What we know, can know and need to know about the causes of health inequality

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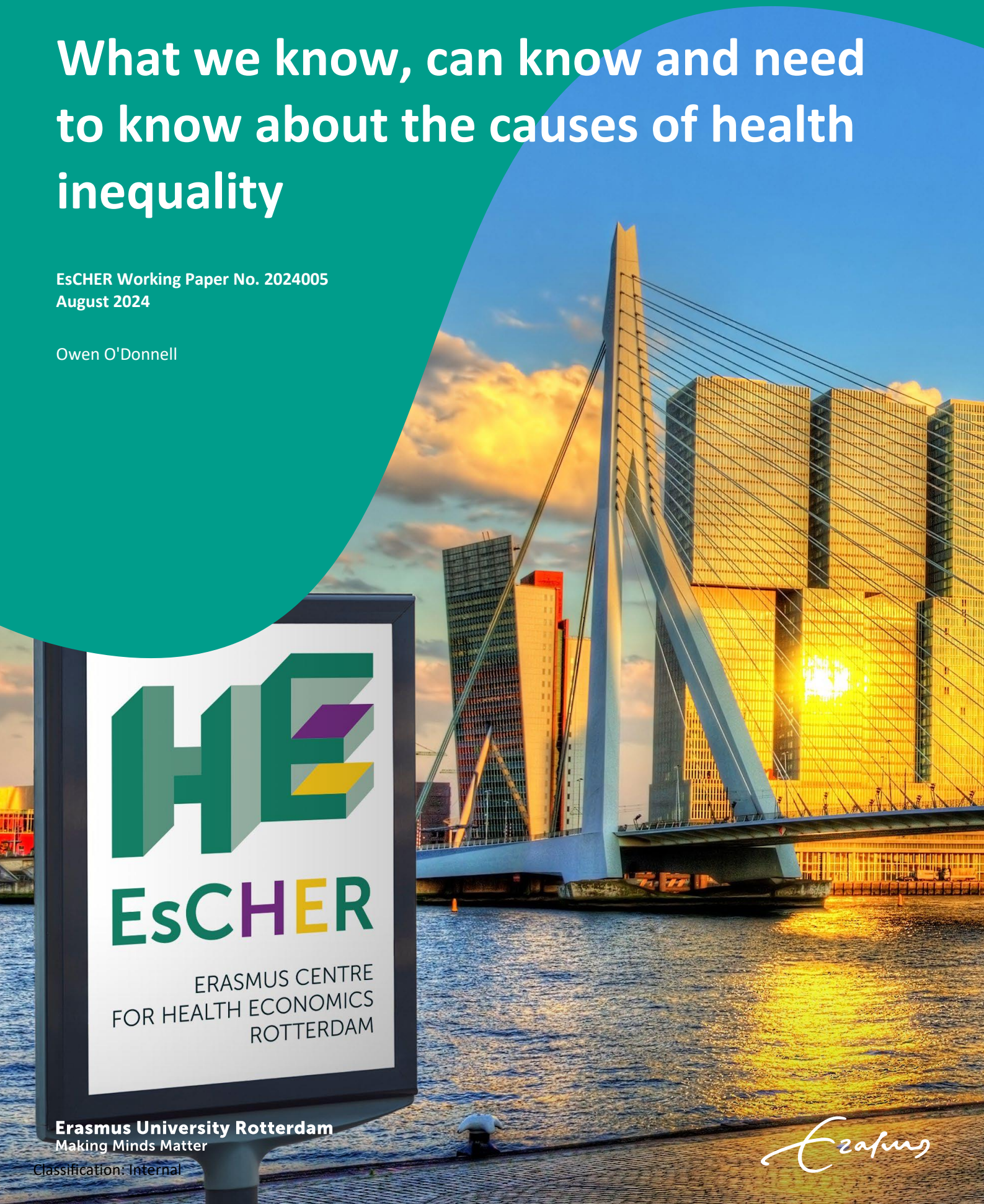

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What we know, can know and need to know about the causes of health inequality[1]

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Abstract

Socioeconomic health inequality is substantial, ubiquitous and persistent. This chapter reviews what is known about its causes in high-income countries and considers what can be known and needs to be known. Causal analyses have not delivered strong, consistent evidence that education, income and wealth impact health in adulthood but have shown that cash benefits paid to low-income households often improve infant and child health outcomes. Changes in adult health have large effects on income and wealth, and childhood ill-health both persists into adulthood and constrains economic outcomes in that phase of life. What can be known about the causes of health inequality is constrained by the limited scope for causal analysis to identify effects of socioeconomic exposures (including the relative concept of status) that potentially take their toll on health over the life course, cumulatively and multiplicatively. To reduce health inequality, its causes need not necessarily be known, provided health policies that improve the health of the socioeconomically disadvantaged can be identified and implemented. Political support for such policies may, however, depend on knowledge (or beliefs) about the causes of health inequality.

Keywords: Health, Inequality, Education, Income, Wealth, Socioeconomic Status, Causal Analysis

JEL classification: I12, I14

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1. Introduction

Health is positively associated with education, income and wealth. Across 21 high-income countries in 2016, 25-year-old female college graduates could expect to live 5.3 years longer than their contemporaries with no more than compulsory schooling (Lübker and Murtin 2023). The respective life expectancy gap was 8.3 years for males. For both sexes, the gap increased by around half a year between 2011 and 2016 (ibid.). In the United States (U.S.), the richest one percent of 40-year-olds could expect (in 2014) to outlive the poorest one percent by about 10 years for females and 15 years for males (Chetty et al. 2016). These gaps are also widening (Bosworth et al. 2016; Chetty et al. 2016; National Academies of Sciences, Engineering & Medicine 2015; Schwandt et al. 2022). Among older Americans, gaps in health and life expectancy by wealth are large and forecast to increase (Bavafa et al. 2023; Hudomiet et al. 2021). When the 1933-38 birth cohort reached the age of 55, (median) female life expectancy was 82.3 years in the bottom wealth quintile group and 89.9 years in the top (Hudomiet et al. 2021). For those born 20 years later, the respective life expectancy was 1.4 years lower in the bottom quintile and 6.4 years higher in the top.

Socioeconomic health inequality – *heath inequality*, hereafter – is substantial, ubiquitous and persistent (Case and Kraftman 2022; Cutler et al. 2012; Evans et al. 2012; O'Donnell et al. 2015). A common lament is that while health inequality is all too evident, its causes are elusive. This is unsurprising since causality plausibly runs in both directions – low income constrains investment in health and ill-health constrains earnings capacity, for example – and there is ample scope for confounding from genes, parental investments, early-life environment, time preference, risk attitudes and more.

