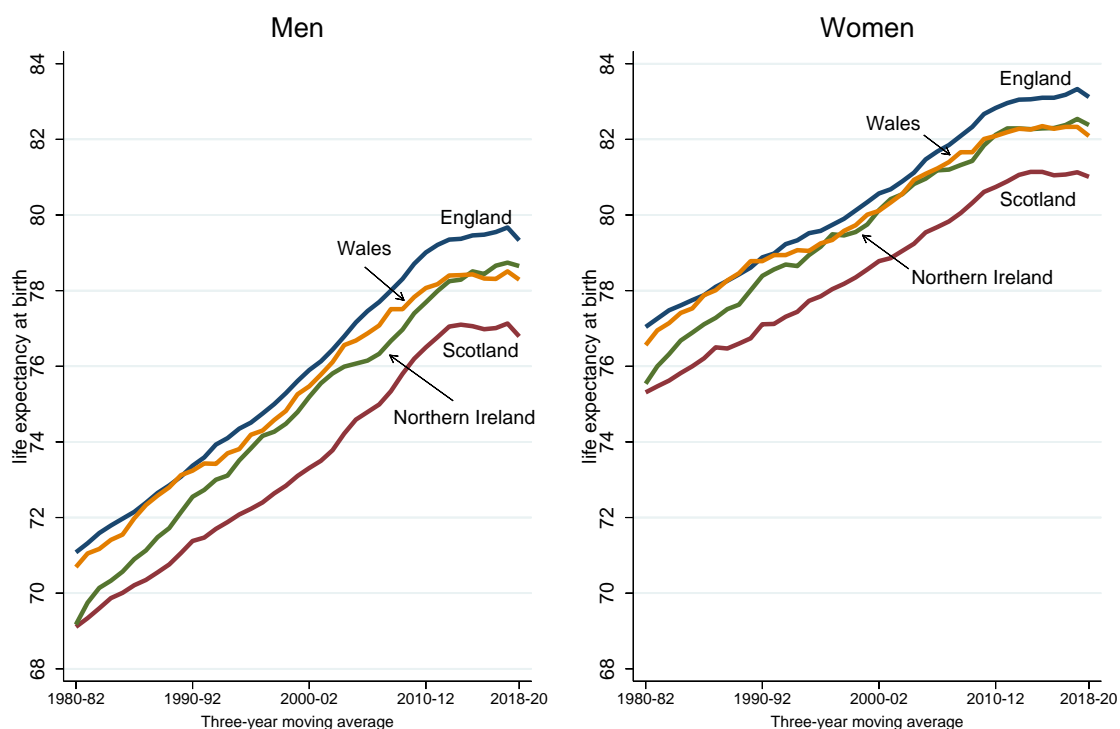
² <https://www.gov.uk/government/statistics/english-indices-of-deprivation-2019>.

³ <https://www.health.org.uk/publications/reports/the-marmot-review-10-years-on>.

(right panel) using three-year moving averages from 1980–82 to 2018–20, for each of the constituent countries of the UK, England, Wales, Scotland and Northern Ireland.

England is and has been consistently healthier than the other three nations. Northern Ireland and, more recently, Wales do less well, and Scotland markedly so, with a life expectancy deficit of more than two years. (England has the highest income per capita, but Scotland has higher income per capita than either Wales or Northern Ireland.)

Figure 1. Life expectancy at birth, by sex and country

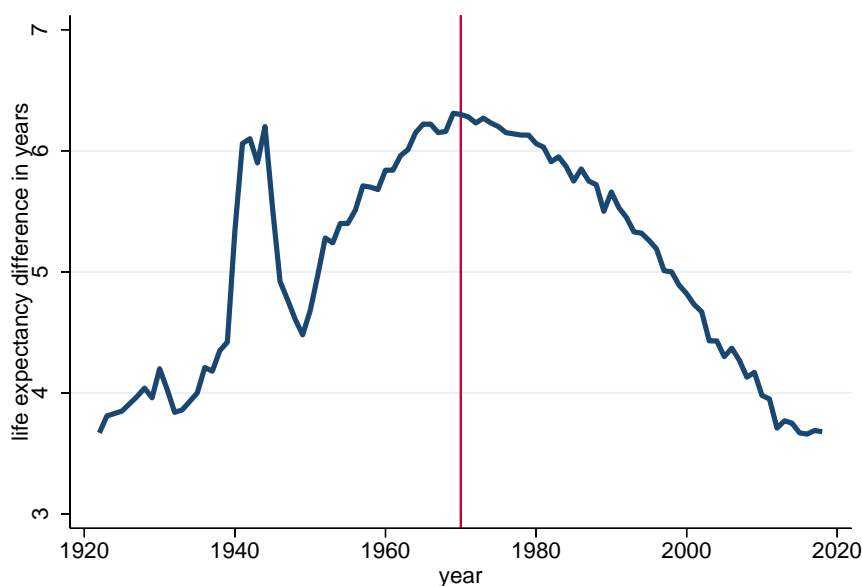


Source: Office for National Statistics.

The panels of Figure 1 show a clear slowdown in improvement in life expectancy after 2010, even prior to the arrival of COVID-19. In Scotland, there was almost no change after 2012–14, and many areas of the country saw decreases in life expectancy (National Records of Scotland, 2020). Scotland, long dubbed the 'sick man' of Europe, has had lower gains in life expectancy than most of Western Europe since 1950, for reasons still being debated (McCartney et al., 2012; Mackenbach, 2012). In February 2022, the Health Foundation announced a review study, *Health Inequalities in Scotland: An Independent Review*,⁴ which plans a thorough examination of factors influencing Scotland's poorer health outcomes.

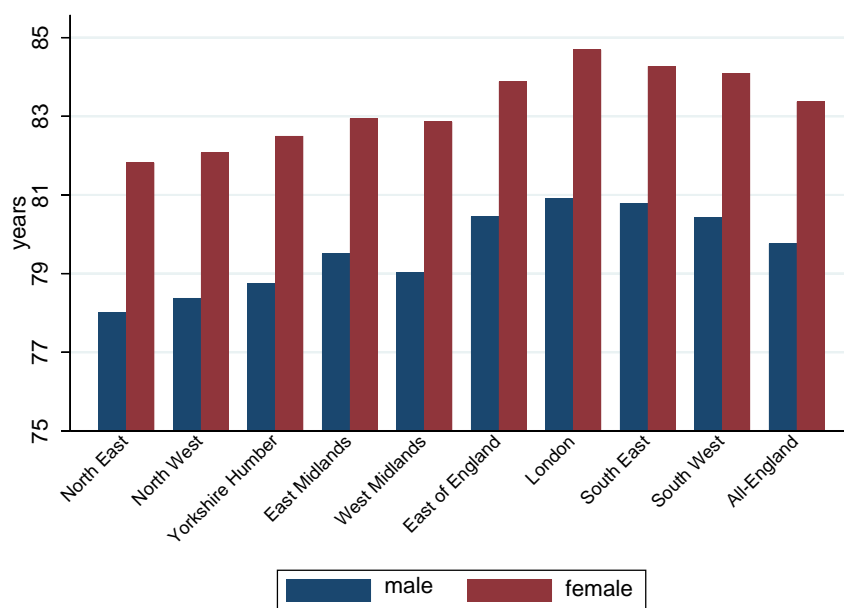
The panels of Figure 1 also show that the increases in life expectancy for women in the last 40 years have been smaller than those for men, with only about two-thirds the gain. The long-run life expectancy advantage of women in the UK can be seen in Figure 2, which plots the difference in life expectancy between women and men over the last 100 years. In the first half-century, women outpaced men in their life expectancy gain. (The toll that the Second World War took on men is highly visible.) However, since 1970, men's life expectancy gains have outpaced those of women. The gap stood at 6.3 years in 1970, but had fallen to 3.7 years by 2018.

⁴ <https://www.health.org.uk/what-we-do/a-healthier-uk-population/mobilising-action-for-healthy-lives/health-inequalities-in-scotland-an-independent-review>.

Figure 2. The difference between female and male life expectancy in the UK, 1922–2018

Source: Human Mortality Database.

Clearly, differences in longevity between men and women are not immutable. The closing of the gap in life expectancy between men and women has long been attributed to different histories of smoking. Women took up smoking many years later than men, and were slower to quit, and because lung cancer lags smoking by many years, women's lung cancer mortality was rising and then falling more slowly relative to men (Appendix Figure 1). The other, and recently more important, development is that the main factor driving down mortality in the last quarter of the 20th century and the first decade of the new century was falling mortality from cardiovascular disease (CVD). Because women die from CVD at much lower rates than men, they had less to gain and gained less, even with equi-proportional reductions in mortality rates (Appendix Figure 2).

Figure 3. Male and female life expectancy, by region of England, 2017–19

Source: Office for National Statistics.

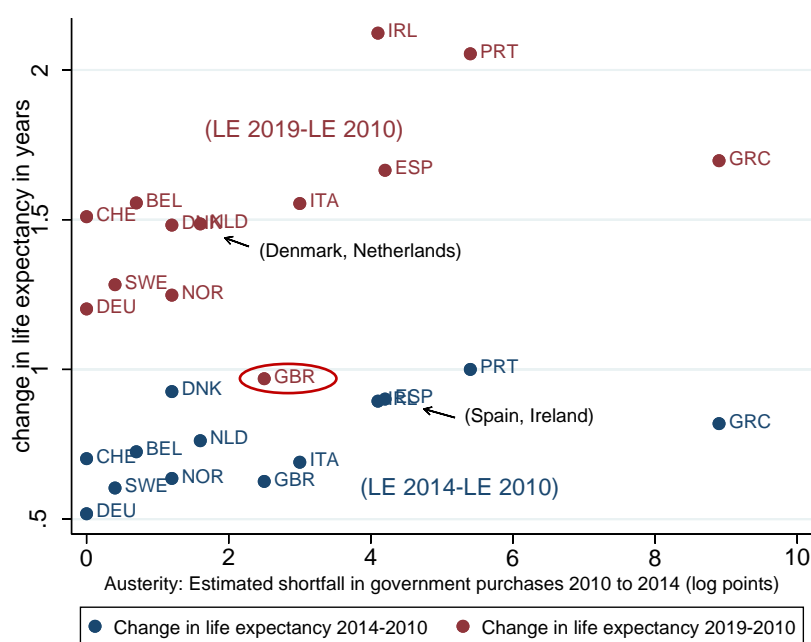
Life expectancy increased modestly in all countries of the UK between 2016–18 and 2017–19, estimated using three-year averages, for both men and women, with the exception of women in Wales, who saw no change (Office for National Statistics, 2021c). In *single-year* estimates, there was a welcome substantial increase in life expectancy in 2019 over 2018 in the UK – of more than 17 weeks for both men and women – the largest increase since 2011. These increases were driven primarily by increases in England, which, given its share of the population, plays a dominant role in UK statistics.

Within England, there are large health differences between regions. Figure 3 shows life expectancy by region just prior to the COVID pandemic. Life expectancy was highest in London, for both men and women, and lowest in the North of England; men and women in the North East experience a nearly three-year life expectancy deficit relative to those in London.

Austerity and mortality. Austere fiscal policies enacted in Britain following the Great Recession included: (net) tax increases, including those for the main rate of VAT and council taxes; reductions in the generosity of the working-age social security system; and reductions in social service spending. The Department of Health and Social Care was spared the large cuts seen in the day-to-day budgets of the Departments for Education, Transport, and Environment, Food and Rural Affairs, the Home Office and the Ministry of Justice (Crawford and Zaranko, 2019). These austerity measures of the 2010s surely made life more difficult for many poorer people.

The slowdown in mortality improvement in the UK has been attributed to its post-Great-Recession austerity programme (Watkins et al., 2017; Martin et al., 2021). Marmot et al. (2020a, 8) states that '[a]usterity has taken a significant toll on ... health'. Banks et al. (2021, 75) note the concurrency of austerity and the 'stalling of the downwards trends in mortality and mortality inequality'.

Figure 4. Austerity and change in life expectancy in Great Britain and Western Europe (2019-2010 and 2014-2010)

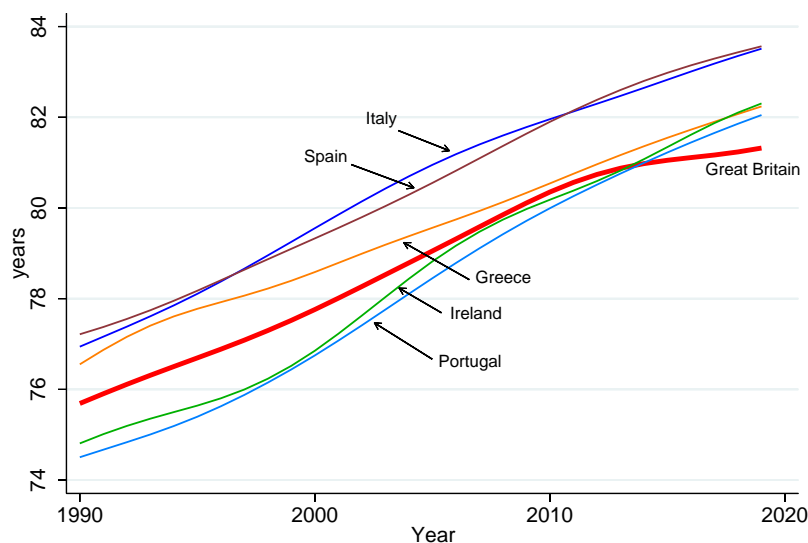


Source: Life expectancy: Our World in Data. Austerity: House, Proebsting and Tesar (2017).

However, it is far from clear that austerity bears primary responsibility for the slowdown in mortality progress. After the financial crisis in 2008, different European countries had different experiences of austerity – some with a great deal, some with none – and there is no systematic negative relationship between the degree of austerity experienced and subsequent health outcomes among wealthy countries. To examine the relationship between austerity and life expectancy across Western Europe, we use an austerity marker developed by House, Proebsting and Tesar (2017). Their measure of the severity of each country's austerity programme is the extent to which government spending in the period from 2010 to 2014 fell below that which they forecast the government *would have spent* if there had been no policy change. Using this measure for 12 countries in Western Europe, we find a *positive* association between the degree of austerity and the change in life expectancy, both from 2010 to 2014 (marked in blue) and from 2010 to 2019 (marked in red) among the countries of Western Europe (Figure 4). (See Wren-Lewis (2017) for a related discussion on life expectancy at age 65.) The '2019-2010' change in life expectancy in Great Britain is especially poor relative to all comparison countries, regardless of their austerity programme.

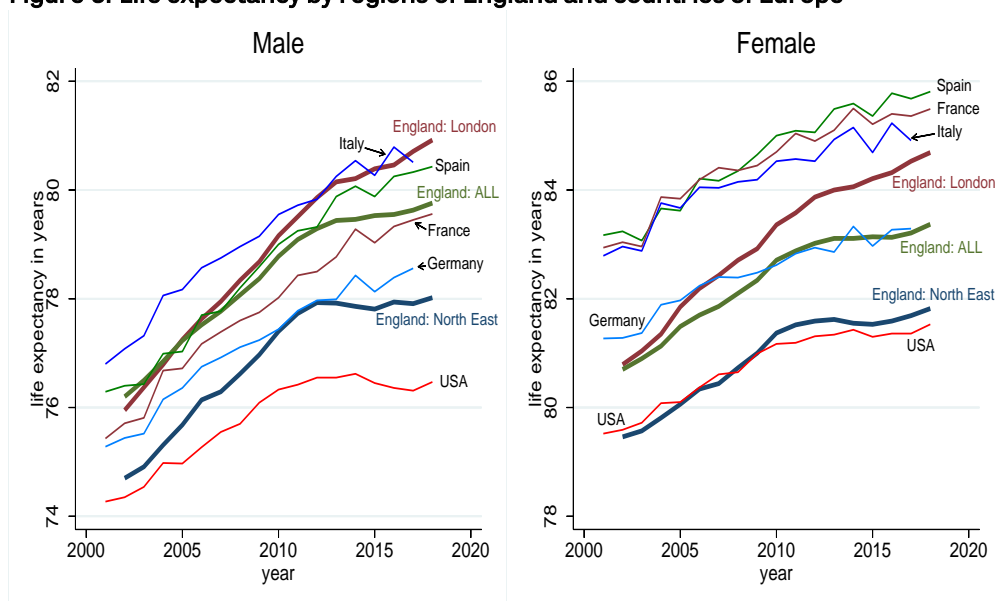
Figure 5 presents life expectancy for Great Britain and countries that faced even greater fiscal austerity following the financial crisis (Portugal, Spain, Ireland, Italy and Greece). Progress in Great Britain was markedly slower than in any other country even in this 'high-austerity' comparison group.

Figure 5. Life expectancy in Great Britain in comparison with other high-austerity countries



Source: Our World in Data.

Disparity in life expectancy between regions of England increased in the 2010s – during the time of austerity. Figure 6 presents trends in life expectancy in the region where life expectancy is highest (London, marked in maroon) and lowest (the North East, marked in navy) as well as for all-England (green), and in Italy, Spain, France, Germany and the US. Life expectancy gains faltered for men and women in the North of England after 2012, but continued at a rapid rate in London. The Centre for Cities reports that cities in the North of England were hard hit by austerity measures; seven of the ten cities with the largest budget cuts were in the North East, North West or Yorkshire. However, London was not spared: with 16% of the population, London accounted for 30% of all budget cuts. Moreover, the Centre for Cities reports that throughout England, social care spending in cities was 'relatively protected'. (Centre for Cities, 2019, 16.)

Figure 6. Life expectancy by regions of England and countries of Europe

Source: Office for National Statistics and Human Mortality Database.

