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Permalink

<https://escholarship.org/uc/item/7w52w02q>

Journal

Journal of Economic Literature, 56(3)

ISSN

0022-0515

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Publication Date

2018-09-01

DOI

10.1257/jel.20171383

Peer reviewed

Mind the Gap: a review of “The Health Gap. The challenge of an unequal world” by Sir Michael Marmot

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Abstract: “The Health gap” documents the large and persistent health gaps that exist across and within relatively rich countries today, and discusses their causes. Marmot’s thesis is that direct factors like health care, or reverse causality (poor health causing low incomes) explain only for a small portion of these gaps. To eliminate them, policy should focus on the real culprits: poverty, education, and occupational mobility among others. While Marmot’s ethical arguments are quite compelling, Marmot’s recommendations are too strong given the current evidence. Policies need to be based on a clearer understanding of why things work, when, and for whom.

Introduction

Marmot’s “Health gap” is a passionate and riveting account of the state of health around the world and of the large and persistent inequities that exist across and within relatively rich countries. Consider the US. According to 2016 estimates in the CIA world book, US life expectancy is 79.80, almost 4 years lower than that of Japan. In fact, it is lower than in 41 other countries.¹ Although the rankings vary from year to year and depend on the measures used, for many years now the US population has been in significantly worse health than populations elsewhere, even substantially poorer ones (National Research Council and Institute of Medicine, 2013).

Within the US there are also substantial gaps in health and mortality by socio-economic status. In 2000 a person with some college education could expect to live about 7 years longer than someone with a high school degree or less (Meara et al. 2008, Hummer and Hernandez 2013). But in 1980 this gap was less than 3 years. This drastic increase does not appear to be driven by changes in composition or behavior within education groups (Cutler et al. 2011). Similarly, Chetty et al. (2016) document that in 2014 the gap in life expectancy between the richest 1% and poorest 1% of individuals was 14.6 and 10.0 years for men and women, respectively. The gap is larger than a decade earlier

** I am grateful to Todd Muney, Titus Galama and particularly to Steven Durlauf for excellent comments on earlier versions of this essay. All errors are my own.

¹ <https://www.cia.gov/library/publications/the-world-factbook/rankorder/2102rank.html>

and is growing across cohorts (National Academies of Sciences, Engineering, and Medicine, 2015). These gaps are staggering. Life expectancy in the US grew by about 30 years (from around age 49 to age 77) in the entire 20th century (CDC 2012), and only by 14 years in the century before (Gartner et al 2006). A gap of ten or fifteen years is thus comparable to 50 to 100 years of development. A significant fraction of Americans are living lives so short, it might as well be the 1950s.

The “health gap” is large and growing, not just in the US but in many countries. Yet the gaps are smaller in some populations, and they have changed drastically over time. This observation leads Marmot to investigate what conditions lead to poor and unequal health within populations; and to ascertain the policies that have led to health improvements *and* to reductions in the health gap.

Marmot reviews the large body of work that has investigated the sources of health disparities, also known as the health gradient. Marmot makes some very important observations. Only a small fraction of the gaps relates to health care access, because most healthcare is remedial, and thus cannot explain why individuals get sick in the first place. Access to good doctors and good hospitals will lengthen your life if you have a heart attack, but simply not having a heart attack (or delaying its onset) will have a far greater impact on longevity. Given that these “proximate factors” cannot explain the majority of the health gaps, Marmot urges us to look at the “deep causes” of disease, which he identifies as socio-economic status.

Marmot then focuses attention on some obvious culprits like poverty, education, and occupation; and also brings attention to other less-frequently considered factors, such as ability to control one’s life, stress, and social capital. These factors have all been shown to have large associations with health and mortality. Marmot goes on to provide specific policy recommendations, urging that we redistribute income (through tax policy); increase education and expand early childhood programs; improve work conditions; improve the social capital of communities; and improve preventive care.

I will comment on a few selected aspects of the book, and on the evidence that I know best through my own work. I first discuss the arguments that I found most compelling and interesting—these concern Marmot’s discussion of the ethical and practical considerations surrounding health policy. I then comment on each of the specific policies that Marmot advocates. Here I attempt to give a more nuanced reading of the evidence regarding the possible effects of each policy—not all the proposed policies have equally good evidence to support

them, and some should be more carefully considered. I end up with a discussion of Marmot's views on health and in particular his reading of the empirical evidence—he interprets a large body of evidence correlating health and socio-economic status as mostly (or sometimes uniquely) consistent with a view that socio-economic status causes health. I believe that causality goes both ways – that while SES does indeed impact health, health also has a significant impact on SES, and that other factors affect both. I conclude with some reflections on directions for future work.

Why do we tolerate health gaps and gaps in the determinants of health?