This chapter reviews evidence on the causes of health inequality and considers the role of such evidence in the motivation and formation of policy. It does not aim to provide a systematic and comprehensive review of the evidence. That would be a colossal task. Effects are expected to

differ across both socioeconomic exposures and health outcomes. And the effect of a given exposure on a particular outcome may well be specific to context. Attention is restricted to associations between health and each of education, income and wealth in high-income countries. In each case, mechanisms that may generate causality in each direction are identified and the balance of the respective evidence is weighed.

Definitive conclusions about the causes of health inequality are not offered. The aforementioned heterogeneity of effects is one reason. Another is the limited scope for causal analysis to identify them. What we can know about the causes of health inequality is constrained by plausibly long lags between socioeconomic exposures and the emergence of any health effects and by the potential for the exposures to take their toll on health cumulatively and multiplicatively. However, these limitations on learning about the causes of health inequality need not form an insurmountable barrier to reducing it. The health gap between the socioeconomically advantaged and disadvantaged could be narrowed by prioritizing the latter in the distribution of healthcare and public health resources. Success with this approach requires knowledge of health interventions (not socioeconomic determinants) that are effective in improving health of socially disadvantaged groups. Societal willingness to prioritize the health of these groups may, however, be contingent on what causes them to be in worse health.

The next three sections examine health inequality by education, income and wealth. While the starting point in each case is variation in health by the purported socioeconomic determinant, causal mechanisms and evidence for an effect from health to each of education, income and wealth are also discussed. In each case, there is reason to expect this *reverse causality* and empirical support for it. This does not rule out socioeconomic determination of health. The fifth section considers the hypothesis that health is determined by socioeconomic status rather than by the absolute level of a single socioeconomic exposure, such as education, operating in isolation. The relative and multidimensional nature of socioeconomic status leave only limited

scope to test this hypothesis with formal causal analysis. The sixth section considers the policy relevance of the evidence and its limitations. Contrary to intuition, it argues that the causes of health inequality need not be uncovered in order to reduce it. Irrespective of what causes the socially disadvantaged to be in worse health, their health could be improved, and their health deficit reduced, by giving them priority in the allocation of effective healthcare. Associations between health and economic outcomes can also be weakened by more generous insurance and income transfers to the sick and disabled. The final section is a summary.

2. Health inequality by education

2.1 Education → Health

Theory. If health behavior is rational and involves combining time with healthcare and other health-improving goods to produce health, then education is expected to improve health by raising the efficiency of health production (Grossman 1972). Education provides generic skills – literacy and numeracy – that facilitate the interpretation and use of health information and the acquisition of health knowledge. It may also enable effective communication with physicians and raise responsiveness to medical advice. Through these skills, education can increase health generated by any given input (technical efficiency) and by the appropriate combination of inputs (allocative efficiency) (Lochner 2011). This reduces the shadow price of health investment and so increases the demand for health (Grossman 1972). Education can also increase health capital indirectly through the acquisition of human capital (Galama and van Kippersluis 2015). By raising productivity in the labor market, and so the opportunity cost of time lost to sickness, education gives an added incentive to invest in health, provided health is produced by more than time (Grossman 1972). By raising lifetime earnings potential, education also reduces the likelihood of becoming rationally addicted to a health-harming good that would jeopardize earnings capacity (Becker and Murphy 1988). Beyond individualistic models of

health behavior, education may impact health (positively or negatively) through exposure to peers (Fischer et al. 2021; Gaviria and Raphael 2001; Sacerdote 2011).

While it is reasonably straightforward to postulate broad, although rather vague (efficient health production?), mechanisms through which education may impact positively on health, it is also easy to think of confounders that may contribute to a positive association. Stronger time preference, for example, is expected to lower investment in both human and health capital (Fuchs 1982). Longitudinal data from Sweden reveal that individuals observed to be less patient in adolecence have worse school performance and lower educational attainment (Golsteyn et al. 2014). They also have more hospitalizations and diagnoses of (lifestyle-related) health conditions in adulthood and are more likley to die before the age of 65 (Norrgren 2022). Other potential confounders are genes that bestow advantage in both health and educational attainment, parental investment in both health and human capital, and early-life cognitive and noncognitive skills that facilitate acquisition of both types of capital.

Empirics. Identification of a causal effect of education on health requires that confounders be controlled, modelled, purged or rendered irrelevant by leveraging plausibly exogenous variation in education. The first strategy, which requires rich (longitudinal) data that include observations of elicited (time) preferences or early life environment/cognition/behavior, generally finds that controls weaken the health-education association without eliminating it (Bijwaard et al. 2017; Heckman et al. 2018; Van der Pol 2011). This begs the question of whether more controls would further weaken the association. Control for IQ eliminated the education-longevity association among Dutch males (Bijwaard and Jones 2019).

The (structural equation) modelling strategy, which requires rich data on some confounders and reliance on assumptions about their relationships with unobserved ones, also tends to find strong selection into (more) education on correlates of future health, although selection usually does not account for all of the association (Bijwaard et al. 2015; Conti et al. 2010; Hong et al.

2020). One study finds no selection and a positive education-health association even in a high IQ population (Savelyev 2022).

Comparison of differences in health between twins that differ in education can purge confounders arising from family background and, with identical twins, genes. This strategy delivers mixed evidence (Amin et al. 2015; Behrman et al. 2011; Lundborg et al. 2016; Madsen et al. 2010; Savelyev et al. 2020). It suffers from limitations arising from small samples, amplification of measurement error bias, the potential for health-correlated unobserved differences between twins distinguished by education and doubts about external validity given the peculiarity of twins that differ in education.

The last strategy – leveraging policy and institutional sources of variation in education, mostly from compulsory schooling laws (CSL) – tends to engender most confidence, at least with respect to internal validity. Unfortunately, it also delivers inconclusive evidence. Some CSL studies find a positive effect of education on longevity (Davies et al. 2018; Lleras-Muney 2005; Van Kippersluis et al. 2011), while others find no effect (Albouy and Lequien, 2009; Clark and Royer 2013; Mazumder 2008; Meghir et al. 2018). In fact, analysis of the same CSL using different data and methods has produced contradictory results on the longevity effect of schooling in both England and Wales (Clark and Royer 2013; Davies et al. 2018) and the U.S. (Lleras-Muney 2005; Mazumder 2008). Ambiguity extends to other health outcomes (Arendt 2008; Clark and Royer 2013; Davies et al. 2018; Janke et al. 2020; Mazumder 2008; Silles 2009). The most consistent finding is that a marginal increase in years of schooling appears to reduce the risk of diabetes (Davies et al. 2018; Janke et al. 2020; Mazumder 2008).