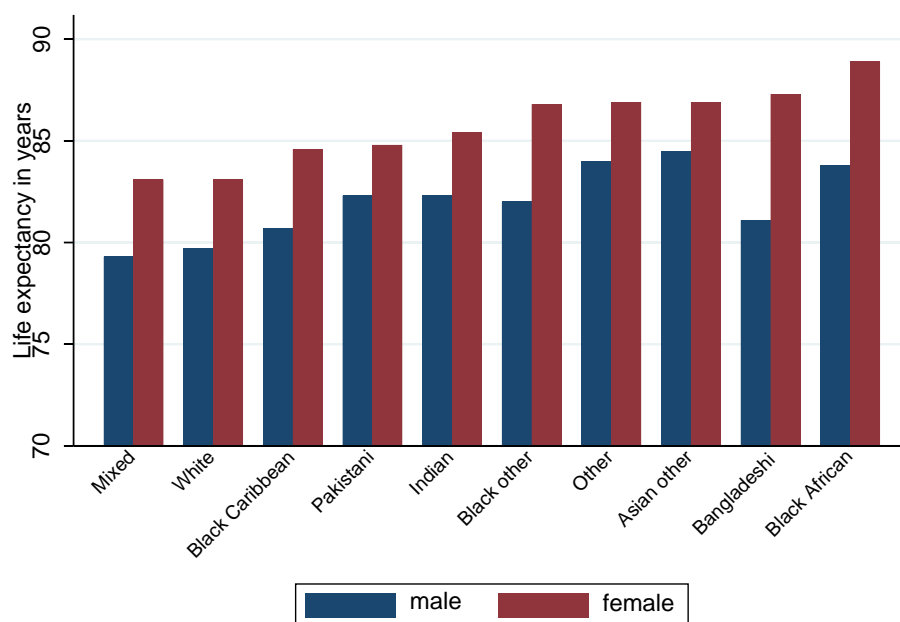
Mackenbach (2019) documents that, for England and Wales, progress in closing education gaps in *partial life expectancy* (expected years lived from ages 35 to 79) between less and more educated adults stalled for both men and women after 2005 – prior to the financial crisis and the period of austerity. Moreover, we find that mortality progress in midlife (ages 25–54) lagged in the UK relative to other wealthy countries in the decades prior to the financial crisis, 1990–2009 (Appendix Figure 3).

The growing gap in life expectancy between London and the North of England, and between the UK and Western Europe as a whole, cannot be explained by austerity alone. Note that we are not defending austerity policies, only questioning their responsibility for the slowing of mortality progress in the UK.

Ethnicity and mortality. In 2021, the Office for National Statistics merged death registration records from the 2010s into the 2011 Census, and for the first time released statistics on life expectancy by ethnicity for England and Wales. (ONS notes that these are *experimental statistics*, meaning that they are ‘in the testing phase and not yet fully developed’.⁵) ONS life expectancy estimates for the period 2011–14 are presented in Figure 7. Taken broadly, black and Asian ethnic groups fare better than whites.⁶ This is consistent with the fact that immigrants consistently report better health than native-born populations, and 50% of Britain’s black, Asian and minority ethnicity (BAME) population is foreign-born. (See Fernández-Reino (2020) and references there.) This is also consistent with the long-standing life expectancy advantage of immigrants, relative to non-Hispanic whites, in the US (Franzini, Ribble and Keddle, 2001; Hendi and Ho, 2021).

⁵ <https://www.ons.gov.uk/methodology/methodologytopicsandstatisticalconcepts/guidetoexperimentalstatistics>.

⁶ Ethnicity is as self-reported in the 2011 Census. In the ONS calculations presented here, ‘white’ includes white British, white Irish, Gypsy and Irish Travellers, and other whites. The ‘mixed’ category includes mixed white and black Caribbean, white and black African, white and Asian, other mixed, and multiple ethnic background.

Figure 7. Life expectancy, by ethnicity and sex, 2011–14

Source: Office for National Statistics, 2021b.

Deprivation and mortality. Much of the recent research on health inequalities in the UK has focused on the association between health at a local level and local area deprivation (measured at the local authority (LA) or Lower Layer Super Output Area (LSOA) level). In this vein, researchers measure differences in outcomes according to the local area's rank in the distribution of an index of multiple deprivation (IMD). However, it is difficult to interpret statistical patterns in the correlation between local area mortality rates and local area rank in the IMD distribution. At this point, we are three-times removed from the underlying economic and social conditions in which we are interested. First, the underlying conditions of interest are combined by category and transformed in a variety of ways to create 'domain indices'. (For example, a number of variables are combined and transformed to create an Education, Skill and Training Domain index.) These domain index scores are then assigned weights and combined to create an IMD. Finally, these IMDs are ranked in ventiles (or deciles or quintiles). The statistical patterns examined are the relationship between IMD rank (1 to 20 in ventile analyses) and mortality rates. The ventile numbers – or other ranks – are non-linear transformations of the underlying deprivation scores, which are themselves transforms of the social and economic measures that policy might hope to address.

Under the layers of statistical calculations, the creation and use of IMD rank lay bare fundamental differences in approach to understanding the role of upstream social factors between different tribes of researchers. Social epidemiologists often focus on the concept of 'socio-economic status' (SES). The concept is not well enough defined to permit direct measurement; instead, it is proxied by one aspect or by a combination of many aspects of the environment – income, employment, education and so on. The use of ranks – both in analyses using IMDs and in creating the domain scores that feed into the IMDs – implicitly adheres to the tenet that differences in rank (rather than, say, poverty itself in one or more domains) drive health outcomes. Economists (on the whole) argue that there is no reason to expect that the different components that go into an SES measure will have identical (or even necessarily similar) effects on outcomes of interest, or to expect that the different components will have the same effect, or even work in the same direction, in different circumstances; different dimensions of poverty need to be analysed

separately, and in context. The former approach has the advantage that it considers many facets of well-being; the latter approach has the advantage that it (at least theoretically) allows research to identify underlying mechanisms and thus inform policy more specifically. How policy should be designed to affect ranks is rarely clear, and effects can be counterintuitive. For example, a universal basic income financed by a uniform increase in income tax rates does not change anyone's rank, though it might have a large effect on reducing post-tax income inequality or poverty rates.

Banks et al. (2021) analyse mortality changes for England using data over the period from 2002–04 to 2015–17. They show that mortality rates declined over this period for both sexes in all age groups (0–4, 5–19, 20–49, 50–64, 65–79, 80+). Absolute declines in rates were greater for men than women, and declines were faster between 2002–04 and 2009–11 than between 2009–11 and 2015–17, consistent with the male and female life expectancy changes noted above. Mortality rates were consistently higher in more deprived areas, measured by dividing local authority areas of the country into 20 areas of equal population (ventiles), ranked by their index of multiple deprivation. Banks et al. find that mortality inequality, defined as the slope of mortality gradients with respect to IMD rank, fell from 2002–04 to 2009–11, with little change in the slope between 2009–11 and 2015–17. An exception here is among young children; consistent with the work of Baker et al. (2021) for the US, the slope of the mortality gradient with respect to IMD rank continued to fall for this youngest group, most prominently among boys aged 0–4.

However, conclusions about progress in the UK depend upon how *mortality inequality* is defined, how geographic units are grouped, and the period under study. Kraftman, Hardelid and Banks (2021) use finer geographic units (LSOAs), and measure mortality inequality as the ratio of mortality in the most deprived to the least deprived decile. Over the 2002–04 to 2015–17 period, the largest inequalities were among 40- to 54-year olds; mortality rates for men and women in this age group were three times higher in the most deprived decile than in the least deprived in 2015–17. Mortality rate ratios increased over this period, but significantly so only among the elderly.

Marmot et al. (2020a) also use data on area deprivation in England, and concentrate on the period from 2010–12 to 2016–18, which shortens the window over which change is measured, but brings the analysis closer to the present. They highlight differences in life expectancy by decile of deprivation, and show, using data from 2016–18, that women living in the least deprived decile can expect 7.7 more years of life than those in the most deprived decile (9.5 years for men). (Note that premature mortality is one of the components of the Health Deprivation and Disability Domain score, which carries weight in the IMD used by Marmot et al. (2020a). Banks et al. (2021) and Kraftman, Hardelid and Banks (2021) remove the health domain from the IMD, because its presence introduces a circularity, and will induce an automatic relationship between socio-economic and physical deprivation and mortality.) Marmot et al. (2020a) find a drop of 0.3 years in life expectancy for women in the most deprived decile over the period 2010–12 to 2016–18, and only weak life expectancy growth for women in the next three most deprived deciles. Men in the bottom decile saw the smallest gain in male life expectancy (approximately 0.2 years), but men in almost all but the bottom two deciles saw gains of half a year or more, *with the highest gains found in the middle of the distribution of areas ranked by deprivation*. Marmot et al. conclude that, during this period, mortality inequalities have widened overall.

Education and mortality. An alternative to analysing changes in mortality inequality by deprivation indexes is to examine adult mortality *differences* by level of education. This (relatively more) concrete variable allows comparisons both within the UK, and with other wealthy countries, and

forges a link between mortality and a magnitude that might be affected by policy. It is, however, challenging to compare mortality outcomes by educational status either across countries or over time within a country. Over time in the US, for example, the fraction of adults without a high-school degree dropped from 28% in 1980 to 8% in 2019.⁷ As the fraction who left high school without a diploma dropped from more than one in four to less than one in ten, the characteristics of the group without a diploma changed dramatically. People without a high-school degree today are more likely than was true 40 years ago to have been ill as children, and to face mental health challenges. Tracking changes in the mortality rate of this group over time will pick up both changes in what the absence of a diploma ultimately does to health and changes in the composition of the pool – a selection effect. Across countries, education confers different benefits, benefits that might be expected to have different effects on health outcomes. The minimum educational qualification needed to secure a good job is likely to be country-specific, as is whether, and the extent to which, social status is attached to education. These issues suggest care is needed in interpreting comparisons, which will be less problematic the shorter the time period analysed and the more comparable the countries.

Mackenbach et al. (2018) use a set of harmonised mortality records for 17 Western and Eastern European countries to study mortality trends among more and less educated groups,⁸ aged 35–79, between 1980 and 2014, a period that covers the financial crisis of 2008. In all cases, mortality rates for the more educated are lower than those for the less educated, a result that seems universal. Mortality rates declined for both less and more educated groups in most countries, including England and Wales. To study trends in mortality inequality, Mackenbach et al. use two measures: *mortality rate differences* between the education groups and *mortality rate ratios* (the ratio of mortality in the less educated group to that in the more educated group). They document a decline in mortality rate differences in most countries, including England and Wales; that is, mortality rates fell by more over this period among the less educated. There were, however, increases in mortality rate ratios; that is, there was a higher percentage drop in mortality among the more educated. Overall, they find no change in the trends of all-cause mortality for either less or more educated people over the crisis period, and conclude that 'the financial crisis had no discernible short-term impact on health inequalities at the population level in Western Europe' (Mackenbach et al., 2018, 6443). Apart from their inherent interest, these results demonstrate the difficulty in talking about changes in health inequalities without precise definitions.

For England and Wales, Mackenbach (2019, figure 2.7) presents data on absolute gaps in *partial life expectancy* (sometimes called temporary life expectancy – the expected years to live between ages 35 and 79) between adults with high and low education qualifications, annually for the period 1970–2014. Gaps began to close for men in the early 1980s, and for women in the late 1990s. However, progress in closing these gaps stalled for both men and women after 2005 – prior to the financial crisis and the period of austerity.

It is not possible to directly compare the results in Mackenbach (2019), Banks et al. (2021) and Marmot et al. (2020a), discussed above. These studies cover different years, during a period when mortality rates were changing rapidly; they focus on different age groups; and they use different metrics to measure health inequalities. However, the common picture that emerges is one in which mortality rate differences between the less and more educated, and between more

⁷ Authors' calculation using the US Current Population Survey.

⁸ For most countries, the less educated group has an International Standard Classification of Education (ISCED, 1997 definition) of 0–2 and the more educated group has an ISCED of 4–6. For England and Wales, the groupings were ISCED 0–3 for the less educated (through NVQ3, A levels, GNVQ intermediate level) and 4–6 for the more educated (higher education (HE) access, NVQ4 and above). (Correspondence with J. P. Mackenbach.)

and less deprived places, remain substantial. Progress in reducing mortality differences appears to have stalled at the same time that overall mortality declines have stalled.

Once ONS releases the full merge of the English census to death records, up-to-date analyses of mortality risk by individual characteristics – educational qualification, and race and ethnicity – rather than by the characteristics of local authorities or LSOAs will be possible, and should add greatly to what we know about mortality inequalities in the UK.

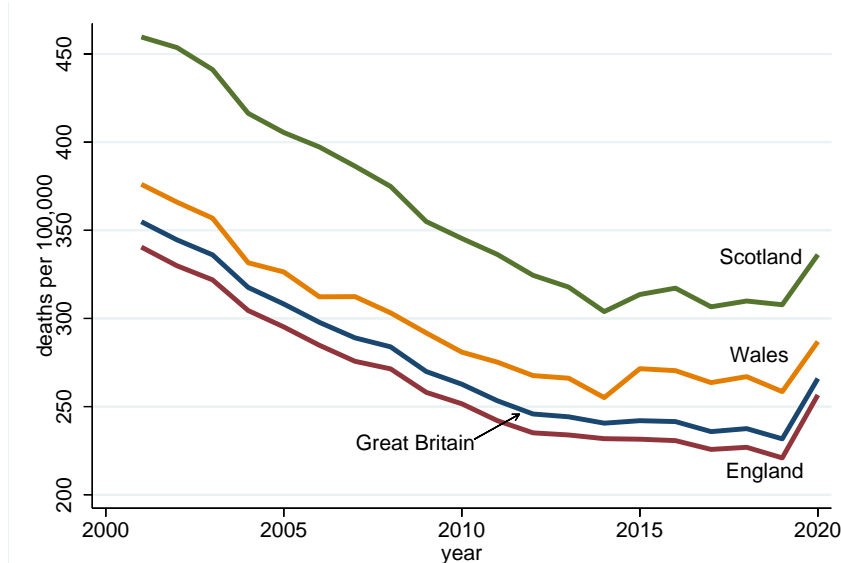
Causes of death

Avoidable mortality. Mortality rates by cause of death are often divided according to whether the mortality is thought to be avoidable, generally defined as mortality *preventable* – through public health and prevention interventions prior to the onset of disease – and/or *treatable* through effective healthcare (OECD, 2022). We note, however, that assignment to the 'avoidable mortality' category is subjective, as are the terms 'preventable' and 'treatable'. The OECD and the ONS have assigned coronavirus as a preventable cause of death in 2020 (Office for National Statistics, 2022c), although it is not clear that COVID-19 was preventable in the early months of the pandemic, before transmission was fully understood and well before vaccines were available.

Marmot et al. (2010) argue forcefully that 'avoidable health inequalities are unfair and putting them right is a matter of social justice' (p. 35). Banks et al. (2021) find that half of all deaths of working-age adults (20–64) were from avoidable causes in England, leading them to note that 'there is considerable scope for improvements in health care and public health interventions to further reduce mortality rates' (p. 67). Large differences exist in avoidable mortality across local areas; age-standardised avoidable mortality rates in England in 2017 were two to three times higher in the most deprived decile than in the least deprived (Marmot et al., 2020a).

Figure 8 presents avoidable mortality for the countries of Great Britain over the period from 2001 to 2020. Scotland is again an outlier, with markedly higher rates. However, Scotland, along with England and Wales, made steady progress in reducing avoidable mortality until 2013, after which progress uniformly stalled. Rates rose in all countries in 2020 with the arrival of COVID-19.

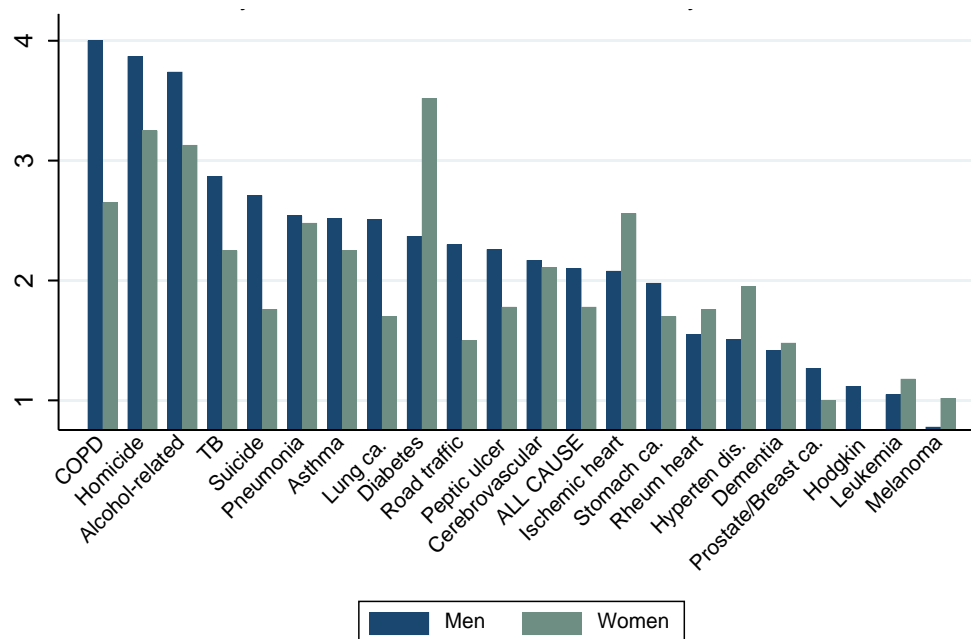
Figure 8. Age-standardised avoidable mortality rates, Great Britain, 2001 to 2020



Source: ONS and National Records of Scotland, Office for National Statistics (2022c).

Disparities across social groups in avoidable mortality are found throughout Europe. Mackenbach (2019) pools data from 16 European countries, and calculates the mortality ratios between less and more educated people, for the main causes of death. We reproduce results from his analysis in Figure 9. As Mackenbach notes, the largest rate ratios are found for causes of death that are 'eminently avoidable': chronic obstructive pulmonary disease (COPD, closely linked to smoking); tuberculosis (TB, a treatable medical condition); and alcohol-related and suicide mortality. Mackenbach notes the 'striking range of health problems for which inequalities in mortality are found ... conditions which at first sight seem to have completely different aetiologies' (p. 31).

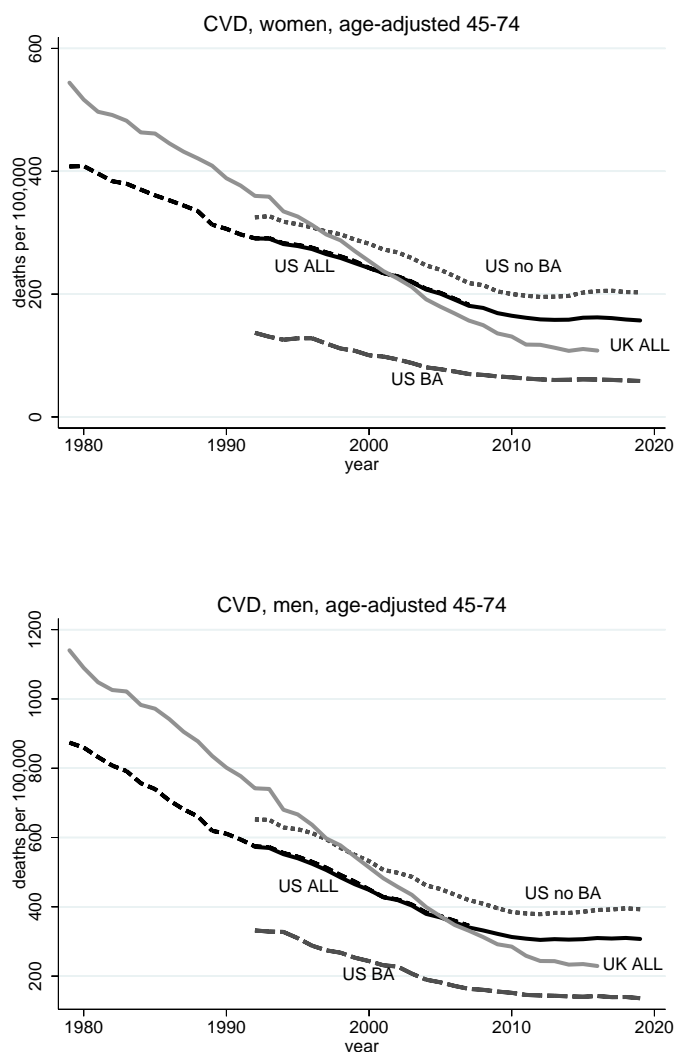
Figure 9. Mortality rate ratios of less to more educated adults in Europe, by cause of death



Source: Mackenbach (2019, figure 2.4) using pooled data from 16 European countries in about 2010.

Cardiovascular disease. The most important causes of mortality declines in rich countries in the last third of the 20th century were falling deaths from cardiovascular disease (CVD), usually attributed to the increased use of antihypertensive and cholesterol-lowering drugs, and a decline in smoking rates. For reasons that are not well understood, progress against CVD mortality has slowed, not only in Britain but also in the US, Canada and Australia (Case and Deaton, 2020). This decline – together with an increase in deaths from accidental drug overdose – accounts for much of the stalling of life expectancy gains since 2010.

Figure 10 compares age-adjusted CVD mortality for those aged 45–74 in the US and the UK, with additional lines by education after 1992 for Americans, separating out people with and without four-year university degrees. The time pattern of the stall is similar for men and women, albeit on different scales. The mortality rate from CVD for men is around twice the rate for women. Scotland has consistently had higher CVD mortality than have England and Wales, and Northern Ireland, but the stalling of progress is seen in all of the countries of the UK. The stalling is much more severe in the US than in the UK, and is largely attributable to reversals among less educated Americans (Case and Deaton, 2021), something we cannot directly assess for Britain.

Figure 10. Cardiovascular disease mortality in the US and the UK

Source: Authors' calculations from WHO Mortality Database and US National Vital Statistics System.

Marmot et al. (2020a) document a large difference in CVD mortality between the highest and lowest deprivation area deciles of England, for the period 2010–17. Men in the highest deprivation areas have CVD mortality rates that are four times as large as those for men in the lowest deprivation areas. But men in the most deprived areas also saw the most absolute progress, with a decline in the CVD mortality rate of almost 40 per 100,000 men at risk between 2010 and 2014. However, that progress nearly ground to a halt after 2014, as it did for women in the most deprived areas.

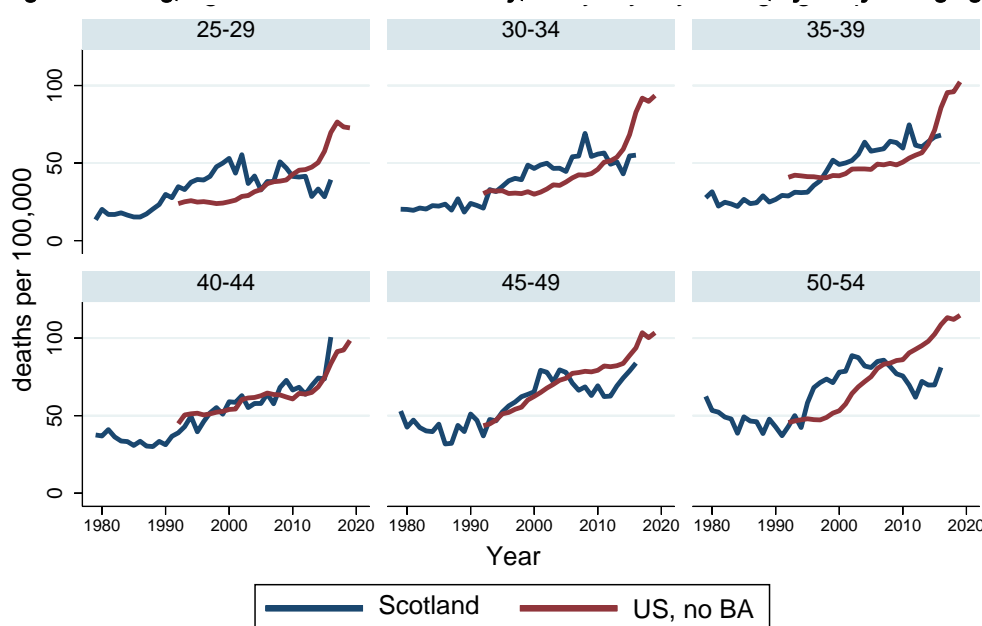
Additional evidence on gradients in CVD mortality in England comes from analysis of the English Longitudinal Study of Ageing (ELSA). Zaninotto et al. (2016) report that, under age 70, CVD mortality risk in the lowest occupational classes (routine and manual occupations) is more than twice that found in the highest class (managerial and professional). Moreover, those with low educational qualifications (leaving school before or at compulsory-schooling age) are at higher risk of CVD mortality than those with A levels or a college degree. A social gradient in obesity levels is likely important here; we return to this below.

Cancer. In all countries of the UK, cardiovascular disease was a larger killer than cancer until 1999. However, with the progress observed against CVD, cancer has been a larger cause of death than CVD since 1999 for those in midlife (broadly defined as ages 25–74). Scotland has consistently had higher cancer mortality rates than England and Wales, and Northern Ireland, and rates for men are uniformly higher than rates for women. But in all countries, and for both men and women, cancer mortality rates have fallen consistently since 1990, with a 1.5% per year decline on average for the UK as a whole.

Deaths of despair. Deaths from suicide, accidental drug overdose, and alcoholic liver disease have been rising in Britain as they have been in the US, though – with the exception of Scotland – the death rates are much lower in Britain. In England and Wales, there has been little increase over time for younger adults (ages 25–34), but a persistent upward trend for those aged 35–54 (Appendix Figure 4).

Scotland shows a disturbing pattern, different from that in the rest of the UK. Figure 11 compares these deaths, by age group, with those in the US among Americans without a college degree. As in the US, these mortality rates have risen markedly over time. Drug deaths doubled between 2014 and 2019, mostly in urban areas (National Records of Scotland, 2021). The death rate from drugs in Scotland is more than three times higher than in any other European country, and while most involve opioids (heroin and methadone), deaths involving benzodiazepines, which can be deadly when mixed with opioids, have also been rising rapidly. It is not well understood why drug overdose deaths have skyrocketed in Scotland, but not in the other countries of the UK.

Figure 11. Drug, alcohol and suicide mortality, Scotland and the US, by five-year age group



Note: US rates are for those without a four-year university degree (BA).

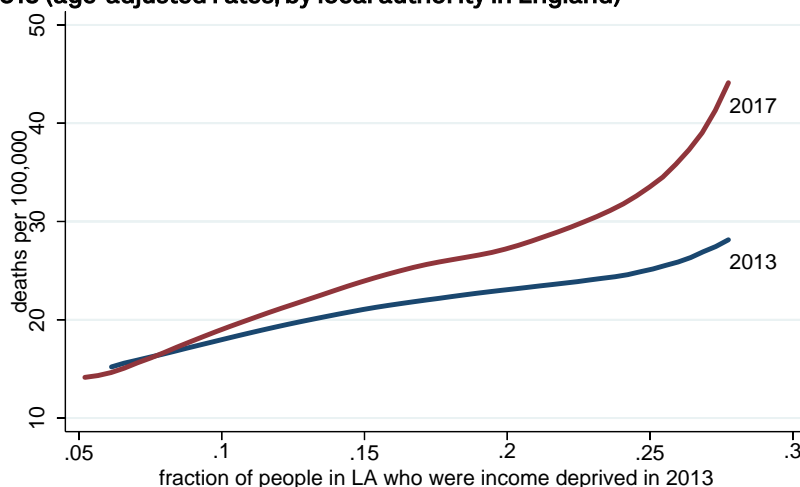
Source: WHO Mortality Database and US National Vital Statistics System.

Although mortality rates from drug overdose were low in England in the period 2016–18, there was a gradient in drug mortality with respect to IMD decile, with 6.6 deaths per 100,000 people in the most deprived decile, compared with 2.8 in the least deprived. Hospital admissions for alcohol-related conditions also show a clear gradient: in 2018–19, there were 350 admissions per 100,000 people in the least deprived IMD decile, and 829 in the most deprived (Appendix Figure 5).

Until we are able to merge death records into the census, we will not be able to break down these deaths of despair in Britain by education or by ethnicity. However, we can combine data provided by the Department for Communities and Local Government with population estimates from ONS to measure the fraction of people counted as income deprived in 2013 in each local authority in England. We find, analysed separately, that suicide, alcoholic liver disease and accidental poisoning⁹ (drug overdose) mortality rates are higher in districts where a higher fraction of people live in income poverty.

Figure 12 shows the age-adjusted mortality rate for the three deaths of despair taken together. There is a substantial mortality gradient with respect to income poverty in 2013, which was steeper still in 2017.

Figure 12. Drug, alcohol and suicide mortality in 2013 and 2017, by the fraction income deprived in 2013 (age-adjusted rates, by local authority in England)



Source: Office for National Statistics; Department for Communities and Local Government.

The relationship between mortality, education, income poverty and economic activity – as opposed to IMD rank – can be seen in Table 1, which presents regression results of cardiovascular disease mortality (column 1) and deaths of despair (column 2) at the local authority (LA) level, annually from 2013 to 2017. Age-adjusted mortality rates are regressed on the percentage of the LA population aged 16–64 with high and medium educational qualifications (relative to the percentage with low or no qualifications, which has been chosen as the benchmark); the percentage of the LA who are income deprived (and the square of this percentage); and the percentage aged 16–64 who are not economically active. The control variables are all measured in 2013. All else equal, CVD mortality rates are lower in LAs where a higher percentage of adults have more education. A 1 percentage point increase in NVQ2 qualifications relative to low or no qualifications is associated with a lower CVD mortality rate of 0.35 per 100,000. Mortality is lower still with a higher percentage of NVQ3 qualification, and still lower with a higher percentage of at least NVQ4 qualifications. These markers of education within the LA are, jointly, highly significant (F -test=41.6, p -value=0.000). Income poverty within the LA is also a significant correlate with CVD mortality, with an F -statistic on the joint significance of income poverty and its square of 232.1. In contrast, the percentage of adults who are not economically active is *not* significantly correlated with CVD mortality in regressions that control for income poverty.

⁹ These are all accidental poisonings (ICD10 X40–49). In addition to drug and alcohol poisoning, the category includes a small number of accidental deaths from other chemicals, which account for 1% of this category.

Table 1. Mortality, education, income poverty and economic activity

Explanatory variables	Dependent variable:	
	Cardiovascular disease mortality	Suicide, alcoholic liver disease and accidental poisoning mortality
Education		
Percentage with NVQ4+, 2013	-1.02 (0.15)	-0.10 (0.05)
Percentage NVQ3, 2013	-0.70 (0.23)	0.43 (0.08)
Percentage NVQ2, 2013	-0.35 (0.24)	0.03 (0.08)
<i>F</i> -test: education (<i>p</i> -value)	41.62 (0.000)	64.92 (0.000)
Income poverty		
Percentage income deprived in LA, 2013	5.96 (0.67)	0.27 (0.20)
Percentage income deprived in LA, 2013, squared	-0.07 (0.02)	0.01 (0.01)
<i>F</i> -test: poverty (<i>p</i> -value)	232.12 (0.000)	87.42 (0.000)
Percentage not economically active, 2013 (ages 16–64)	0.15 (0.18)	0.06 (0.05)
Number of local authorities	324	304
Number of observations	1,620	1,221

Note: Explanatory variables are observations at the local authority (district/unitary prior to 2015) level, measured in 2013. The omitted education category is the percentage with no or low (NVQ1) qualifications. Dependent variables are age-adjusted mortality rates at the LA level from 2013 to 2017. Twenty LAs were missing information on death from drugs, suicide or alcoholic liver disease for this period. Numbers in parentheses below coefficients are standard errors, except where indicated as *p*-values for *F*-statistics.

Turning to deaths of despair, similar to the results for CVD mortality, we find that the percentage of the LA in income poverty is positively and significantly associated with mortality, while the percentage economically active is not. There is a difference between CVD and deaths of despair, however, in the relationship between educational qualifications in the LA and mortality from drugs, alcohol and suicide. The LA percentage with NVQ4 or higher (which includes first degrees and Key Skills level 4) is associated with a significantly lower mortality from drugs, alcohol and suicide. However, a higher percentage with NVQ3 (A levels and Key Skills level 3) is associated with a significantly *higher* mortality rate. This is a result worthy of more study. A deeper analysis will be possible when the census is merged with individual death records. Mortality data at the local area level are not sufficient to provide an understanding of this result.

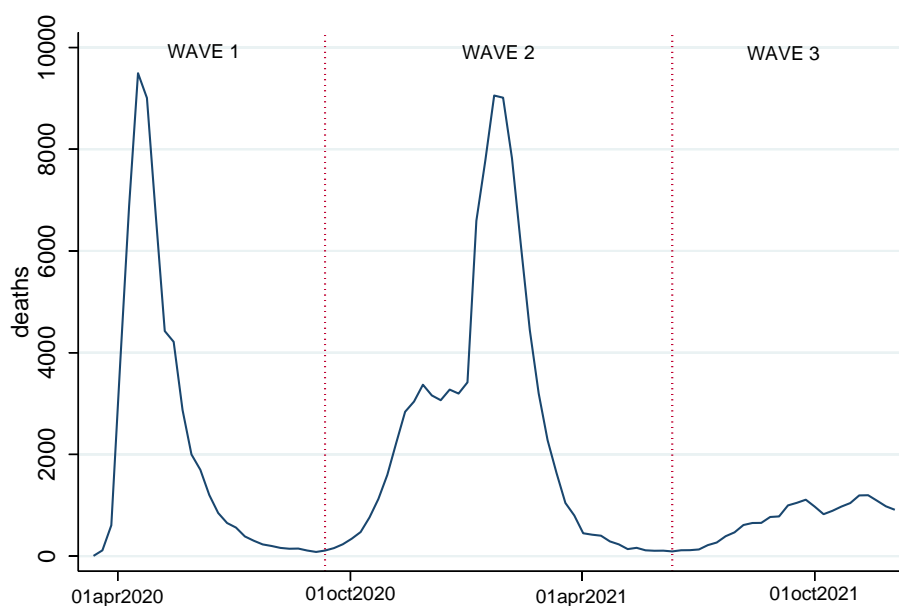
COVID-19 mortality

The effects of COVID-19 on pre-existing health inequalities help us think about the origins of the inequalities themselves. One account focuses on social position, arguing that lower position in the social hierarchy puts individuals at greater risk. This status-based account is consistent with higher risk in more deprived places, as well as worse health for lower-status racial and ethnic groups. Fundamental cause theory extends this account, accepting the importance of status, but argues that status – through its association with power, knowledge and wealth – works in ways that depend on the epidemiological environment (Link and Phelan, 2004). Health inequalities by status may change, for example, if higher-status people are the first to learn how to avoid risks that were previously unavoidable or, more generally, when the epidemiological environment changes.

COVID-19 brought new risks that were patterned by occupation and by location. Occupation and location are, of course, themselves patterned by status, so that the risks of COVID-19 follow pre-existing mortality risks by status. However, there are differences too, particularly if some groups are heavily represented in occupations or locations that put them at higher risk and if the new risks are not perfectly related to status.

While a full reckoning of the pandemic's impact on mortality, morbidity and health inequalities will not be possible for some time, we discuss what is currently known about the geographic, ethnic, social and economic correlates of COVID-19 mortality and mortality inequality in the UK. To do so, it is useful to keep in mind the timing of the pandemic. Figure 13 shows, for England and Wales, the number of deaths involving COVID-19, registered in each week of 2020 and 2021. The first wave of deaths peaked in April 2020, and was followed by a period of decline that lasted through to mid September. A second wave started then, and deaths involving COVID-19 rose until mid January 2021 and then declined until mid June 2021. A third wave began in June 2021, and rates rose again, especially among those not fully vaccinated (Office for National Statistics, 2022b).