More extensive reviews of evidence on health effects of education (Cutler and Lleras-Muney 2008; Grossman 2015; Galama et al. 2020) are also inconclusive and suggest that variation in the evidence may be attributable to differences in measurements of health and education, context defined by place and time, and methods. Contrasting the strong, ubiquitous and

persistent positive correlation between education and health with the lack of robust, consistent estimates of causal effects of education on health, a conservative conclusion would be that confounding contributes to the association.

2.2 Health → Education

Since health in adulthood is usually observed in data collected after the respective individuals have completeed their education, causality running from health to education is not an obvious explanation for the positive cross-sectional association between adult health (and longeivity) and education. However, childhood health conditions may both interfere with human capital acquisition (Lee 2024) and persist into adulthood. Given accumulating and highly consistent evidence of long-run health and socioeconomic (including education) consequences of early-life health conditions (Almond and Currie 2011; Almond et al. 2018; Barker 1990; Bleakley 2010; Currie and Vogl 2013), these conditions are strong candidates to explain part of the education-health gradient, and they potentially bias estimates of the effect of education on adult health.

Childhood illness may interfere directly with the quanttiy and quality of schooling acquired. Indirectly, if ill-health in infancy impedes development of vital organs or leaves a permanent physiological weakness, it may lower life expectancy and so reduce the expected return on investment in human capital (Ben-Porath 1967). Evidence supports the expected positive effect of longevity on education (Baranov and Kohler 2018; Oster et al. 2013; Fortson 2011; Hansen 2013; Jayachandran and Lleras-Muney 2009; Stoler and Meltzer 2013). While this mechanism provides further reason to exercise caution in interpretating education gradients in mortality and adult health, the warning comes with three caveats. First, a U.S. study finds that the "tax" that premature death levies on investment in education is not economically significant (Bleakley 2018). Second, ill-health in childhood and adolesence, by lowering potential earnings in those stages of life where child labor is not uncommon, can reduce the opportunity cost, and

not only the return, of investment in education, leaving the net effect on quantity of education ambiguous (Yamauchi 2008; Bleakley 2010). Third, ill-health in childhood may also change comparative advantage in cognitive relative to physical job tasks, such that the (relative) returns to education rise. This latter effect is demonstrated by a remarkable study of a polio epidemic in Denmark in 1952 (Gensowski et al. 2019). Chilhood survivors who were paralyzed by the disease were more likely to go on to graduate from college, to get white-collar jobs and to do work requiring IT skills than survivors of non-paralytic polio. These findings are consistent with paralysis shifting comparative advantage from *brawn jobs* to *brain jobs*, prompting investmnent in education to gain access to the latter. They demonstrate that childhood ill-health may not only affect education (positively, in this case) but also sorting into occupations, and it can leave its mark on health in adulthood.

Early-life and childhood health problems can persist into adulthood or leave vulnerabilities to disease that lie dormant until middle age (Barker 1990). They can also directly interfere with education and alter incentives to invest in education. Hence, health experience early in life can spark long-run dynamics that generate a complex relationship between education (and occupation) and health in adulthood. Attempts to infer causality from snapshot cross-sectional observations of education and adult health are prone to bias arising from these dynamics. Without phenomenally rich longitudinal data, which fortunately are becoming less scarce, it is a daunting task to deal with such complexity and so uncover the causes of the association between health and education.

3. Health inequality by income

3.1 Income → Health

Theory. If health is a normal good, then demand for it rises with income. It may even rise more than proportionately with income if, as might be expected, the marginal utility of health –

particularly longevity – diminishes less rapidly than the marginal utility derived from other goods (Hall and Jones 2007). Richer individuals, and richer countries, are expected to be in better health because they can afford to spend more on a vital component of wellbeing and the opportunity cost of their health spending is lower because they already have so much of the material joys of life. If high income results from a high wage rate, then richer individuals also have an incentive to remain in good health because sickness costs them more in lost earnings and the opportunity cost of investing in health rises less than proportionately if health is produced from market goods in addition to time (Grossman 1972).

While a positive effect of income on health would be expected under free market allocation of resources, such as healthcare, that determine health, many would judge the resulting pro-rich distribution of health to be unfair (Robson et al. 2024a). This partly explains why, in most countries, the distributions of healthcare and, to a lesser extent, other goods that determine health, such as food and housing, are not left entirely to the market. Stricter institutional constraints on ability to turn money into health, and a higher social safety net that aims to prevent poverty from damaging health, are expected to weaken any effect of household income on health. The extent to which higher income improves health is anticipated to vary across countries with the financing and organization of healthcare and the regulation of other markets.

Effects on adult health. A previous review concluded that consistent evidence from high-income countries of a positive impact of income on health in adulthood is lacking (O'Donnell et al. 2015). It did not rule out that an effect, if it exists, may materialize with a lag that is longer than the observation period of studies undertaken, and so has remained undetected. The evidence base of this conclusion is quasi-experimental, leaving elements of doubt about internal and external validity. Recent U.S. experimental evidence of null health effects of income assuages some of the doubt, at least regarding the question of whether higher income improves the health of low-income American adults (Miller et al. 2024; Yoo et al. 2022). Compared with

a control group of low-income, young (21-40 years) adults in Illinois and Texas who were not receiving mean-tested or disability-related transfers and were given an income supplement of $50 per month for three years, a treatment group who were randomly selected to receive a larger unconditional income supplement of $1000 per month did use more hospital and emergency care and spent (out-of-pocket) around $20 per month more medical care (Miller et al. 2024). However, the treatment group's physical health, assessed through multiple measures and biomarkers, did not improve (relative to the control) and mental health (stress and psychological distress) improved only in the first year. Precisely identified null effects on health outcomes are substantially (and significantly) smaller than respective cross-sectional correlations between income and health. Income effects on preventive care (vaccinations and cancer screening) and health behaviors (exercise and sleep) are also ruled out with a high degree of confidence. The negative results from this study are particularly striking because they are obtained from a sample of low-income adults for whom income may have been expected to be a binding constraint on the production of health in the context of the U.S. health system. At baseline, 29% of the sample were uninsured, 27% reported having forgone medical care because it was unaffordable, and most assessed their health to be no better than *good* (ibid.).

Like the quasi-experimental evidence, the finding of no short-term adult health response to experimentally manipulated income – as well as the absence of health impacts of one-off cash payments made to low-income U.S. households during the COVID-19 pandemic (Jacob et al. 2022; Jaroszewicz et al. 2024; Pilkauskas et al. 2023) – does not rule out that an effect may emerge beyond the observation period of the study. Health gains from income-induced increased utilization and spending on medical care may take years to materialize.