Figure 13. COVID-19 mortality, registered by week, England and Wales



Source: Office for National Statistics, 2022b.

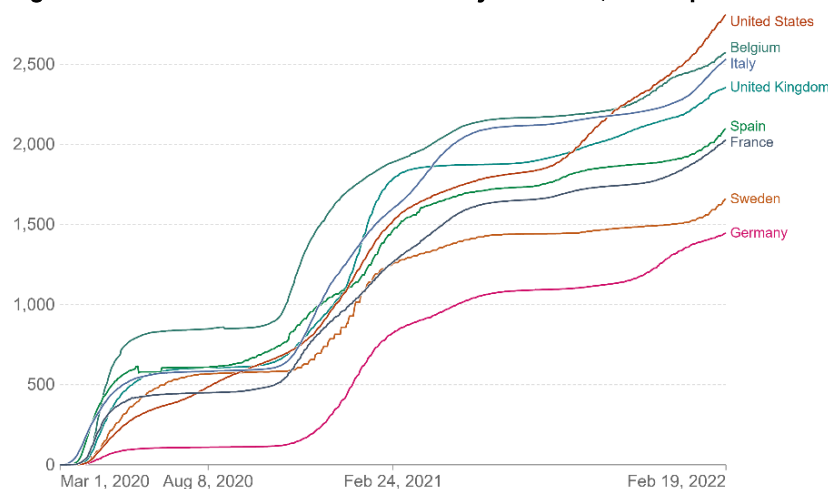
ONS reports both on deaths *involving* COVID-19, which signals that COVID-19 appeared somewhere on a death certificate, and deaths *due to* COVID-19, where a determination has been made that COVID-19 was the underlying cause of death. To date, the fraction of deaths involving COVID-19 that are determined to be due to COVID-19 has been higher when deaths involving COVID-19 are cresting, and lower during each trough (Office for National Statistics, 2022a).

Data sets currently available to analyse different aspects of the pandemic vary in the time periods that they cover – some data are available throughout 2021, others only through to the end of the first or second wave.

Excess deaths from COVID-19 approximately follow the standard patterns of all-cause mortality with respect to age and sex, with COVID-19 mortality rates higher at higher ages, and higher among men than women. More than 75% of deaths during the first wave were among those aged 75 or above. However, the age distribution of COVID-19 deaths shifted toward younger ages with the arrival of vaccines; during the second (Delta) wave, 59% of deaths were among those aged 75 or above (McIntyre et al., 2022).

Inequality between countries. Cumulative COVID-19 deaths per million people are presented in Figure 14 for the UK, the US and comparison European countries through to mid February 2022 (Ritchie et al., 2020). The UK fared better than the US, Belgium and Italy, but markedly worse than Germany, Sweden, France or Spain.

Figure 14. Cumulative COVID-19 mortality in the UK, in comparison with other wealthy countries



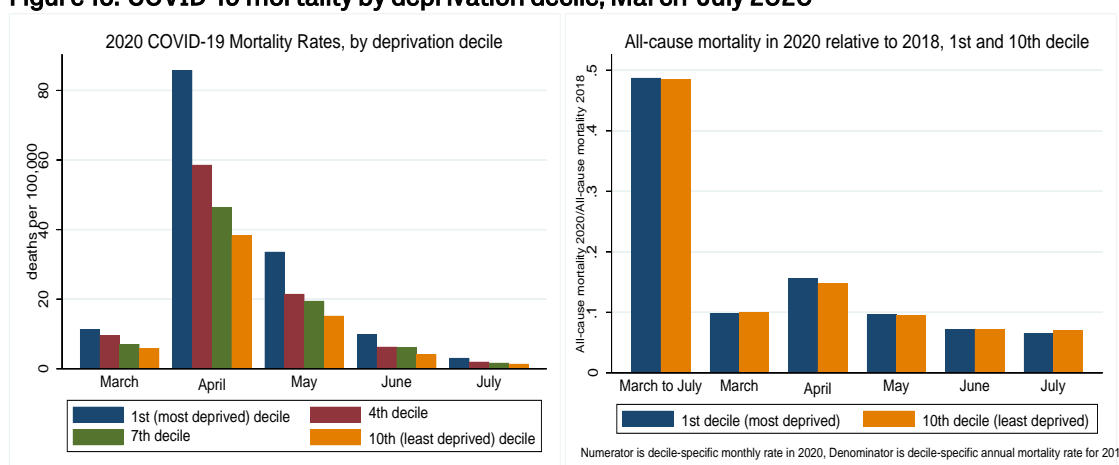
Note: Cumulative confirmed COVID-19 deaths per million people. For some countries, the number of confirmed deaths is much lower than the true number of deaths. This is because of limited testing and challenges in the attribution of the cause of death.

Source: Ritchie et al. (2020), using Johns Hopkins University CSSE COVID-19 data.

The COVID-19 Marmot Review (Marmot et al., 2020b) argues that growing inequality and government austerity policies of the last 10 years provide an explanation for why England had among the worst infection and mortality rates in 2020. However, without further analyses, including a deeper description of mechanisms, it is not possible to quantify whether and which austerity measures, and which components of deprivation, were drivers of the waves of the pandemic in the UK. Indeed, the dynamics of the pandemic are so little understood – in Britain as elsewhere – that it is almost certainly premature to highlight one policy, whether austerity or anything else.

Deprivation and COVID-19 mortality. The COVID-19 pandemic is occurring against a backdrop of the pre-existing health inequalities discussed in this chapter. There is a higher risk of mortality among those with pre-existing health conditions, which occur more frequently in areas of higher deprivation. As a result, equal exposure to the virus will lead to higher mortality rates in more deprived areas. In addition, there may be higher risk of exposure to the virus for certain groups, such as those who live in cramped housing (Platt and Warwick, 2020; Marmot et al., 2020b), or work in frontline occupations (Marmot et al., 2020b), facing adverse working conditions (Bambra et al., 2020), or are unable to work from home (Blundell et al., 2020). These factors taken together likely account for some part of the higher COVID-19 death rates observed in the most deprived areas during the first wave of the pandemic, seen in Figure 15.

Figure 15. COVID-19 mortality by deprivation decile, March-July 2020



Source: Office for National Statistics, 2020a and 2020b.

The left panel of the figure shows COVID mortality rates in the first (most deprived), fourth, seventh and tenth (least deprived) deciles, over the course of the pandemic's first wave. There is a clear gradient in COVID-19 deaths; in every month from March through July, COVID-19 mortality is higher, the more deprived the area. In April, at the peak of the first wave, the COVID-19 mortality rate in the most deprived decile was over twice that in the least deprived decile.

That said, the disproportionate COVID-19 mortality seen in the most deprived areas had little impact on the all-cause mortality gradient with respect to IMD decile. Mortality in general is higher in more deprived areas. If COVID-19 were to steepen the overall mortality gradient, the COVID-19 mortality gradient would need to be more severe than the pre-existing gradient. We examine this in the right panel of Figure 15 by expressing all-cause mortality rates for the most and least deprived deciles during the first wave of the pandemic as a fraction of the 2018 all-cause mortality rates in those deciles. We find that COVID-19 increased all-cause mortality in proportion to existing mortality in the highest and lowest deciles. Aggregating over the period from March to July, the all-cause mortality rate in the most deprived decile was 48.7% of the annual mortality rate in that decile in 2018, compared with 48.5% in the least deprived. This is a remarkable finding. If the virus were an equal-opportunity killer, and all people were at equal risk of dying from it, we would expect COVID-19 mortality to increase all-cause mortality relatively more in less deprived areas, because there are fewer deaths there to begin with, which would dampen the all-cause mortality gradient. Conversely, if those in the poorest deciles were at higher mortality risk from COVID-19 than from the other perils they face, we would expect the all-cause mortality gradient to steepen. But, rather than reducing or increasing the overall mortality

gradient, the virus appears to have cemented the mortality gradient by deprivation area that existed pre-pandemic, at least through to the end of the first wave.

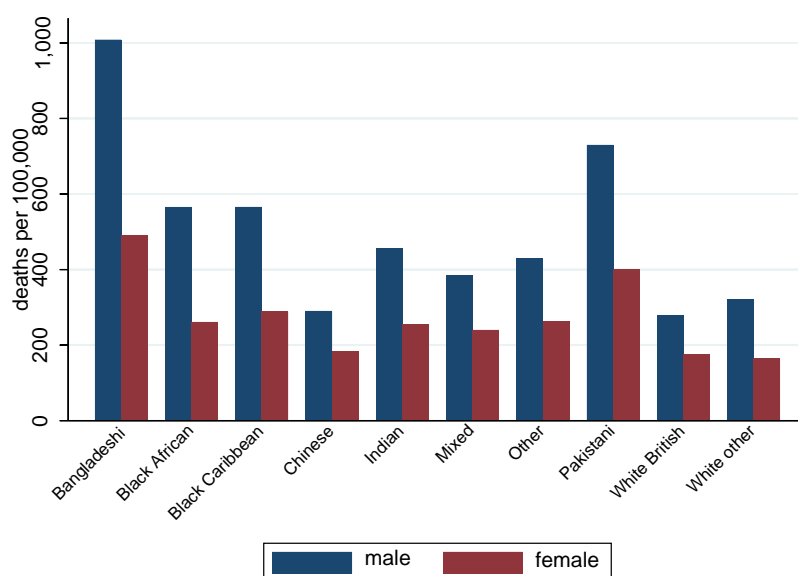
Regional differences in COVID-19. The nature of the spread of the virus means that some areas are at higher risk. Urban areas, which are more densely populated and contain more deprived areas, have had higher mortality rates than rural areas in the first wave of the pandemic (Office for National Statistics, 2021d). This occurred even though people living in urban areas are younger on average, which would counteract the mortality risk. In the first COVID wave (20 March to 31 July 2020), the percentage excess mortality was highest in London, the West Midlands, the North West and the North East. From August through November 2020, percentage excess mortality was highest in the north (North West, North East, Yorkshire and the Humber) (Marmot et al., 2020b).

Public Health England (2021) presents the percentage excess mortality, by deprivation decile, for the first year of the pandemic (to 5 March 2021) for each region of England. Most regions saw a relatively uniform (approximately 20%) increase in excess deaths across IMD quintiles, consistent with evidence presented above and in Dowd et al. (2020) for first-wave deaths. Exceptions are for London – where the percentage excess mortality in the three most deprived quintiles were elevated relative to the two least deprived quintiles – and the South East and South West, where the percentage excess mortality increase was *highest* in the least deprived decile. The regions that experienced proportional increases in mortality across quintiles will not see change in existing regional mortality gradients.

COVID-19 is also likely to increase mortality differences between the least and the most vulnerable people, independent of region. Homeless people's living arrangements, whether formal (shelters or halfway houses) or informal (encampments or abandoned buildings), and their lack of access to healthcare supplies and good hygiene, make them susceptible to a disease epidemic. Refugees and asylum seekers also face disproportionate risk. Lack of basic sanitation and health resources leads pandemics to have a higher rate of contingency in overpopulated refugee camps (Eiset and Wejse, 2017).

COVID-19 and ethnicity. Large inequalities in COVID-19 mortality exist by ethnic group, despite the younger age profiles of most ethnic minority groups. ONS has released provisional estimates of age-standardised COVID-19 mortality rates by ethnicity for the period from 24 January 2020 to 1 December 2021, shown in Figure 16. COVID-19 hit Bangladeshis and Pakistanis harder than any other ethnic groups, followed by black Caribbeans and black Africans. White and Chinese groups saw the lowest COVID-19 mortality rates.

These patterns stand in sharp contrast to those observed prior to the COVID-19 pandemic, presented in Figure 7. According to ONS experimental data, also used in Figure 16, pre-pandemic all-cause mortality rates were highest for whites and lowest for black Africans, with Pakistani and Bangladeshi rates in between. As shown in Figure 16, COVID-19 mortality patterns are starkly different.

Figure 16. Age-standardised COVID-19 mortality rates for those aged 30–100, by ethnicity

Note: ONS figures based on death registrations up to 15 December 2021 for deaths involving COVID-19 that occurred between 24 January 2020 and 1 December 2021 of people aged 30–100 years that could be linked to the 2011 Census and General Practice Extraction Service Data for Pandemic and Planning Research. These figures are provisional.

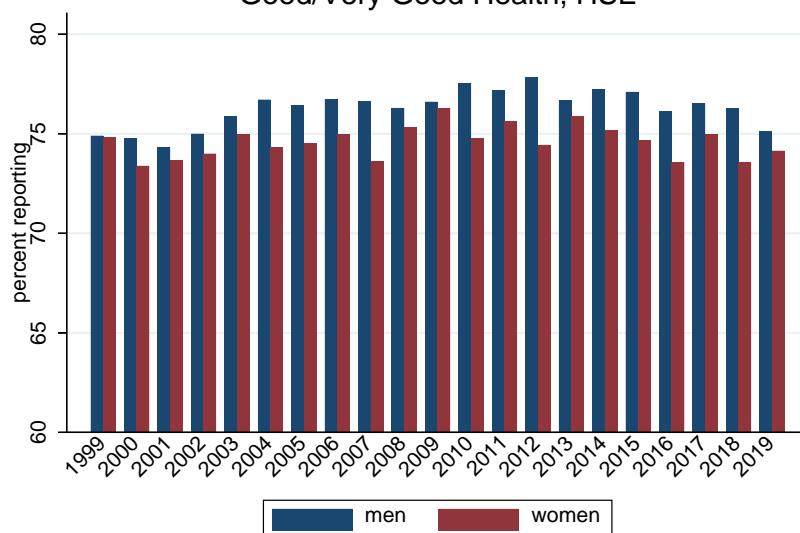
The break between pre- and post-pandemic racial and ethnic mortality patterns was also seen in the US. Pre-COVID education–mortality gradients in the US were maintained or increased slightly during the pandemic, similar to the deprivation–mortality gradients in the UK before and during it. In both countries, COVID-19 brought marked changes to racial/ethnic mortality patterns. Immigrants, who historically had better health and longer lives, were on average less well protected against COVID-19 than were native-born people (Rodriguez-Diaz et al., 2020). In the US, Hispanics were disproportionately hit; in Britain, black and Asian groups bore the brunt of COVID-19. Occupation likely contributes to these patterns. Pakistanis and Bangladeshis in the UK and non-Hispanic blacks and Hispanics in the US are, for example, over-represented in healthcare support occupations (US Department of Health and Human Services, 2017), where risks increased during the pandemic.

In some dimensions, such as those highlighted by IMDs, the COVID-19 pandemic cemented pre-existing disparities, while in others, such as ethnicity and nativity, it opened up new disparities.

Morbidity

Self-assessed health. Nearly three-quarters of men and women in England report themselves to be in 'good' or 'very good' health. The percentage of adults reporting good health grew from 2000 to 2009 (a period in which life expectancy was rising), but has fallen since 2010 (a period in which gains in life expectancy stalled). Figure 17 shows that, in each year of the past two decades, a higher percentage of men than women report good health – a finding that holds in most wealthy countries. The puzzling finding that women are less likely to report good health but are also less likely to die at any age is reconciled by differences in the distribution of chronic conditions faced by men and women. Women are more likely to suffer from pain, which affects self-assessed health, while men are more likely to suffer from smoking-related conditions (emphysema, cardiovascular disease, respiratory cancers) that carry higher mortality risk (Case and Paxson, 2005).

Figure 17. Self-assessed health in England, 1999–2019
Good/Very Good Health, HSE



Source: Health Survey for England.

The fraction of men reporting themselves in 'bad' or 'very bad' health has held relatively stable in England over the past two decades, with an average of 6.8% reporting poor health, but the fraction of women reporting poor health rose from an average of 6.7% in the decade 2000–09 to 7.4% in the decade 2010–19 (Appendix Figure 6).

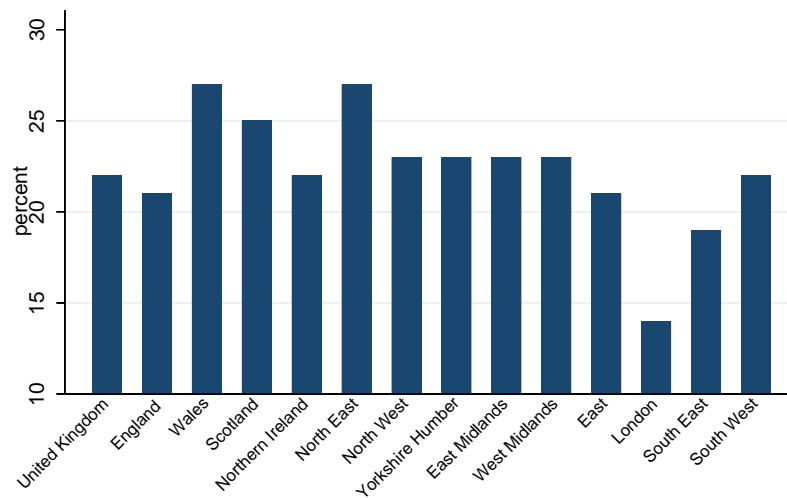
Disability. The Equality Act 2010 defines a disability as 'a physical or mental impairment that has a "substantial" and "long-term" negative effect' on a person's ability to carry out normal daily activities.¹⁰ By this definition approximately one in five people in the UK is disabled (Figure 18), with rates in Wales and Scotland (27% and 25%) higher than that in England (21%), and rates in England markedly higher in the North East (27%) than in London (14%). The most common impairments are those to mobility (7 million people) and stamina/breathing/fatigue (5.1 million). Mental health disabilities have been rising, and are estimated to have affected 4.1 million people in 2019–20. Mental health impairment is the largest single impairment among working-aged adults.