Effects on infant and child health. The lack of consistent evidence of a positive income effect on adult health in high-income countries contrasts with accumulating evidence that income – specifically, (near) cash benefits – improves birth and child health outcomes. Quasi-

experimental evidence from England & Wales (Reader 2023), Spain (Gonzalez and Trommlerová 2022), the U.S. (Almond et al. 2011; Hoynes et al. 2015) and Uruguay (Amarante et al. 2016) suggests that higher benefits paid to pregnant women increase birth weight. A potential mechanism is through the mothers' health. A deviation from the predominant finding of no income effect on health, on average, in the (low-income) adult population is that cash benefits (Earned Income Tax Credit, EITC) appear to improve the self-reported health, lower risky biomarkers and reduce smoking of low- and middle-income American mothers (Evans and Garthwaite 2014; Hoynes et al. 2015; Lenhart 2019), although possibly increasing obesity in this population (Schmeiser 2009).

There is also evidence of long-run health benefits from receipt of (increased) cash benefits early in life. Exposure to higher EITC from conception until the age of 5 is found to lower incidence of metabolic risk factors (hypertension, obesity and diabetes) in adulthood (Hoynes et al. 2016). Male offspring of poor women who received cash benefits from the Mothers' Pension Program – the first government-sponsored welfare program in the U.S. (1911-1935) – were less likely to be underweight, received one third more years of schooling, had higher incomes in adulthood and lived one year more than comparable individuals whose mothers applied for but did not receive the benefit (Aizer et al. 2016). Most of this evidence is either specific to a low-income population eligible for benefits or the effect is estimated to be larger in such a population. An income windfall paid to the entire population of Alaska is estimated to have had a modest, positive average effect on birthweight and a larger effect among the low-educated population (Chung et al. 2016).

Inconsistent with the quasi-experimental evidence, a randomized experiment conducted in four U.S. cities found that payment of an unconditional income supplement of $333 per month (compared with $20 in the control group) to low-income mothers for three years from the birth of an infant had no impact on maternal assessments of children's health, sleep or healthcare

utilization, although children's reported consumption of fresh produce was higher in the treatment group (Sperber et al. 2023).

Notwithstanding the mainly null effects from this one experiment, the general picture emerging from the predominantly U.S. evidence is that money can matter for health when it reaches poorer families (women) expecting or caring for infants whose development still has the potential to benefit from the nutrition, shelter, healthcare and relief from psychosocial stress that income can afford. Combined with the substantial evidence of long-run health and economic returns to early-life health (Almond 2006; Almond and Currie 2011; Almond and Mazumder 2011; Heckman 2007), this suggests that income transfers targeted on poorer pregnant women and mothers can potentially set off mutually reinforcing positive impacts on the health and economic wellbeing of their offspring.

Income source. All sources of income may not be equal in their consequences for health. In theory, labor income has consequences for health behavior through the opportunity costs of sickness and time spent on health investment (Grossman 1972). In contrast, earnings from non-human capital have a pure income effect on health. Except for this difference, a model of rational health behavior would predict that health is invariant to the source of income. Logically, the amount of income, and not where it comes from, should determine how much is spent on health. But health behavior, like others, may not be entirely rational. Mental accounting (Thaler 1985) may cause some to spend money they receive as a transfer on risky health behavior but to be more cautious in spending money they earn. The health effect of income would then depend on whether it is received from the labor market, capital gains, a pension, welfare benefits or a windfall. There would be obvious policy implications.

Income receipt. For any given level of income, health may be sensitive to its receipt, as well as its source. Rather depressing evidence from the U.S. shows spikes in mortality on the first day of the month, when many receive salaries, pensions or transfers (Evans and Moore 2012).

Mortality remains elevated in the days immediately after the first of the month (ibid.) and it spikes at other times when income is received (Evans and Moore 2011).

Coincidence of elevated mortality with income receipt is consistent with a strong liquidity constraint on health-harming consumption, such as illicit drug use. Drug-related deaths and hospitalizations spike at times of income receipt (Dobkin and Puller 2007), although these deaths do not account for all of the discontinuities in mortality (Evans and Moore 2011). Health-improving consumption can also be liquidity constrained. Elderly Americans with Medicare that only partially covers spending on medicines fill more prescriptions on receipt of Social Security payments (Gross et al. 2022). Receipt of tax rebates in the U.S. substantially increases visits to hospital emergency departments (Gross and Tobacman 2014). In this case, however, the mechanism does not appear to be through weakening a constraint on access to affordable healthcare. Rather, it is through increased risky consumption: visits increase for alcohol- and drug-related problems.

This U.S. evidence shows that income receipt can impact health positively, through access to healthcare, and negatively, through increased consumption of health-harming goods and related activities that jeopardize health. The mortality spikes at the time of income receipt indicate that the negative effect dominates. Constrained liquidity is the most likely cause of these effects. Relevance to the income gradient in health lies in the fact that low-income households are more likely to be liquidity constrained (Japelli 1990; Rhine et al. 2006). With little or no scope to borrow against future income and limited options to hold savings securely, these households may have to forgo healthcare when it is needed and they may consume health-harming goods disproportionately when income is received. Both mechanisms may contribute to the health disadvantage observed at low incomes.

3.2 Health → Income

Logically, ill-health reduces income. While all health conditions do not limit productivity in all jobs, the essence of ill-health is interference with physical or cognitive functioning. Disability can bring comparative advantage in a particular job, but rarely gives absolute advantage in any. In an unregulated labor market, lower productivity caused by ill-health or disability would be met with a lower wage offer. Capacity to compensate by working longer hours is constrained by functional limitations, and so earnings are expected to fall with the onset of a health condition. Regulation can limit the scope for employers to offer lower pay to people with disabilities, but it may also reduce offers of employment (Acemoglu and Angrist 2001). Disability insurance can improve welfare by protecting incomes from the risk of disability (Autor et al. 2019; Deshpande et al. 2021), but the protection that can be offered is constrained by work disincentives that arise from the inherent difficulty of observing disability (Bound 1989; Diamond and Sheshinski 1995; Parsons 1980).

There is unambiguous evidence of income losses resulting from ill-health and disability (O'Donnell et al. 2015; O'Donnell 2024). Effects are large, lasting and ubiquitous (Blundell et al. 2021; Dobkin et al. 2018; Fadlon and Nielsen 2021; Garcia-Gomez et al. 2013; Lenhart 2019b; Meyer and Mok 2019; Tanaka 2021; Trevisan and Zantomio 2016; Simonetti et al. 2022). They are larger in the U.S., where insurance of earning capacity is less complete and the social safety net lower, than in northern Europe, where more generous social insurance offsets illness-induced earnings losses to a greater extent (Dobkin et al. 2018; Fadlon and Nielsen 2021; Garcia-Gomez et al. 2013; Meyer and Mok 2019; Mommaerts at al. 2020).