Figure 19 presents a breakdown of disability in the UK by sex and age. The differences in disability rates between the countries of the UK and between the regions of England, presented in Figure 18, are due in part to differences in population–age profiles of constituent countries, and regions of England. The median age in the North East (41.7 years) is substantially higher than that in London (35.8), for example.

Adults living with a disability, on average, have poorer educational outcomes, some of which likely comes from difficulties in obtaining education among those with long-term disabilities. Among adults in the UK aged 21–64, those with a disability are 40% less likely than non-disabled persons to have completed a degree as their highest educational qualification (23.0% versus 39.7%) and almost three times more likely to have no qualifications (15.1% versus 5.4%) (Office for National Statistics, 2021a). Qualifications among those with disabilities have been improving since 2014 (Office for National Statistics, 2022d), but the differences between those with and without disabilities remain stark, which puts the disabled at risk for poorer outcomes along many dimensions.

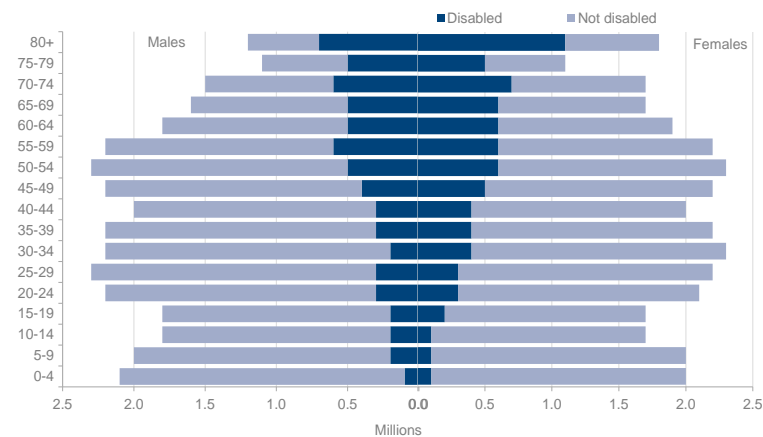
¹⁰ <https://www.gov.uk/definition-of-disability-under-equality-act-2010>.

Figure 18. Disability prevalence in the UK, by country and region, 2019–20



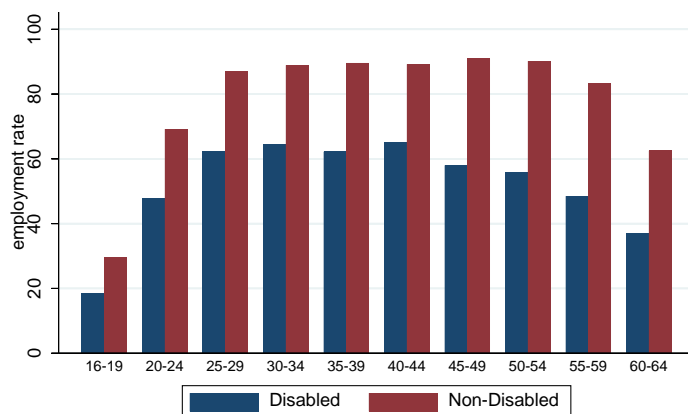
Source: Department for Work and Pensions, Family Resources Survey for financial year 2019 to 2020.

Figure 19. Disability prevalence in the UK, by age and sex, 2019–20



Source: Department for Work and Pensions, Family Resources Survey for financial year 2019 to 2020.

Figure 20. Employment rates, by age and disability status



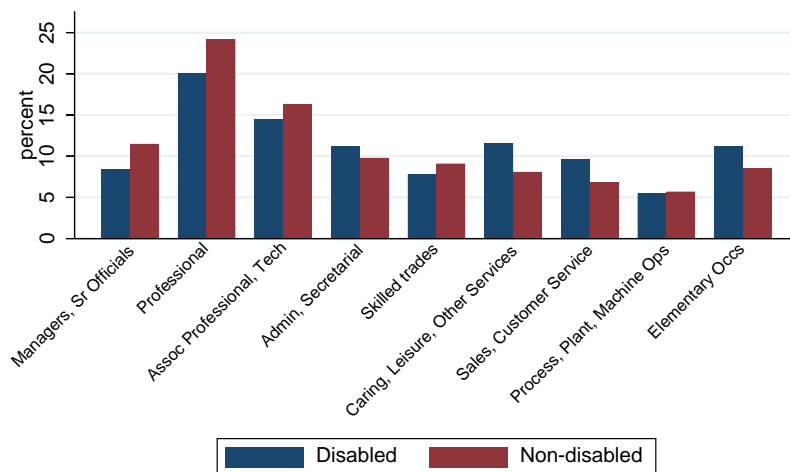
Note: Ages 16–64, UK, year ending June 2021.

Source: Office for National Statistics, 2022d.

There is a large employment gap between disabled and non-disabled people, shown in Figure 20. At all ages, disabled people are less likely to be employed, with gaps largest for those above age 45. Employment gaps vary by disability; people with severe or specific learning difficulties, autism or mental illness are the least likely to be in employment.

Disabled people in employment are under-represented in higher-status occupations (managerial, professional, technical, skilled trades) and over-represented in lower-status occupations (service and elementary occupations), as shown in Figure 21.

Figure 21. Occupation of working disabled and non-disabled people



Note: Ages 16–64, UK, year ending June 2021.

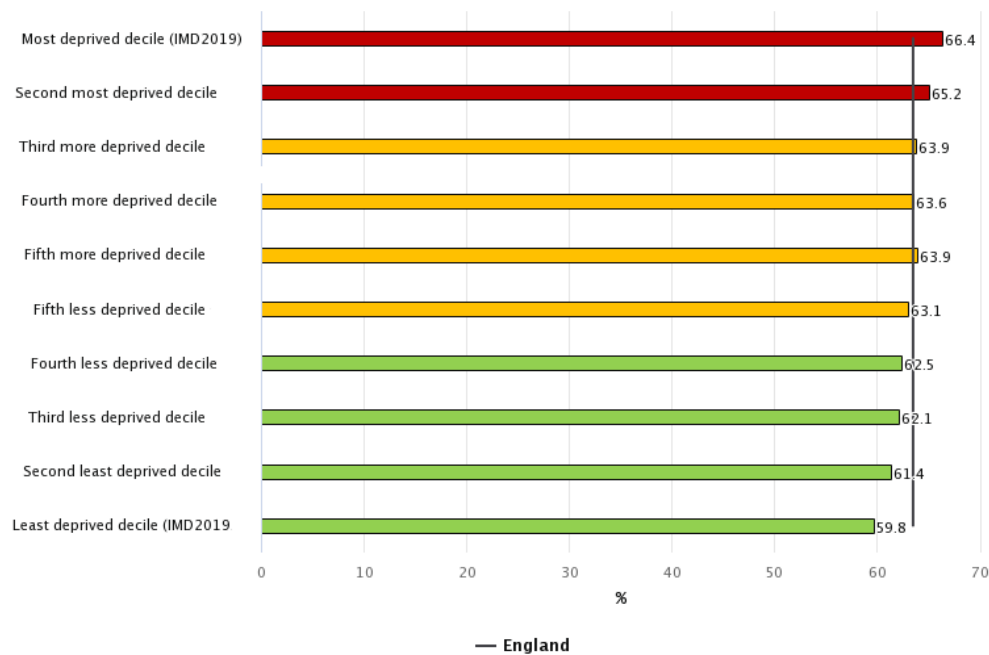
Source: Office for National Statistics, 2022d.

Disabled people are much more likely to report feelings of loneliness 'often or always' than are the non-disabled (15.1% versus 3.6%) – even though measures of civic engagement and volunteerism are quite similar for disabled and non-disabled people. Disabled people score more poorly on measures of well-being – happiness, life satisfaction, meaning and purpose in life, and anxiety.

Obesity. Between 1993 and 2019, the prevalence of adult obesity (Body Mass Index (BMI) >30) nearly doubled in England, from 15% to 28% of the population, and the percentage overweight or obese (BMI >25) rose from 53% to 64% (Baker, 2021).

The majority of adults at every level of educational qualification were of *excess weight* (either overweight or obese) in 2020–21, although those with a degree (level 4 qualification or higher) were at lower risk (59%) than those with no or low qualifications (68–71%).¹¹ Moreover, adults living in the most deprived decile were more likely to be overweight or obese in 2020 (66%) than those in the least deprived decile (59%). Figure 22 shows the obesity gradient with respect to IMD decile rank in 2020–21.

¹¹ https://fingertips.phe.org.uk/profile/public-health-outcomes-framework/data#page/7/gid/1000042/pat/159/par/K02000001/ati/15/are/E92000001/iid/93088/age/168/sex/4/cat/-1/ctp/-1/yr/1/cid/4/tbm/1/page-options/car-do-0_ine-ct-108_ine-pt-0_ine-yo-1:2020:-1:-1.

Figure 22. Percentage of adults (aged 18+) classified as overweight or obese in England, 2020–21

Note: District and UA deprivation deciles in England (IMD2019, 4/21 geography). Red: worse than the average for England. Yellow: similar to the average for England. Green: better than the average for England.

Source: Office for Health Improvement and Disparities, 2022.

Research in many wealthy countries has found poorer people more likely to be obese (Branca, Nikogosian and Lobstein, 2007). A variety of non-competing explanations are offered for this phenomenon, many relating to the marketing of low-cost energy-dense foods in poorer areas and to the more sedentary lifestyles of poorer people. Policy proposals to counter growing obesity often focus on nutrition education, stricter guidelines for food marketing, improved access to healthier foods in poorer communities, and increased availability of facilities for physical activity in poorer areas. It may be difficult, however, to address the obesity epidemic without recognising the role that food plays as balm to the soul. People with more resources have outlets to reduce depression, stress and anxiety that are not available to poorer people. Instead of soothing the beast with talk therapy, tennis, and vacation days – strategies available to wealthier people – poorer people may use food to combat the effects of poor mental health.

That said, it is clear that excess weight is a problem in all IMD deciles, at all education levels, for men and women, and for almost every ethnic group. The majority of adults in every ethnic group (with the exception of the Chinese) were of excess weight in 2020–21. The proportion of blacks either overweight or obese exceeds that of white British (72% versus 65%), but white British are at higher risk than other white, Asian, mixed, and Chinese ethnic groups (Office for Health Improvement and Disparities, 2022).

Hypertension, obesity and the increased risks of cardiovascular disease. For decades, researchers have argued that higher rates of obesity would lead to higher rates of cardiovascular disease and CVD mortality (Powell-Wiley et al., 2021). However, it is not clear why, for nearly two decades, as obesity rates rose in the UK, CVD mortality rates fell rapidly for those aged 45–74, with an average decline of 4% per year from 1993 to 2012, before abruptly stalling. It

is possible that interactions with the healthcare system – being tested for diabetes, hypertension and high cholesterol, and being adherent to anti-hypertensive medications and statins – held at bay some of the negative effects of excess weight. It will be important to investigate whether changes in healthcare delivery or changes in health-related behaviours, such as adherence to medications among those at risk of CVD, can explain why CVD mortality rates stopped falling, and whether those with a lower level of educational qualification or those living in more deprived areas became less able or less willing to protect against the risks caused by obesity.

From 2003 to 2019, the prevalence of uncontrolled or untreated hypertension has fallen in each ten-year age group from 25–34 to 55–64 (Appendix Figure 7), as has the proportion of prime-aged adults with total raised cholesterol (Health Survey for England, 2019). These findings deepen the mystery of why CVD mortality rates stopped falling. It is possible that those who, in 2019, remain hypertensive have more severe hypertension than those who were hypertensive in 2003. This is an area where additional analysis is encouraged.

Risk behaviours. Reducing avoidable mortality requires greater success in reducing unhealthy behaviours – smoking, heavy drinking, physical inactivity and poor diet. Individually, these behaviours are associated with higher mortality risk, even with controls for occupational social class, BMI and blood pressure. Engaging in multiple risk behaviours concurrently adds to the increased risk of death (Kvaavik et al., 2010), and in England, as elsewhere, risk behaviours do commonly co-occur (Poortinga, 2007).

Poortinga (2007) specifically examines *clustering* – risk behaviour combinations that are more frequent than predicted if they were independent – using data from the Health Survey for England (2003) and focusing on the risk behaviours noted above. He finds that while men are more likely than women to report multiple risk behaviours, stronger clustering of behaviours is found among women, specifically the combination of smoking, heavy drinking and poor diet. Clustering suggests multiple-behaviour interventions (in contrast to single-behaviour interventions) may be more effective in reducing such behaviours, and in turn reducing avoidable mortality.

People residing in lower-occupational-class households (III–V) are more likely to engage in two or more risk behaviours compared with occupational classes I and II (professionals and intermediate occupations). Also, those with no qualifications are more likely to engage in multiple risk behaviours compared with those who attended higher education (Buck and Frosini, 2012). Risk behaviours provide part of the through-line from education and social class to mortality.

Mental health. In England, an Adult Psychiatric Morbidity Survey is conducted every seven years, and has assessed mental health using consistent methods in 1993, 2000, 2007 and 2014. One in six adults reported recent symptoms of a common mental health problem, such as depression or anxiety, when surveyed in 2014 (McManus et al., 2016), with prevalence higher among women (21%) than men (13%). In adults aged 16–64, there has been a long-term steady upward trend in the prevalence of severe symptoms of common mental disorders (CIS-R scores of 18 or above¹²), rising from 6.9% in 1993 to 9.3% in 2014. Documented increases from 1997 to 2000, and 2000 to 2007, occurred during a period of relatively high GDP growth in the UK, and prevalence continued to rise through the financial crisis and beyond. This pre-existing trend makes it difficult to

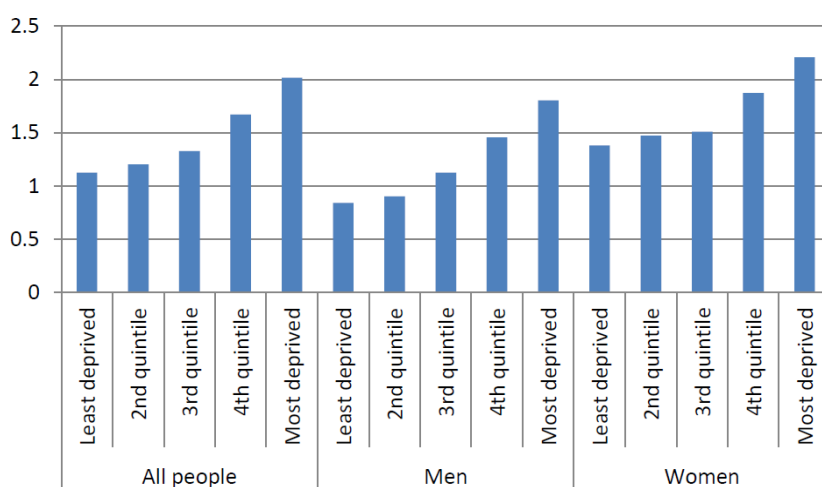
¹² The NHS notes that a CIS-R score of 18 or above suggests 'more severe or pervasive symptoms of a level very likely to warrant intervention such as medication or psychological therapy' (<https://digital.nhs.uk/news/news-archive/2016-news-archive/survey-shows-one-in-three-adults-with-common-mental-disorders-report-using-treatment-services>).

attribute increases in the prevalence of severe symptoms to the financial crisis; the root causes eroding mental health appear to be older.

Mental disorders are more prevalent among those living alone, those in poor physical health and those not employed. We do not attempt to assess the direction(s) of causality here.

Among those aged 50 and above, there are large gaps in the prevalence of depression by quintile of local area deprivation (Figure 23). In the English Longitudinal Study of Ageing, those in the most deprived IMD quintile scored twice as high as those in the least deprived quintile, on average, on a battery of questions asked to elicit risk for depression (CESD-8) (Matthews et al., 2018). (We note that the Health Deprivation and Disability Domain of the IMD contains a marker for mood and anxiety disorders.)

Figure 23. Mean CESD-8 (depression index) scores, by IMD quintile, ages 50+



Source: Matthews et al., 2020.

There is often a vicious circle between poor mental health and risky health behaviours. Drugs, alcohol and cigarettes may relieve symptoms of anxiety and depression, at least in the short run. At the same time, addiction to substances can lead to anxiety and depression. (See Case and Deaton (2020) and references there.) Both addiction and depression are correlates of suicide.

Public Health England reports that individuals with poor mental health die 10–20 years earlier on average than the general population, and that smoking is the largest contributor to this life expectancy gap. In 2014–15, the prevalence of smoking was 16.4% in the general population, but 28% for those suffering from anxiety or depression and 34% among those with a long-term mental health condition.¹³

Mental health and COVID-19. The COVID-19 pandemic has been associated with a substantial rise in symptoms of mental ill health. Banks and Xu (2021) document changes in markers of mental health between wave 9 of the panel study Understanding Society (data collected in 2017–19) and a special module of the study conducted in April 2020, covering the beginning of the pandemic. Women of all ages and younger men saw large increases in reported poor mental health. The

¹³ <https://publichealthmatters.blog.gov.uk/2020/02/26/health-matters-smoking-and-mental-health/>.

groups with a higher prevalence of poor mental health pre-crisis – women, and younger adults – suffered the largest deterioration in mental health.

4. How do health inequalities develop and why do they persist?

Introduction

To understand why mortality rates differ between groups, or why some groups are more likely to suffer from chronic conditions, it is useful to begin at the beginning, with prenatal and childhood health. Inequalities in health in early life can set children on different health trajectories for the remainder of their lives. Ill health in childhood can compromise education, and thus subsequent earnings, or even compromise ability to work. In these and other ways, inequalities in childhood health interact with many of the inequalities covered in this Review, including education, other elements of early childhood development, and geographic and economic inequalities.

There are several overlapping schools of thought on the contribution of prenatal and childhood health to health outcomes in adulthood. These schools vary in the weight they place on the forces at work, and differ in the emphasis placed on events at different ages, but all find agreement in the importance of early-life health. The 'fetal origins hypothesis' emphasises the role of health *in utero* for health over the life course, noting that while some developmental and health outcomes caused by a poor uterine environment will be observed immediately after birth and may persist, other poor outcomes may not become visible until midlife. (Joseph and Kramer (1996), Rasmussen (2001) and Almond and Currie (2011) review this literature, and discuss the difficulties of isolating the impact of the uterine environment on health.) A second school of thought emphasises the cumulative effects of health in early life, which affect physical, social and economic well-being over the life course. Cable (2014) introduces this literature in epidemiology; Cattán et al. (2022) provide a careful discussion of these issues for this Review.

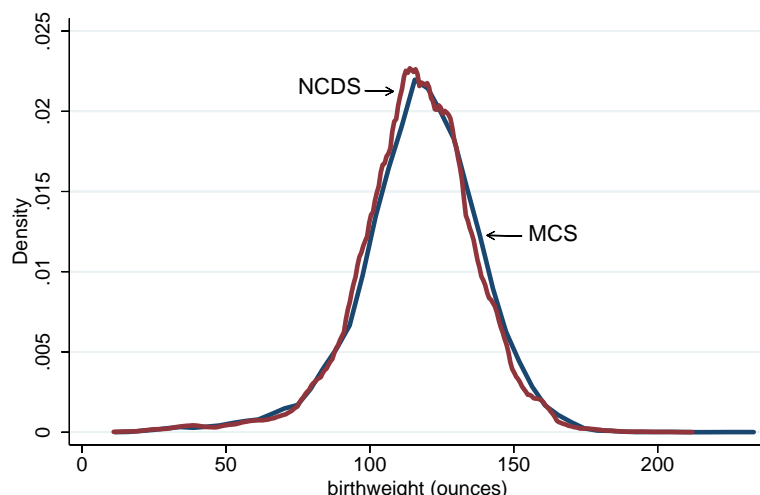
The British birth cohort studies are well suited to the examination of how health inequalities developed and why they persist. Complementary to Cattán et al. (2022), who present evidence from the 1970 British Cohort Study and the 2000–02 Millennium Cohort Study (MCS), we here examine health inequalities in two birth cohorts, born four decades apart. The National Child Development Study (NCDS) has followed all children born in one week of March 1958 from birth to age 55 – by which time chronic health conditions become more prevalent and more severe. (Additional data, from age 62, are currently being collected. Information on the NCDS is available at <https://cls.ucl.ac.uk/cls-studies/1958-national-child-development-study/>.) Using the NCDS, we can link health in childhood to developmental and educational outcomes, and to health and mental health outcomes in adulthood. We also draw on the 2000–02 MCS, to examine changes in child health between the 1958 and 2000 cohorts. This allows us a better understanding of how much progress has been made in 42 years, both in protecting children's health and in ameliorating the impact of poorer health in childhood. In addition, the use of two birth cohorts allows the opportunity to see whether health correlates found in one study are observed repeatedly. One study alone tells us about children born *at one point in time*; repeated findings of the same phenomena using multiple cohort studies add to the generalisability of results found.

Inequalities in childhood health

Figure 24 shows there was remarkably little change in the distribution of birthweights for children born in the NCDS (1958) and the MCS (2000). We can only speculate on why there has been so little change over a period when Britain saw so much progress in health, and in income. The proportion of births by Caesarean delivery increased in the UK (Black, Kaye and Jick, 2005); in the US, Caesarean delivery has been associated with earlier delivery and smaller infants

(Tilstra and Masters, 2020). Children in the MCS were, on average, slightly heavier at birth (mean birthweight = 117.5 ounces) than those in the NCDS (116.2 ounces). However, a slightly higher fraction of children in the MCS were born at low birthweight (below 88.2 ounces) – 7.8% in the MCS, compared with 7.6% of children in the NCDS.

Figure 24. Distribution of birthweights in the NCDS (1958) and MCS (2000)

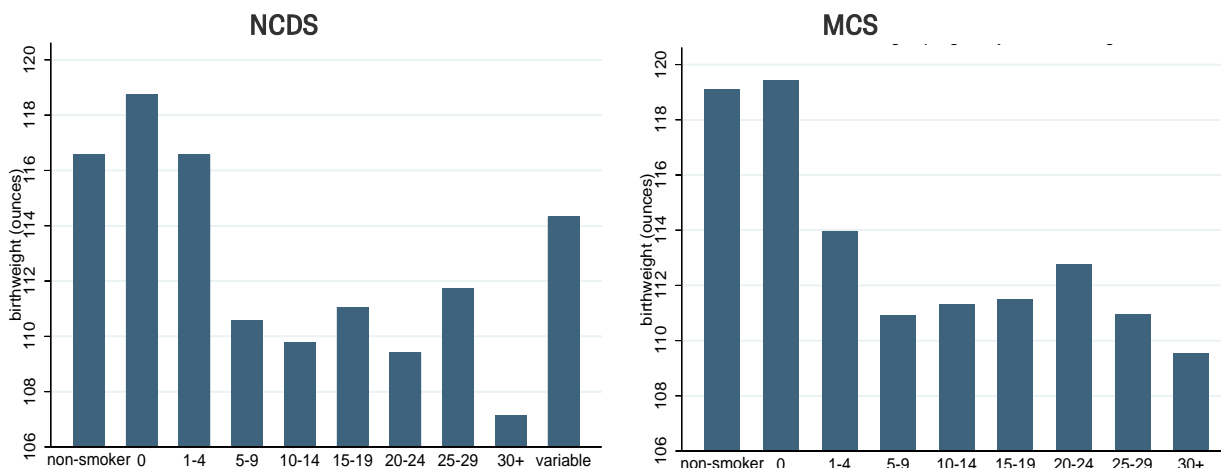


Note: Epanechnikov kernel, bandwidth = 2.3321.

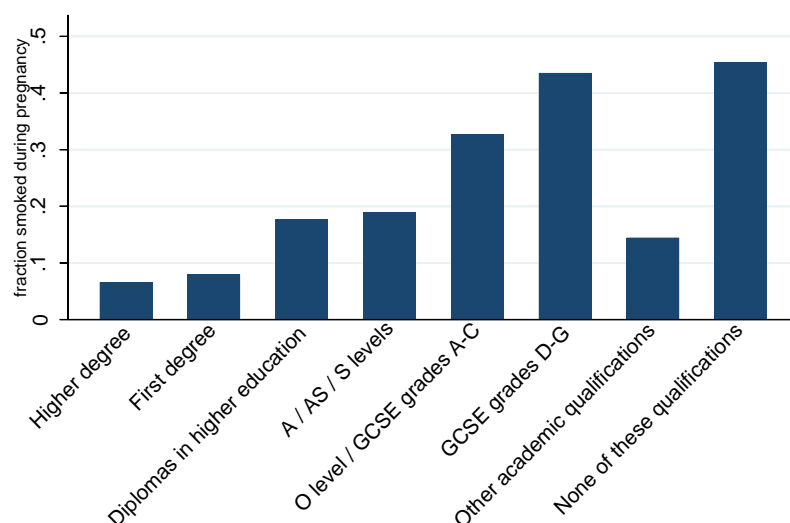
Source: NCDS birth sweep; MCS age 9 months sweep.

Birthweight also varies with maternal behaviours. Figure 25 presents average birthweight by categories of maternal smoking during pregnancy, for mothers in the NCDS (left panel) and the MCS (right panel). In the NCDS, cohort members born to mothers who were not smokers, or who smoked fewer than five cigarettes a day, were 4.5 ounces heavier at birth on average than those born to women who smoked more heavily during their pregnancies. Although reports of smoking while pregnant have fallen in England since 2000 (NHS England, 2019), a higher fraction of mothers in the 2000 MCS cohort reported smoking during their pregnancy (29%) than was true in the 1958 NCDS cohort (14%). The association between prenatal smoking and child birthweight did not change in the four decades between the birth cohorts.

Figure 25. Mothers' reports of smoking during pregnancy in the NCDS (1958) and MCS (2000)



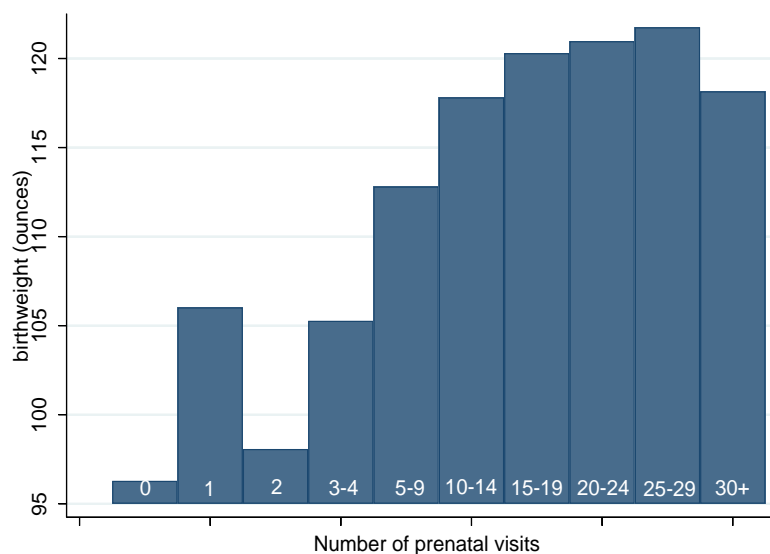
Source: NCDS birth sweep; MCS age 9 months sweep.

Figure 26. Maternal smoking during pregnancy, MCS

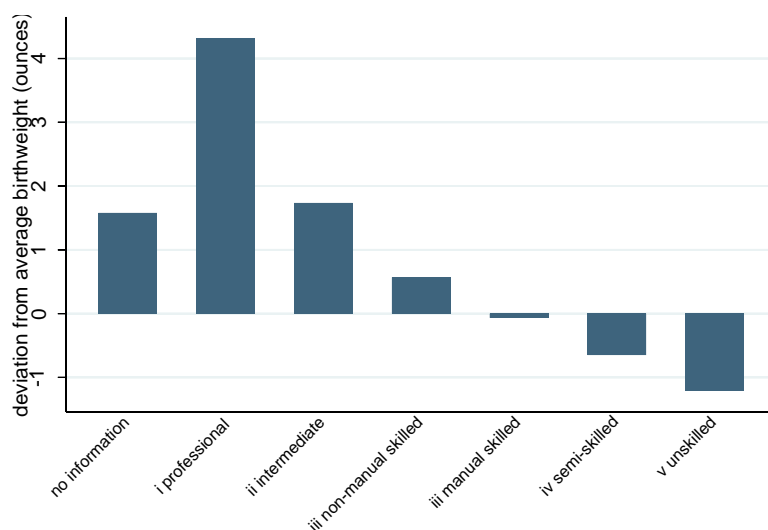
Source: MCS age 9 months sweep.

There is a gradient of prenatal smoking with respect to mother's highest educational qualification in the 2000 MCS cohort, which is apparent in Figure 26. This is one (of several) mechanisms fuelling the intergenerational transmission of poor health, and poor outcomes.

Figure 27 demonstrates that prenatal clinical visits were positively associated with birthweight in the 1958 cohort. While being born at higher gestational age gives women a chance to have more prenatal visits, which would induce a correlation between prenatal visits and birthweight, the pattern continues to be seen with controls for gestational age. In the 2000–02 MCS, birthweight is negatively and significantly correlated with the time to first prenatal visit during pregnancy.

Figure 27. Prenatal visits and birthweight, NCDS

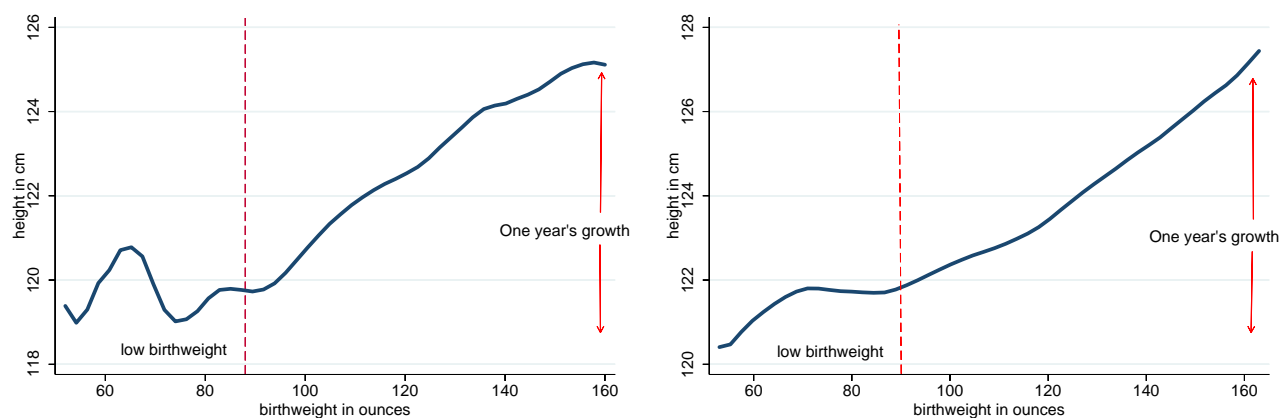
Source: NCDS birth sweep.

Figure 28. Birthweight and father's social class, NCDS

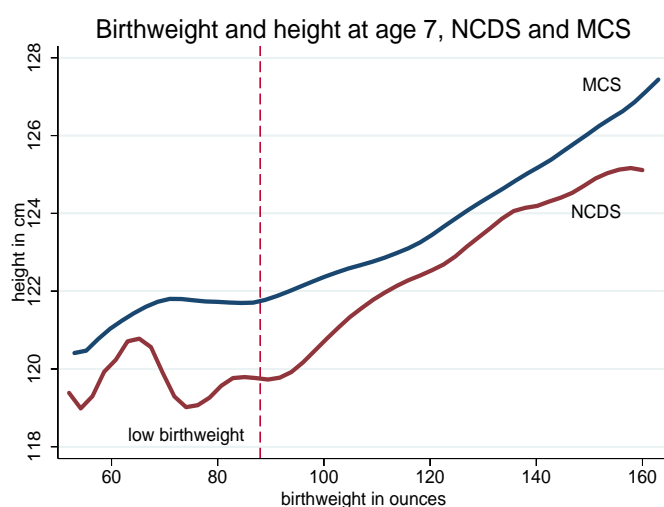
Source: NCDS birth sweep.

In the NCDS, birthweight is also significantly associated with father's social class at the time of the birth. Figure 28 presents deviations from the 1958 birth cohort's average birthweight (in ounces) by the Registrar General's social class categories. Children born to professionals were, on average, 5.5 ounces heavier at birth than those born to unskilled manual workers. In the NCDS, all three sets of variables – maternal smoking, prenatal visits and father's social class – are significantly correlated with birthweight when added simultaneously to a regression of birthweight regressed on gestational age and its square, and child sex (Appendix Table 1).

Figure 29 shows the strong, positive association between birthweight and child height at age 7, in the 1958 cohort (left panel) and in the 2000 cohort (right panel). In both cohorts, the difference in height at age 7 between children born at 80 ounces and those born at 160 ounces is on average 6 centimetres, which is the average amount of height that children are expected to gain *each year* before reaching puberty. Birthweight remains significantly correlated with height in adulthood, with controls for sex and mother's and father's heights.

Figure 29. Birthweight and height at age 7

Source: NCDS birth and age 7 sweeps; MCS 9 months and age 7 sweeps.

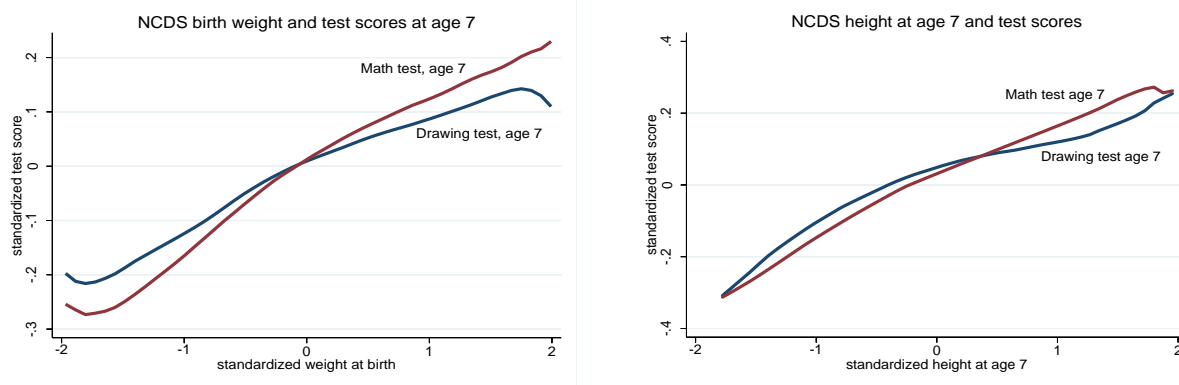
Figure 30. Progress in children's heights between 1958 and 2000

Source: NCDS birth and age 7 sweeps; MCS 9 months and age 7 sweeps.

At each birthweight, children born in the 2000–02 MCS cohort are taller on average at age 7 than were children born four decades earlier, in the 1958 NCDS cohort. Figure 30 shows the progress that was made in children's heights between the two cohorts.

Height in childhood is a marker for health and nutrition. Children who are well nourished and healthy in early life experience an advantage in both physical and cognitive function (see Case and Paxson (2008 and 2010) and references there). Throughout childhood, birthweight and height are strongly correlated with cognitive function, as measured by test scores (Case and Paxson, 2010). Figure 31 shows the association in the NCDS between test scores and birthweight (left panel) and between test scores and height at age 7 (right panel). Similar results obtain at age 11; these associations persist. In the NCDS, test scores at ages 7 and 11 are significant correlates of final educational qualifications, and earnings, in adulthood.