Evidence that health impacts income positively is stronger and more plentiful than evidence that income improves health (O'Donnell et al. 2015). Once this is recognized, the income gradient in health can be viewed as a health gradient in income. Both perspectives contribute to understanding the association. An effect of health on income does not preclude an effect of income on health. It may simply be easier to find evidence of the former, although clearly the

mechanisms through which health can impact income are more direct than those through which income can impact health. The available evidence shows that worse health makes people poorer and this explains substantially, although not comprehensively, why poorer people are observed to be in worse health.

4. Health inequality by wealth

4.1 Wealth → Health

Theory. The effect of wealth on health may differ from that of income. A lucky recipient of a positive shock to wealth may be willing to blow some of the windfall on unhealthy indulgencies, while hard-earned income may be spent more frugally and healthily. More rationally, since taking health risks directly threatens earnings potential and the opportunity cost of being sick increases with the wage rate, an increase in labor income (per hour) discourages unhealthy behavior (Grossman 1972; Galama and van Kippersluis 2015). For those with more health insurance than disability/sickness insurance, wealth is less immediately sensitive to a health shock than is labor income. An increase in wealth does not change the opportunity cost of being sick, removing that incentive for healthy behavior. However, through diminishing marginal utility of (non-medical) consumption, greater wealth lowers the opportunity cost of investing in health and encourages healthy behavior (Grossman 1972; Galama and van Kippersluis 2015). Those with less financial (and human) capital would, rationally, be more willing to run down their health capital, by engaging in physically demanding work, in order to maintain their consumption (Galama and van Kippersluis 2015). At a given level of financial wealth, however, exposure to less risk on that wealth is expected, through an effect akin to risk diversification, to encourage more risky health behavior (Hugonnier et al. 2013).

While wealth received in certain circumstances may lead to unhealthy consumption, the wealthier are expected to be healthier because they can better afford to spend money and time on keeping themselves healthy and they have stronger incentives to make these investments in order to prolong enjoyment of an already high level of consumption (Hall and Jones 2007). However, as with the income effect on health, at the individual (not country) level, the scope for greater wealth to bring better health depends on the extent to which the market allocates healthcare and other health determinants.

Evidence. Excellent research delivers inconclusive evidence regarding the effect of wealth on health in high-income countries. Conscious of the potential for strong reverse causality and confounding, Adams et al. (2003) test for non-causality rather than attempting to identify a causal effect. Using longitudinal data from a cohort of Americans aged 70 years and older, they regress each of a number of health outcomes on its lagged value, lagged wealth and a battery of controls. An insignificant coefficient on lagged wealth is consistent with no Granger (1969) causality from wealth to health. Aside from whether this is the appropriate concept of causality to gain understanding of the wealth gradient in health, the approach is most informative when non-causality is not rejected, which is the finding for most health outcomes. However, the null is rejected much more frequently when using a larger sample of 50+ – rather than 70+ – Americans observed over a longer period, which provides more variation in health insurance and is less susceptible to mortality selection (Stowasser et al. 2012). This leaves ambiguity since rejection of the null could arise from confounding without causality.

Lottery winnings provide a plausibly exogenous source of wealth variation from which to identify effects on health outcomes (Apouey and Clark 2015; Cesarini et al. 2016; Gardner and Oswald 2007; Lindahl 2005). The best study adopting this strategy uses comparisons between players and winners of lotteries entered by all bank savings account holders in Sweden (Cesarini et al. 2016). Amounts won can be large: seven times median annual income, on average. From

population data on winners linked to mortality and hospital admission registers over a 10-year follow-up, the authors estimate precise null effects on mortality and other health(care) outcomes.

An apparently obvious explanation of this finding would be that universal healthcare and a generous welfare state in Sweden leave little scope for wealth to impact health. While this may be true, the cross-sectional association between mortality and wealth is as strong in Sweden as it is in the U.S., and the estimates rule out an effect of only one sixth of this association. Another interpretation would be that the null effect results from a positive effect of wealth on health through spending on health-improving goods being offset by a negative effect of impulsive, unhealthy spending of the lottery windfall. However, winning has a small, persistent, negative effect on labor supply, suggesting that most winners do not give up work to live a decadent life. The distinction between health effects of wealth and income also does not appear to explain the null finding since the estimated effects are not different for one of the lotteries that pays out as (substantial) monthly income streams over a period of 10-15 years.

This study provides very strong evidence that the association between mortality (and other health outcomes) and wealth, which is as strong in Sweden as it is in the U.S., is not due to a causal effect of wealth. In contrast, another study leverages movements in the U.S. stock market to estimate substantial wealth effects on the health of older Americans: a 10% loss of wealth is estimated to reduce indicators of physical health, mental health and longevity by 2-3% of the respective standard deviation (Schwandt 2018). The inconsistency between these two excellent studies leaves the question of whether the difference in context or in the source of wealth variation explains it.

4.2 Health → Wealth

Theory. Health can affect the accumulation of wealth through lifetime earnings, saving behavior and the depletion of savings to pay for uninsured medical expenses. Effects on incentives to save are subtle. Better health reduces mortality risk and increases life expectancy. The latter effect increases the incentive to save in order to stretch consumption over a longer life and retirement, and the former effect further intensifies the incentive by reducing the risk of premature death that would leave savings unconsumed (Yaari 1965). While incurred (uninsured) medical expenses deplete savings and other assets (Adams et al. 2003; Smith 1999), cause debt (Kluender et al. 2021) and can induce bankruptcy (Dobkin et al. 2018; Gross and Notowidigdo 2011), the expectation of higher future medical expenses incentivizes saving (De Nardi et al. 2010) and greater variability in those expenses gives an additional precautionary motive to save (Palumbo 1999).

The effect of health on saving, and so wealth, is further complicated if preferences are heath-state-dependent (Evans and Viscusi 1991). If the marginal utility of non-medical consumption were to fall with deteriorating health, then an increased expectation of ill-health would weaken the incentive to save to afford future consumption that is likely to be less enjoyable. This would offset – partially or fully – an incentive to save more to pay for uninsured medical care. If ill-health were to push the marginal utility of consumption in the opposite direction, then both effects would incentivize greater saving in the face of future ill-health, and they would jointly push against the weakened incentives to save arising from shorter life expectancy and greater mortality risk. Unfortunately, evidence on the direction of the effect of health on the marginal utility of consumption is contradictory (De Nardi et al. 2010; Evans and Viscusi 1991; Finkelstein et al. 2013; Viscusi and Evans 1990).

Evidence. While theory is ambiguous regarding the effect of health on saving incentives, positive effects on wealth accumulation through higher lifetime earnings and avoided medical expenses would likely dominate even if the net effect on saving incentives were negative. The

healthier are likely to be wealthier, as is observed (Smith 1999; De Nardi et al. 2024). For example, in the U.S., median wealth of healthy male high school graduates at the age of 65 is 65% greater than that of their unhealthy counterparts (De Nardi et al. 2024). Viewing this as a health gradient in wealth, De Nardi et al. (2024) seek to explain it by first modelling health dynamics and then feeding them into a life cycle model of labor supply, health insurance and consumption. The first exercise reveals that ill-health is highly persistent: most of the lifetime variation in health is due to fixed differences – possibly due to genes or early life conditions – between health types as opposed to health shocks striking any type. Those with persistently poor health accumulate much less wealth because they incur higher medical expenses and, even more so, earn less over their lifetimes. However, these two mechanisms are not sufficient to explain the health gradient in wealth. The large remainder can be explained by extending the model to allow correlation between predisposition to bad health and strong time preference. That is, those who persistently experience bad health save less, not only because of their lower earnings, higher medical expenses and shorter expected longevity, but also because they value future consumption less. According to this inference, which is contingent on structure imposed to make the model fit the data, the strong positive association between health and wealth arises from processes that accumulate over lifetimes but are, to a large degree, preordained early in life. This conclusion is pessimistic regarding the scope for labor market and adult health (insurance) policies to impact the health-wealth gradient substantially. It is more optimistic regarding the potential return on early-life policies that would be effective in improving lifetime health and weakening time preference.

5. Health inequality by socioeconomic status

Theory. In public health, (socioeconomic) health inequality is interpretated as variation in health with status or social position (Mackenbach 2019). From that perspective, the income gradient in health, for example, is an association between health and a unidimensional proxy

for socioeconomic status (SES). The association is not expected to arise from an effect of income on health. Estimation of that parameter would be considered somewhat irrelevant to testing the hypothesis of the socioeconomic determination of health, which is the socially conditioned evolution of health over the life course through mutually reinforcing processes in multiple dimensions of SES rather than the immediate response of health to an abrupt, *ceteris paribus* change in one dimension, such as income. A sudden, isolated change in income may be inconsequential for health, and yet low income could still take its toll on health when combined with low education, unskilled and unrewarding work, or no work at all, absence of family responsibility and support, and a small, diminishing social network.

Drawing on sociology and biology, social epidemiology proposes theories of health inequality that postulate mechanisms – behavioral, material and psychosocial – through which low SES causes health to worsen over the life course (Bartley 2015; Mackenbach 2019). The common thread of these theories is that low SES increases exposure to disease risk factors and, given limited resources (cognitive as well as material), reduces resilience to them. Differences lie in the hypothesized fundamental causes of the sociobiological, economic and political environments in which causal mechanisms from low SES to poor health can emerge.

Empirics. Testing these theories is challenging. At the very least, it is difficult to use formal causal analysis based on the potential outcomes framework (Rubin 1974) to identify effects of a relative and composite construct, such as SES. One reason is that there is greater scope for a relative exposure to be endogenous. Position in a group hierarchy depends on group membership, which individuals may influence, at least to some extent. A second reason is that it is impossible to contemplate a counterfactual in which the SES of an individual – or a group – is changed and that of all others remains constant. If someone moves up the social ladder, someone else must move down. The stable unit treatment value assumption (SUTVA) causal inference of is violated. A third obstacle to identification – contemplation, even – of a causal

effect of SES is that when it is proxied by a composite index, such as one of multiple deprivation, there is a multitude of ways in which the index (rank) can change by a given magnitude. Each may have a different consequence for health depending on the combination of index components responsible for the change. The consistency assumption of causal analysis is violated. An effect cannot be identified essentially because the treatment is not uniquely defined.

Given the difficulties that must be overcome to identify a causal health effect of SES, most economists would gladly leave that challenge to other disciplines. In any case, the concept of status is foreign to the individualistic behavior that is the primary focus of economics. Why struggle to identify an effect that cannot even be postulated with conventional economic theory? A riposte would be that neglect of SES leaves the scope of analysis too narrow to gain insight into the causes of health inequality. Restricting attention to health effects of reasonably well-defined, absolute and separate socioeconomic exposures – education, income and wealth – may miss a bigger picture of health inequality evolving over the life course through cumulative and multiplicative effects of multidimensional deprivation.

Cumulative disadvantage. Case and Deaton (2017, 2020, 2022) argue that the declining health and rising (absolute and relative) mortality – particularly *deaths of despair* attributable to drug use, alcohol and suicide – of lower educated Americans result from their worsening circumstances in multiple life domains over an extended period. They do not turn to evidence on the causal effect of education to explain these changing education gradients in health and mortality. Education is merely the socioeconomic marker that is consistently available in mortality data. The authors claim that no single factor, such as stagnant incomes, caused the decline in health. Rather, they attribute it to deterioration in broader life circumstances, prospects and status, with losses in one dimension exacerbating the negative health impact of

those in another. For example, shrinking labor market opportunities damage marital prospects and bonds, which weaken incentives to invest in health to support and enjoy a family.

This argument that the worsening health of low-skilled Americans in the 21st century has its roots in their declining economic and social status is challenged, mainly on the grounds that the data are more consistent with the supply of opioids driving the death of despair epidemic (Currie and Schwandt 2021; Ruhm 2019, 2022) and racial differences in the mortality trends are inconsistent with changing economic prospects being the fundamental cause (Ruhm 2022). Without denying the important role of the supply of opioids, Case and Deaton's (2020, 2022) narrative is of a vicious interaction between that supply and demand arising from weakened opportunities and incentives to invest in health driven by structural changes in the broader life circumstances of the low skilled.

Relevant here is the proposition that health inequality emerges and evolves through complex processes involving interactions between social and economic conditions and prospects, the supply of healthcare and health-damaging goods, and health. If that were true, then there is only limited scope to gain insight into the root causes of the inequality by using causal analysis to estimate the partial, reduced form effect of any one socioeconomic exposure in isolation.