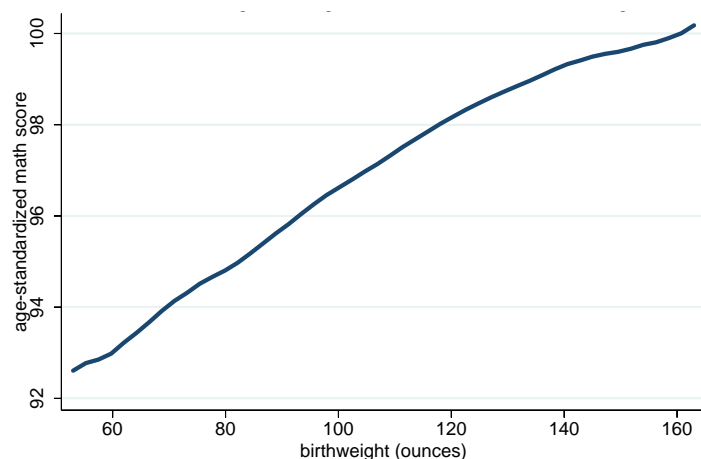
Birthweight and height advantages continue to appear among children born four decades later, in the 2000 MCS cohort. Figure 32 shows the association between birthweight and scores on maths tests taken by the MCS cohort at age 7.

Figure 31. Birthweight, height at age 7 and test scores at age 7, NCDS

Source: NCDS birth and age 7 sweeps.

Together, Figures 31 and 32 show that for both the 1958 cohort and the 2000 cohort, as birthweight (or height) increases, cognitive test scores increase, on average, and that this relationship holds throughout the range of birthweight (height).

Figure 32. Birthweight and age-standardised maths score at age 7, MCS



Note: Birthweight from 1st to 99th percentile.

Source: MCS age 9 months and age 7 sweeps.

Table 2. Childhood height and test scores, NCDS

Dependent variable	Coefficient on height for age z-score at age 7	
	Controls for age, sex and ethnicity	With additional controls
Reading score at age 7	0.154 (0.008)	0.109 (0.009)
Maths score at age 7	0.123 (0.008)	0.081 (0.009)
Drawing score at age 7	0.112 (0.008)	0.076 (0.009)
Verbal language score at age 11	0.170 (0.009)	0.109 (0.009)
Non-verbal language score at age 11	0.179 (0.009)	0.117 (0.009)
Maths score at age 11	0.184 (0.009)	0.120 (0.009)
Copy design score at age 11	0.077 (0.009)	0.047 (0.010)

Note: Additional controls are for child and household circumstances: an indicator for low birthweight, an indicator for prenatal smoking, the height of each parent, indicators for parents' school-leaving ages, indicators for parents' social class at the time of the birth, and the log of family income when the child is 16. Standard errors are presented in parentheses.

Source: Case and Paxson, 2008.

Significant associations between childhood height and test scores continue to appear, even when one controls for individual and household circumstances that are correlated with both. Table 2, drawn from Case and Paxson (2008), shows results for the association between height and cognitive scores for 1958 cohort members. Each number is from a different regression, for a different cognitive score (listed in the left-most column), regressed on standardised height at age 7, with controls for the age of the child at the time of measurement, a complete set of indicators for ethnicity, and sex. The results in the last column add controls for child and household circumstances: an indicator for low birthweight, an indicator for prenatal smoking, the height of each parent, indicators for parents' school-leaving ages, indicators for parents' social class at the time of the birth, and the log of family income when the child is 16. These extended controls will absorb part of the association between height and cognitive scores that could be attributed to other factors – indeed, height at age 7 depends in part on birthweight and maternal smoking. Standard errors are presented in parentheses.

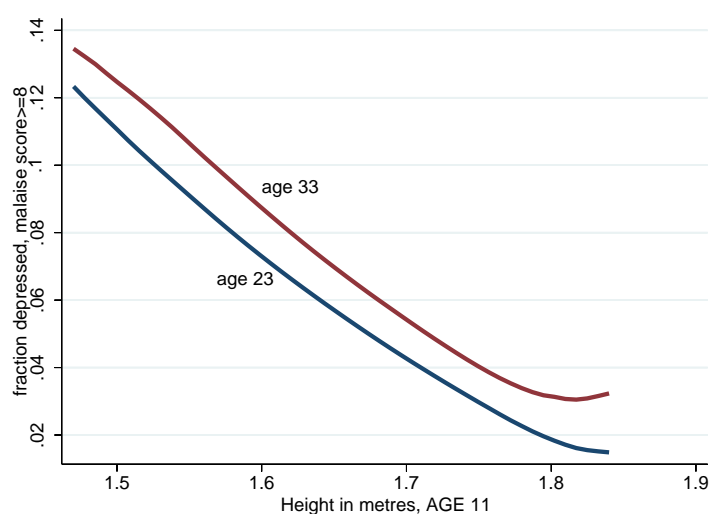
Height is significantly associated with all test scores, even with extended controls for individual and household circumstances. The controls reduce the association between height and cognitive tests by a third to a half, but they do not remove height as a marker for the importance of uterine environment and early-life health.

Childhood health and adult outcomes

Childhood health and adult earnings. Children born at higher birthweight are, on average, taller through childhood and adulthood (Appendix Figure 8). As adults, taller people on average have higher earnings. In the 1958 birth cohort, surveyed at age 33 and age 42, each inch of height is associated with a 2–2.5% increase in gross hourly earnings. This is true for both men and women. In the literature, this is referred to as the 'height premium'. However, when test scores of cohort members at ages 7 and 11 are added to the analysis, the height premium is cut in half for both men and women. Test scores in childhood explain approximately a third of the variance in log hourly gross earnings. By contrast, controls for other childhood circumstances – mother's and father's school-leaving ages, mother's and father's social class, low birthweight, mother's and father's heights, and log family income – explain only 5% of the variance in earnings for men and 10% of the variance for women. In the absence of childhood test scores, height is a significant correlate of earnings – because it is a marker for cognitive function. But in the presence of childhood test scores, height plays little role in explaining differences in earnings. (See Case and Paxson (2008) for details.)

This is a case in which roots of adult earnings inequality can be traced to childhood, in particular to childhood health and nutrition. Being well nourished and healthy affects both height and cognition – the ability to reach physical and cognitive potential – which leads to a better life trajectory.

Mental health. That childhood health bears on mental health in adulthood can be seen in Figure 33, which tracks the association between depression at ages 23 and 33 and a cohort member's height at age 11 in the NCDS. (Depression is identified using a 24-question instrument, and reporting eight or more issues related to malaise is scored as depression.) Of those who score in the 'depressed' range at age 33, 27% report worrying about their health, and many report pain (back pain 60%, headaches 52%, eye pain 24%, rheumatism 15%). Among those who do not score in the depressed range, reports of pain are less than half as high (e.g. back pain 21%, headaches 12%).

Figure 33. Childhood height and adult depression, NCDS

Source: NCDS age 11, age 23 and age 33 sweeps.

Tables 3 and 4 show the relationship between individuals' malaise scores at age 33 and height at age 7, test scores at ages 7 and 11, cohort members' highest qualifications and fathers' occupations at birth. Without controls for test scores and educational attainment, height at age 7 is significantly related to malaise for both women and men. However, height in childhood loses most of its significance once test scores at age 7 are included in the regression. Test scores in childhood continue to be significant correlates of depression in adulthood, even conditional on the cohort members' highest educational qualification.

Table 3. Women's malaise scores at age 33, 1958 NCDS

Explanatory variables	(1)	(2)	(3)	(4)
Height age 7 (standard error)	-3.91 (0.78)	-2.55 (0.80)	-1.68 (0.87)	-1.34 (0.87)
F-test: scores age 7 (p-value)	-	42.26 (0.000)	4.04 (0.007)	2.55 (0.054)
F-test: scores age 11 (p-value)	-	-	20.35 (0.000)	6.50 (0.000)
F-test: highest qualification age 33 (p-value)	-	-	-	11.49 (0.000)
F-test: father's occupation at birth (p-value)	6.27 (0.000)	3.17 (0.000)	2.09 (0.012)	1.71 (0.052)
Observations	4,596	4,440	3,952	3,888

Note: Test scores at ages 7 and 11 are for reading, maths and drawing. Fathers' occupations are provided in 14 categories, including a category of 'single, no husband'.

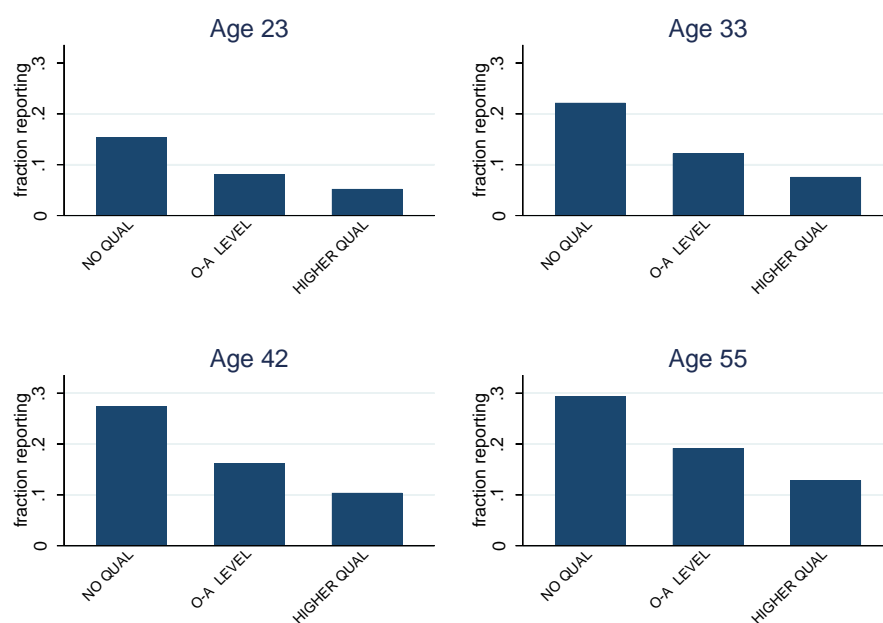
Allen and Donkin (2015) report that, in the 1958 NCDS birth cohort, the probability of early death (before age 50) was 60–80% higher for men and women who had had two or more adverse childhood experiences relative to those who had experienced none. Within this birth cohort, the risk of suicide was heightened by the experience of emotional adversity at age 7.

Table 4. Men's malaise scores at age 33, 1958 NCDS

Explanatory variables	(1)	(2)	(3)	(4)
Height age 7 (standard error)	–1.87 (0.71)	–1.13 (0.72)	–0.80 (0.76)	–0.53 (0.77)
F-test: scores age 7 (p-value)	-	30.65 (0.000)	2.37 (0.069)	1.10 (0.347)
F-test: scores age 11 (p-value)	-	-	14.03 (0.000)	6.58 (0.000)
F-test: highest qualification age 33 (p-value)	-	-	-	7.00 (0.000)
F-test: father's occupation at birth (p-value)	3.11 (0.000)	1.44 (0.1333)	1.09 (0.358)	0.99 (0.460)
Observations	4,473	4,308	3,846	3,756

Note: Test scores at ages 7 and 11 are for reading, maths and drawing. Fathers' occupations are provided in 14 categories, including a category of 'single, no husband'.

Figure 34. Fair or poor health in NCDS, by educational qualification and age



Source: NCDS.

Health in adulthood. Members of the 1958 birth cohort are more likely to report themselves in only 'fair' or 'poor' health as they age. This is not particularly surprising. The arrival of chronic conditions, and bodily wear and tear, take a toll. However, there is a marked difference in the prevalence of reporting poor health by educational qualification. At age 55, cohort members with a high level of education are less likely to report poor health than were those with low qualifications at age 23 (Figure 34).

Summary

As seen above, there are many channels through which early-life health and circumstance can affect educational attainment. In turn, both early-life health and educational attainment may affect health and economic outcomes across the life course. Birth cohort analyses can help us to further unpack these relationships, and shed light on the role of health in the intergenerational transmission of deprivation. That said, the British birth cohort studies are not large enough for many analyses (e.g. when studying less-common causes of death) and the waves of each study occur at fairly wide intervals – the 1958 cohort is seen at ages 23, 33, 42 and 55. The intervals between waves are far from ideal to study, for example, the timing between the onset of a disability and job loss. Annual administrative data, merged with decadal census data and with death records for the adult population, would allow for an even richer set of analyses.

5. Conclusions

Why study health inequalities?

The documentation of social and economic deprivation has often been a powerful force for change. Most people understand that material resources are unequally distributed – though the depths of poverty often still have the power to shock – but it is not as widely appreciated that there are systematic differences in health and in life expectancy across groups. The years that a person can expect to live is only one of many indicators of well-being, but it is surely one of the most important. In the US today, where life expectancy is rising for the more educated while falling for the less educated, it is clear that something is seriously wrong, something that is much starker and where the findings are less challengeable than widening differences in material well-being. People may be less poor than they seem to be, but they are not less dead than they seem.

Beyond eliciting sympathy, it is not clear what to do with the evidence on health inequalities. One recurrent thread is that the health of the most favoured group – the richest or most educated – shows what is potentially attainable for everyone, so that gaps in health can be attributed to inequality itself. Marmot (2015) makes such a claim: 'if everyone in England over 30 had the same low mortality rate as university-educated people, there would be 202,000 fewer deaths each year – almost half the current total'. He adds that these deaths come from 'a preventable condition: health inequality'. This is excellent arithmetic, but it does not in itself tell us much about how to close the inequalities, and save all of those lives. The field of social epidemiology seeks to find the social (and economic) causes of differences in mortality and morbidity. As in this chapter, it turns out to be much easier to document the differences in health than it is to identify the causal mechanisms in society that produce these differences. Almost everywhere we look, we find that people who are deprived in one space – facing income poverty, poor educational opportunities, or racial discrimination – suffer other deprivations too – particularly in health, so that there is always the temptation to argue that eliminating differences in health could be achieved by eliminating income and educational differences and systemic racism, and ensuring that everyone has good housing.

It is also true that health is not the only thing that people care about and some might prioritise other goals more heavily. This is not to deny that behaviour and lifestyles are themselves conditioned by the social environment, nor do we wish to blame the poor for behaving poorly. Yet, we cannot command people's priorities. Certainly, public policy can emphasise the dangers of drinking or of smoking, but we would not want to deprive people of what may be their most affordable pleasures. If so, the complete elimination of health inequalities is neither possible nor desirable.

Even if elimination is our goal, getting to paradise is no easy matter; without better understandings of mechanisms, we cannot design effective policy. The more modest and more realistic question is whether we can identify specific mechanisms that are amenable to policy changes that are short of implementing a manifesto for an egalitarian utopia.

One such mechanism that we have discussed here is what we might call the 'austerity' account, that the policies of austerity – cutting services, including health services – that were followed after 2010 have affected the most deprived places in Britain the most, and have slowed or stopped the increase in life expectancy that would otherwise have taken place. This is exactly the sort of thing that we are looking for but, as we have said above, the thesis is far from obviously correct, and there are several facts that it appears not to explain.

For the US, Case and Deaton (2020) argue that changes in labour markets, brought on in part by the cost of American healthcare, have been an ongoing disaster for less educated Americans over the last half century. This may or may not be correct, but if it is, there are policies that would help, such as reducing the cost of healthcare, financing it through something other than a flat-rate tax on employment, and stopping the subsidisation of excessive automation. Giving working people more power through unions would help too. Clearly, not all of this applies to Britain.

Another line of work in Britain, discussed extensively above, is the examination of health differences across areas ranked by measures of deprivation. We understand the appeal of this, but we do not believe that this work is helpful in identifying mechanisms that could be used to improve things, because the links between instruments and outcomes are hopelessly tangled by aggregation, scaling and ranking. It might be time to assess whether deprivation indices should be replaced by other metrics that would give researchers a better chance of pinpointing the characteristics in the social and economic environment that hold the most promise for policymakers. Adding education, race and ethnicity to death certificates would seem a good place to start. We hope that the imminent release of the census linked to death records will enable much more convincing and more useful connections to be drawn between the lives people lead and their health outcomes.

One area where there already has been important work on health inequalities in Britain is the collection and use of the birth cohort studies. Those have yielded important findings on the importance of early-life environment on health and material achievement in adulthood and, together with work on early-life education, have informed policy aimed at ensuring that the deprivations of each generation are not passed on to the next.