6. Health inequality causes and policy

Research seeking to uncover causes of health inequality typically claims that knowledge of causes is prerequisite to the design of policy to reduce inequality. This motivation may also be used to justify focus on estimation of the effect of a separate, manipulable socioeconomic exposure – education, income or wealth – rather than analysis of health consequences of the ambiguous and multidimensional SES that emerges from complex processes that are difficult or impossible to manipulate (Harper and Strumpf 2012). What is to be gained from learning that low SES is a fundamental cause of ill-health if available policies are blunt instruments for

changing social status? A counter-argument is that (quasi-) experimental evidence of a health effect of any one of education, income or wealth is local to the source of variation in that exposure. It does not necessarily transfer to the effect of a policy-induced change in the same exposure. Ideally, evidence on the effectiveness of alternative policy options would guide policy selection. Evidence of the health effect of a socioeconomic exposure leaves doubt about the effectiveness of a policy in impacting that exposure and the relevance of the evidence from another context to the health effect of the socioeconomic change that the policy can induce.

The notion that knowledge of the causes of health inequality is required to reduce it is intuitive but incorrect. For example, if causal analysis were to provide externally valid evidence that low income causes ill-health, then reducing the income gap between the rich and poor would narrow the health gap between them (unless the socially privileged were to find a way to maintain their health advantage through some channel other than income (Link and Phelan 1995)). But even if there were no causal effect of income on health, the rich-poor gap in health could be reduced by prioritizing the health of the poor through policies such as (fully) subsidized health insurance or healthcare for low-income individuals, as well as primary care and public health interventions targeted on this population. Effectiveness would rest on the health impact of the policies. Supporting evidence from the U.S. shows that health insurance for low-income households (Medicaid and the Children's Health Insurance Program) improves health (East et al. 2023; Finkelstein et al. 2012; Goodman-Bacon 2021; Miller and Wherry 2019; Thompson 2017; Wherry et al. 2018) and reduces mortality (Currie and Gruber 1996ab; Goldin et al., 2020; Goodman-Bacon 2018; Miller et al., 2021; Sommers et al. 2012; Wherry and Meyer 2016), with effects manifesting in the short run (Currie and Gruber 1996ab; Finkelstein et al. 2012; Goldin et al., 2020; Goodman-Bacon 2018; Miller et al., 2021; Sommers et al. 2012; Wherry and Meyer 2016) and in the long run from childhood to adulthood and even across generations (East et al. 2023; Goodman-Bacon 2021; Miller and Wherry 2019; Thompson 2017). There are

also substantial positive long-run effects on socioeconomic outcomes (Brown et al. 2015; Cohodes et al. 2016; Goodman-Bacon 2021; Miller and Wherry 2019), suggesting that targeted health policy can reduce economic inequality, as well as health inequality, and the intergenerational transmission of poverty. These results are not contingent on what caused low-income individuals to be in worse health.

Evidence on the socioeconomic determination of health is needed to assess the potential to reduce health inequality through education, income-support, tax and social policies. If there were causation from income to health, for example, then income redistribution through tax-transfer policy would get to one root of the inequality. This also appeals to intuition. If it is low income that causes ill-health, then subsidizing healthcare for the poor merely repairs damage done elsewhere. Intervening directly on income would prevent the damage. While there is much to be said for this argument, its weakness, besides the lack of consistent evidence that income does causally impact adult health, lies in the fact that any health impact of income redistribution would be a spillover from tax-transfer policy. Ideally, the policy would be designed to take account of the spillover. That would complicate an already difficult optimal tax problem. Even if its solution and implementation were feasible, the health spillover would likely be a marginal consideration in the policy design. In a realistic scenario with information and political constraints on tax-transfer policy, the indirect health effect would get even less attention. In contrast, the health effect is the primary motivation for a targeted health policy. A health policy is presumably more cost effective than an income or education policy in generating health. A health policy is likely to weaken work disincentives less than a cash benefit policy. And through the health effect, the former may even impact labor supply positively.

While socioeconomic causes of health inequality need not be known to identify policies that can reduce it, political support for those policies may be contingent on the causes. Health inequality caused by income inequality, for example, may be considered more unjust than

variation in health caused by health behavior that is partly driven by time preference that also influences income and is responsible for its association with health. If it were known (or believed) that health is not socioeconomically determined, then would there be (as strong) willingness to prioritize the health of the less privileged? The answer depends on what motivates the prioritization. If it is a belief that, for example, income-caused differences in health are unjust, then evidence on the causal effect of income on health would impact support for policies that prioritize the health of poorer individuals. But that support could also arise from concern about inequality in general wellbeing that is a function of both income and health. For given marginal distributions of income and health, positive correlation between them can increase inequality in wellbeing. The health of poor may then be prioritized in order to compensate for their income poverty, particularly if there are constraints on income redistribution policy.

An experiment conducted with a broadly representative sample of the United Kingdom population finds evidence consistent with this motivation – prioritization of the health of lower-income individuals (and aversion to income-related health inequality) does not weaken, on average, when participants are informed of the causal effect of income on health (Robson et al. 2024a). But support for prioritization is not unconditional. Those who believe that people are more responsible for their incomes are less willing to prioritize the health of the poor (Robson et al.2024b), which suggests that support for using health policy as an instrument of redistribution is, to an extent, contingent on beliefs about responsibility for the socioeconomic correlates of health.

7. Summary

Attempts to understand the causes of health inequality in high-income countries by estimating effects of education, income and wealth have not produced strong evidence of the socioeconomic determination of health, at least within adulthood. For education, the estimates

are variable, even with respect to whether or not the effect is null, not to mention magnitude. For income and wealth, mostly null effects are estimated, with important exceptions. Heterogeneity in evidence on the socioeconomic determination of health is expected. Even when attention is restricted to *the effect* of any one of education, income or wealth, the estimand is still not precisely defined given scope for qualitative differences in each of those exposures and in health. Further, the respective effect is plausibly context-specific and its estimate may be local to the source of variation in the exposure.

Constructs matter. Health is multidimensional, and so there is no single health effect of any one of the purported socioeconomic determinants. Attempts to circumvent the multidimensionality by using subjective assessments of overall health leave scope for bias through socioeconomic differences in the perception and reporting of health (Bago d'Uva et al. 2008).

Measurement problems extend to the socioeconomic determinants. Education is a somewhat loose concept that lacks a unique construct. Data are usually restricted to measurement of quantity, which captures neither variation in content nor quality both of which may have health consequences. Despite these measurement limitations, there is abundant evidence of causal effects of education on economic outcomes (Buscha and Dickson 2023; Psacharopoulos and Patrinos 2018) but not on health outcomes. The same variation in education is often found to impact wages (Oreopoulos 2006) but not health (Clark and Royer 2013). This implies that any health effect is smaller than the wage effect, but it leaves open the question of whether a health effect is entirely obscured by measurement error or, in fact, it is null.

Income and wealth are more precisely defined than education, and yet health consequences may differ across sources of income and forms of wealth. Variation in the definition and measurement of health and its hypothesized socioeconomic determinants make it impossible to give a definitive answer to an imprecise question: What causes health inequality?

Context matters. Even after restricting attention to a particular measure of education, income or wealth, its effect on a specific health outcome may differ across place and through time with physical, social, economic and health system environments. For example, what wealth can do for health depends on how healthcare is financed and distributed. Excellent studies based on credible identification strategies find a large effect of wealth on health in the U.S. (Schwandt 2018), where there is greater reliance on private health insurance and out-of-pocket spending to finance healthcare, and no health effect of large gains in wealth in Sweden (Cesarini et al. 2016), where there is more universal and comprehensive insurance coverage as well as a higher social safety net.

Methods matter. (Quasi-) Experimental methods offer the potential to move beyond description of health inequality to identification of its causes. However, even if study-, policy- or institutional-induced variation in a purported socioeconomic determinant is as good as random, its health effect in sample data may not align with the respective effect in the population of interest over the same or a different time period. For example, the same variation in education (years of schooling) generated by a compulsory schooling law in England and Wales has produced different estimates of health/mortality effects depending on the specific samples, follow-up periods and empirical strategies used (Clark and Royer 2013; Davies et al. 2018; Janke et al. 2020).

What we know. While constructs, context and methods matter, evidence on the causes of health inequality is not entirely ambiguous. There is consistent (mainly U.S.) evidence of positive effects of cash benefits on infant and child health outcomes. These benefits are mainly targeted at low-income households, although there is some evidence of universal benefits having larger effects on the health of poorer children. Given the importance of early-life health for health and economic outcomes in adulthood (Almond and Currie 2011), this evidence supports the hypothesis that the origin of socioeconomic gradient in health lies in infancy (Case et al. 2002;

Currie 2009). It also suggests that targeted cash benefits, as well as targeted health policies, can help flatten the gradient. Consistent with this narrative, the association between child mortality and poverty in the U.S. weakened over a period in which there was an increase in welfare and an expansion of public health insurance coverage for low-income households with children (Currie and Schwandt 2016). Less consistent is the obstinate persistence of health inequality among European adults who grew up with protection from high social safety nets (Mackenbach 2019). For example, the social class (occupation) gradient in mortality appears to have emerged in Sweden only after the institution of that country's extensive welfare state (Bengtsson et al. 2020). This suggests a strong role of differential health behavior in generating health inequality. The origin of the gradient may lie in childhood, and social and health policies may sap the seed of inequality sown in that phase of life, but these policies are not all that matter. The causes of health inequality are a lot more complex and may evolve with the structures of society, the economy and the health system (Link and Phelan 1995).

Part of the complexity is causation running from health to socioeconomic outcomes. There is consistent evidence of large effects of changes in adult health on income and wealth. Further, child ill-health both persists into adulthood and constrains adult socioeconomic outcomes. The evidence suggests that this reverse causality could account for a substantial part of the cross-sectional association between adult health and each of income and wealth, and, to a lesser extent, education. Quantifying this contribution is not straightforward. The suggestion that it is substantial may well be challenged, although the grounds for doing so would be stronger for health inequality by socioeconomic status (proxied by occupation) than by income or wealth. Unfortunately, debate about the causes of health inequality can descend to vociferous defense of simplistic, singular explanations. Logic and evidence point to a more complicated story with multiple plots unfolding simultaneously. There are reasons to expect (context-dependent) socioeconomic determination of health and health effects on socioeconomic outcomes. Neither

logic precludes causality in the other direction. There is also good reason to expect both health and socioeconomic outcomes to be correlated with observable and unobservable third factors. Evidence can be found to support causality in each direction and for the influence of confounders.

What we can know. Formal causal analysis based on (quasi-) experimental methods has generated more consistent evidence of health effects on socioeconomic outcomes than of the socioeconomic determination of adult health. This is a statement about evidence, not a claim that, in general, education, income and wealth do not determine health. Causal analysis may simply fail to consistently detect the effects. One reason would be the aforementioned effect heterogeneity over dimensions of health as well as the nature and context of the socioeconomic exposure. Another would be that lags between socioeconomic exposures and emergence of health effects exceed the observation period of most studies. A third would be that the variation in a socioeconomic exposure that remains after purging or controlling variation generated by confounders could be inconsequential for health, although null effects precisely estimated from plausibly exogenous and substantial variation in income (Miller et al. 2024) and wealth (Cesarini et al. 2016) go against this explanation.

A fourth reason why consistent evidence of causal effects of education, income and wealth on health may not emerge even if health were, in fact, socioeconomically determined, would be that these are the wrong exposures. Health may be determined not by the absolute level of each of education, income and wealth operating in isolation but by their multiplicative combination into relative socioeconomic status. Social status may be critical to the opportunities and motivations someone has to invest in their health and to seek and receive treatment for illness and injury. But the relative and multidimensional nature of social status make it ill-suited to the deployment of formal causal analysis to estimate its effect on health. With such analysis, attention is necessarily confined to the effect of any one dimension of socioeconomic status in

isolation, which may miss the health consequence of an accumulation of negative circumstances across multiple domains of life. It would be challenging, at the very least, to test this hypothesis with formal causal analysis, although its plausibility could be assessed with other research methods.

What we need to know. Research into the causes of health inequality is usually justified by appeal to its policy relevance. Knowledge of causes can both motivate and guide policy intervention. Claims about the latter of these two relevances sometimes go too far. While we need to know whether income, for example, impacts health to evaluate the potential for tax-transfer policy to change the distribution of health, that knowledge is not needed to flatten the income gradient in health. That could be achieved by a health policy, such as subsidized health insurance for low-income households, that prioritizes the health of the poor irrespective of whether poverty causes ill-health. Political support for such prioritization, which has proved effective in improving long-run health and economic outcomes of lower-income Americans while still leaving substantial health inequalities in the U.S., may be contingent on knowledge (or beliefs) about the causes of health inequality. Knowing that poverty causes ill-health may bolster support for pro-poor health policies. But knowledge of causes is not a prequisite to identification of interventions that could reduce inequality. Knowledge if disease etiology is necessary for effective prevention but not for effective treatment. Likewise, while uncovering causes of health inequality may help identify societal change that would prevent that inequality from emerging and persisting, ignorance of the causes does not preclude direct intervention on the health of the socioeconomically disadvantaged to improve their health and lessen their health deficit. To reduce health inequality, we need to know what these interventions are and how to target them appropriatetely. The association between health and income can also be reduced through better insurance of sickness and disability.

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