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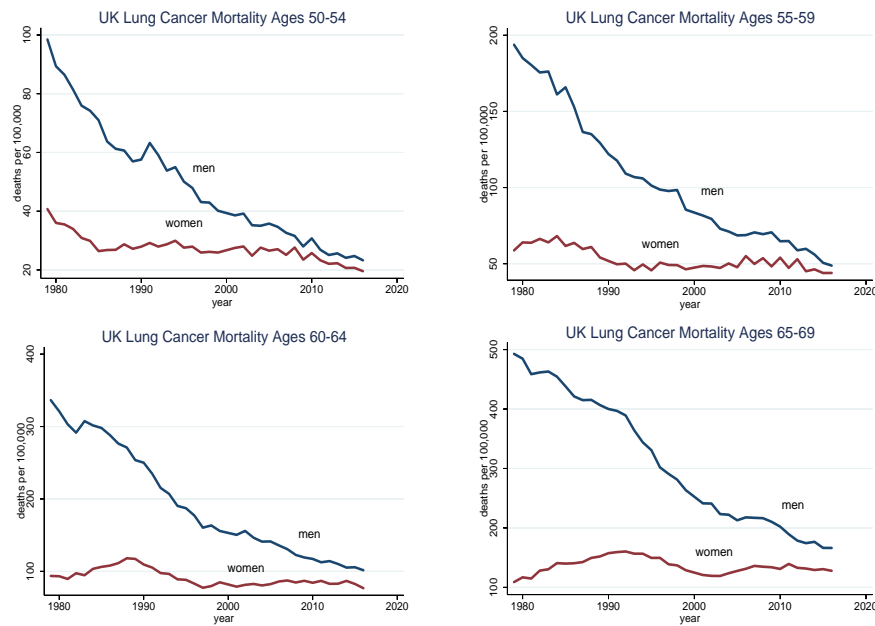
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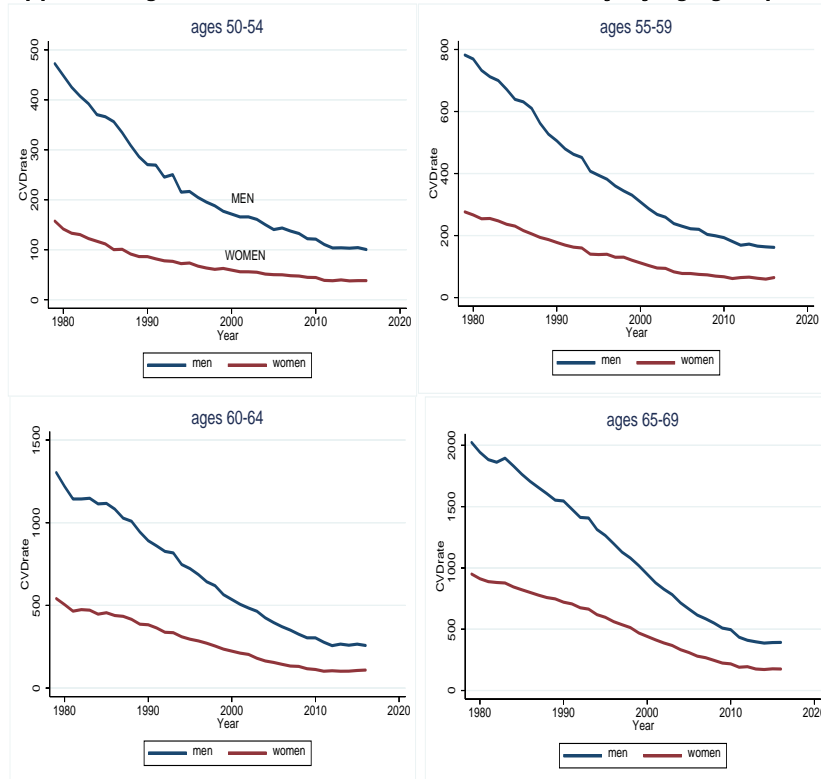
Appendix. Additional figures and table

Appendix Figure 1. Lung cancer mortality, by age group, UK



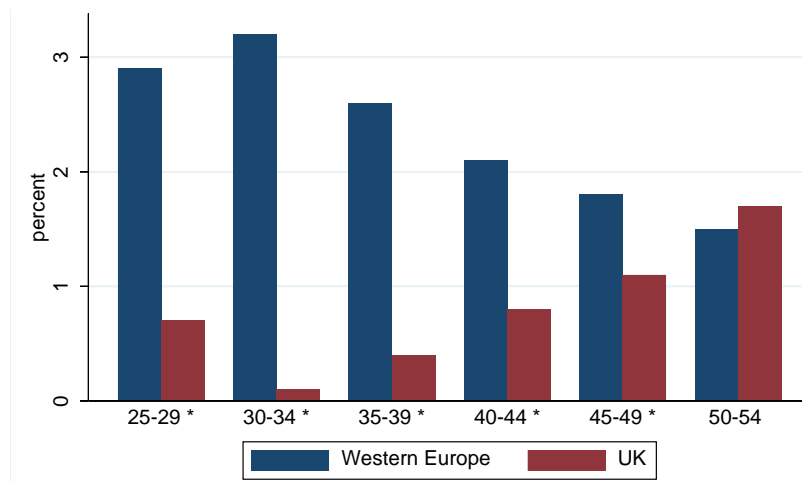
Source: WHO Mortality Database. Individual countries within the UK show the same patterns separately.

Appendix Figure 2. Cardiovascular disease mortality, by age group and sex, UK



Source: WHO Mortality Database.

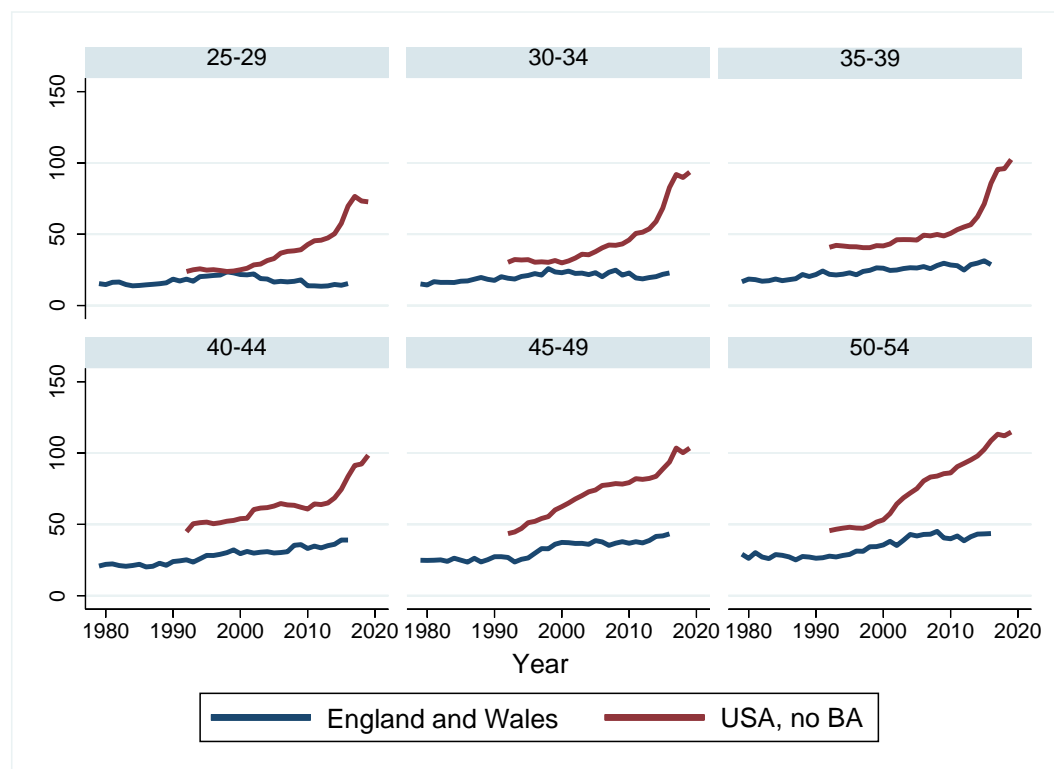
Appendix Figure 3. Average annual rate of decline in mortality, by age group, UK and Western Europe, 1990–2009



Note: Comparison countries include Belgium, Denmark, France, Germany, Ireland, Italy, Netherlands, Norway, Portugal, Spain, Sweden and Switzerland.

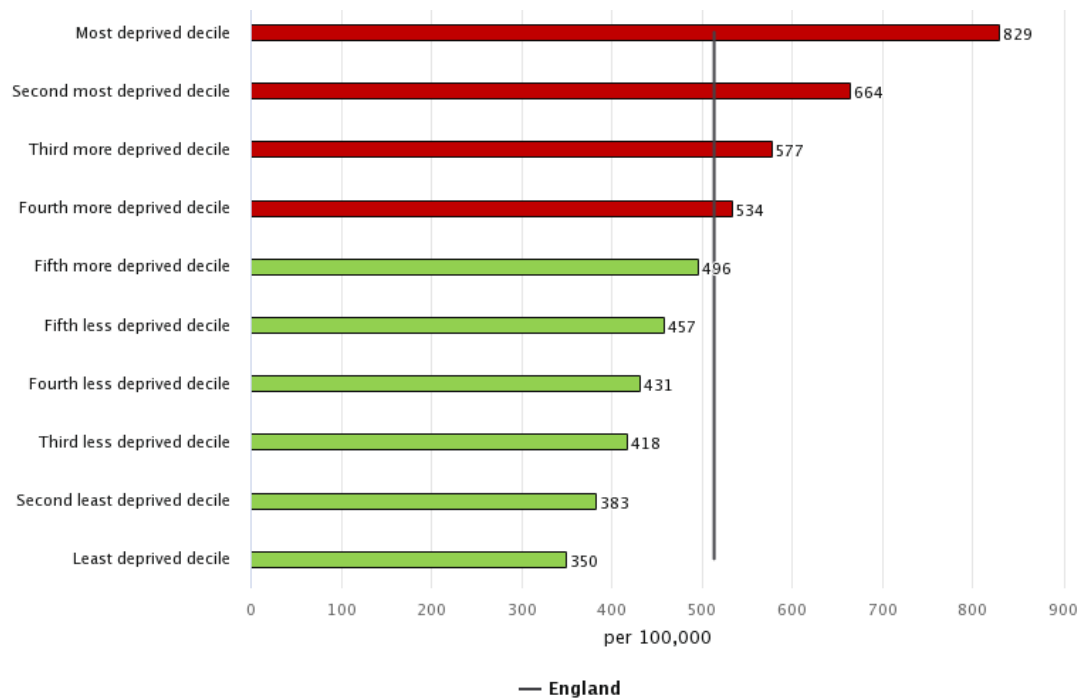
Source: WHO Mortality Database.

Appendix Figure 4. Drug, alcohol and suicide mortality, England and Wales in comparison with less educated Americans, by five-year age group



Source: WHO Mortality Database and US National Vital Statistics System.

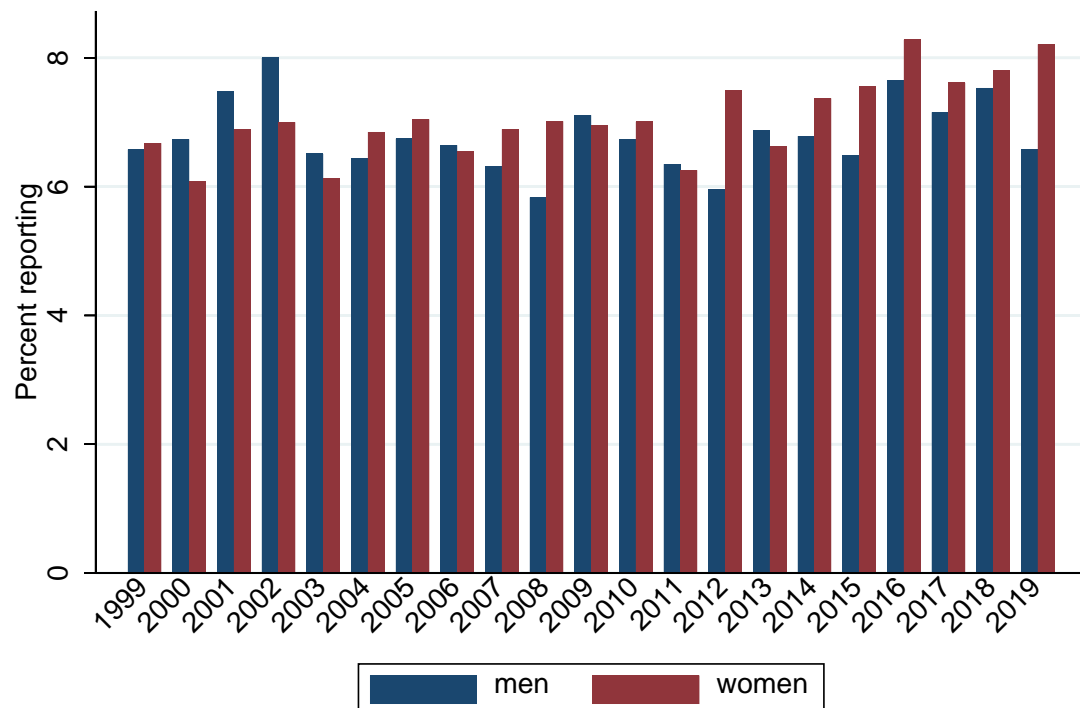
Appendix Figure 5. Alcohol-related hospital admissions, 2018–19, by IMD decile in England



Note: LSOA11 deprivation deciles in England (IMD2019). Red: worse than the average for England. Green: better than the average for England.

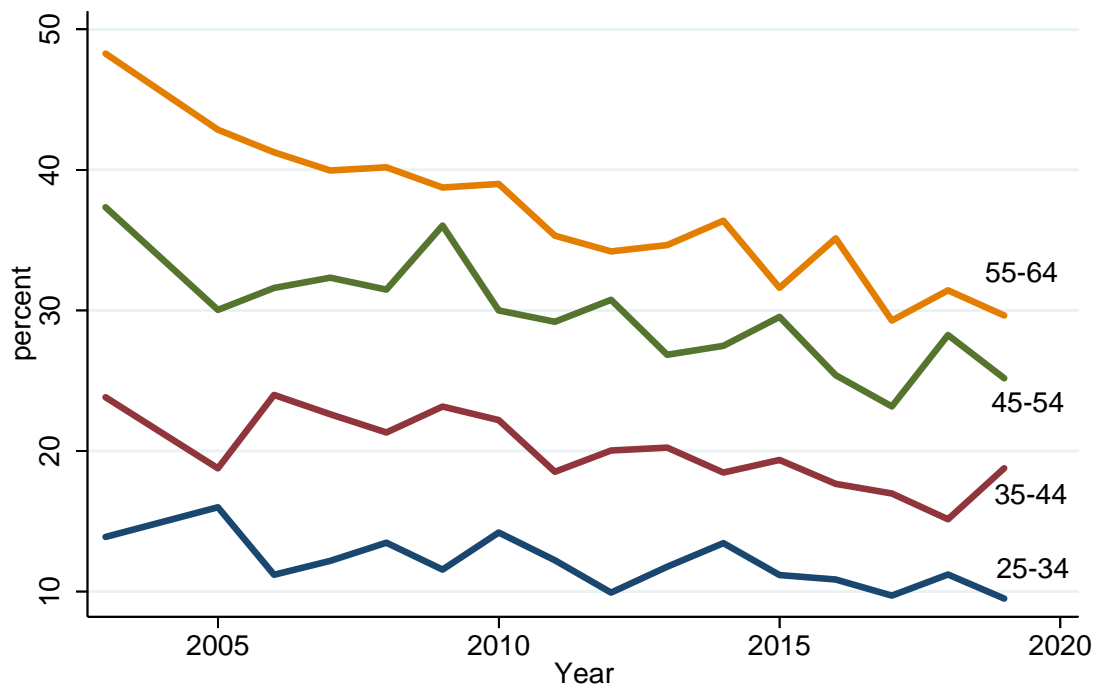
Source: Office for Health Improvement and Disparities, 2022.

Appendix Figure 6. Self-assessed health, percentage bad / very bad, 1999–2019



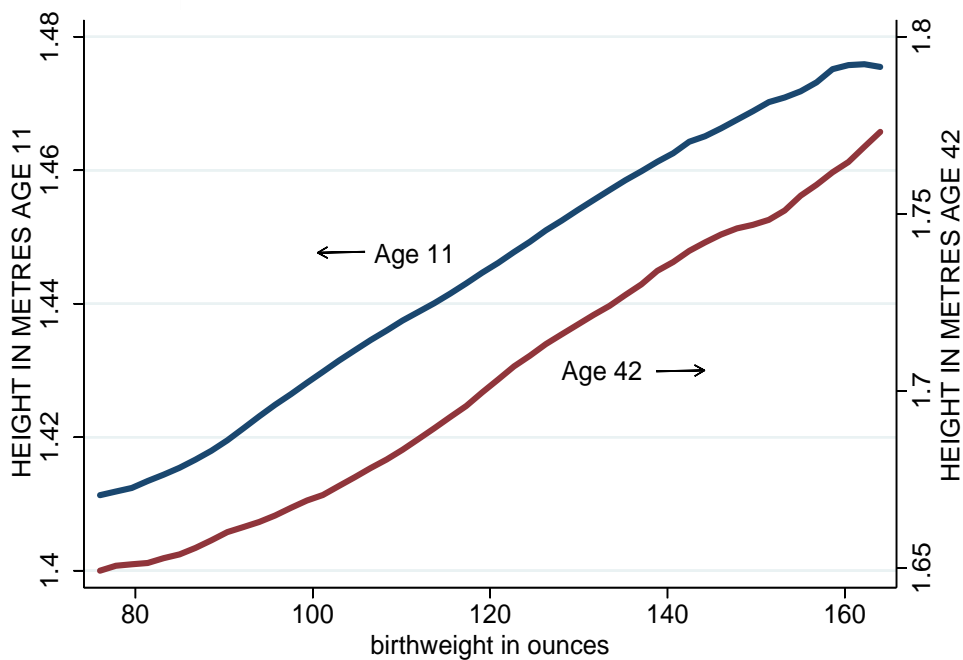
Source: Health Survey for England.

Appendix Figure 7. Uncontrolled or untreated hypertension, 2003–19



Source: Health Survey for England.

Appendix Figure 8. Birthweight and height at ages 11 and 42



Source: National Child Development Study birth, age 11 and age 42 sweeps.

Appendix Table 1. NCDS birthweight = f(prenatal visits, cigarettes, father's occupation)

	(1)	(2)
<i>F</i> -test: prenatal visits (<i>p</i> -value)	-	8.68 (0.0000)
<i>F</i> -test: cigarettes when pregnant (<i>p</i> -value)	-	8.77 (0.0000)
<i>F</i> -test: father's occupation at birth (<i>p</i> -value)	7.63 (0.0000)	5.89 (0.0000)
Number of observations	14,380	14,380

Note: Regressions control for child's sex, gestational age in days and gestational age squared.

Source: National Child Development Study birth sweep.