Marmot presents an insightful discussion of why countries and peoples tolerate gaps in health and other outcomes. In great part, he attributes our willingness to tolerate disparate outcomes to a deep-rooted view that health is mostly determined by individual choices. For example, obesity is generally viewed to be the result of poor individual behavior. As a result, the willingness of society to pay for obesity-related health expenses is low.

Yet a large and substantial amount of research supports Marmot's claim that "Health is not simply a matter of personal responsibility." The environment that individuals live in (measured, e.g., by characteristics of one's place of birth or place of residence) is very predictive of their health, their eventual longevity and cause of death. Durkheim demonstrated 120 years ago that even something as seemingly personal as suicide can be predicted by geographic conditions. This notion has been thoroughly documented in the US by the Dartmouth Atlas, which maps disease and health care utilization for small geographic areas in the US (<http://www.dartmouthatlas.org/>). Many local conditions (e.g., weather, air-quality, water, food, the level of other public goods) predict health and mortality. And many of the greatest improvements in health have been achieved at the local level though public regulation and investments in infrastructure.

Many infectious diseases, the greatest killers at the beginning of the 20th century, were eradicated by the provision of clean water and sewer systems (Cutler and Miller 2005). Decreases in air pollution due to regulation, such as the Clean Air Acts in the US and the UK resulted in significant reductions in mortality (Chay and Greenstone 2003). Malaria was eradicated in many countries through fumigation (Bleakley 2010). Broadly stated, the environment has been made healthier. When such policies were implemented, they benefitted all, including those of low socio-economic status. For instance, clean water and sewer provision greatly decreased black-white gaps in mortality in the

US South (Troesken 2004). Public health interventions also have some interesting features—they tend to benefit the poor the most without distorting their behaviors (e.g., they provide no disincentives to work).

Yet today's approach to health improvement focuses attention on individuals, their agency, and their responsibility. Around the world policymakers pursue deworming pills, iron-supplementation, latrines and other such individual-use technologies, often through the price mechanism (Dupas 2014). Efforts to fight malaria in Africa concentrate on bed nets—yet in the US and other countries malaria was eradicated by systematic DDT fumigation. Similarly, policymakers pursue the use of individual filtration to improve water quality, though in the West, *cities* implemented water chlorination and filtration systems, and developed sewer systems for the populations as a whole. In the US today health policy is focused on changing individual habits such as eating, drinking, and exercising.

This is not to say that we should abandon efforts to educate individuals or to provide access to technologies, like bed nets, which can improve individual well-being, particularly in settings where governmental capacity for large public works is limited. The point is that global “public health” approaches have had the largest impact, at the lowest cost. Despite great past successes, we no longer follow that approach. Although I know of no rigorous evaluation of this claim, Marmot's book convinced me that this distinction is worth paying more attention to, and that our current policy focus might be misguided.

A related point is that the focus on individual behavior misses important phenomena. There are now several papers that suggest that there are important “peer effects” in eating, drinking, smoking, exercise and other health behaviors (Christakis and Fowler, 2007; Christakis and Fowler, 2008). These behaviors are fundamentally social, and while individual rationality and choice play a role, it is foolish to ignore the role of sorting and norms, and other inherently social forces. Individuals also face laws, health care and food prices, and other factors (e.g. weather) at the local level—a comprehensive view of health must consider environmental and other aggregate factors that affect all individuals living in a particular area. So, I agree with Marmot that “People's ability to take personal responsibility is shaped by their circumstances. People cannot take responsibility if they cannot control what happens to them.”

Lastly, I appreciate Marmot's discussion of what it is that we should care about when thinking about social outcomes, viz., the equality of outcomes, not just of opportunities. This is of course a function of our perception of the role of luck. If luck determined all outcomes, and

individuals had no way to affect these outcomes, then it would be easy to justify redressing unequal outcomes. If effort and virtue determined outcomes, then it would be harder. In the US, evidence suggests that inequality is tolerated or even championed because of a misconception that there is high social mobility: that those who make it are deserving, while those who don't are not. Data from the World Values Survey illustrate this: "60 percent of Americans versus 29 percent of Europeans believe that the poor could become rich if they just tried hard enough; and a larger proportion of Europeans than Americans believe that luck and connections, rather than hard work, determine economic success" (Alesina and Angeletto 2005). Not surprisingly, support for social spending across countries is strongly correlated with the belief that one's income is largely determined by luck (Alesina, Glaeser, and Sacerdote 2001). Ironically, the US has both higher inequality and lower social mobility than other western countries, as shown by the so-called "Great Gatsby" curve (Corak 2013).

Inclusive approach

Marmot argues for universality, and his argument is persuasive. Marmot insists that health differences exist not just between the "haves" and the "have-nots"; but rather that there is a continuum, along which gradual increases in status (e.g., education or income) are associated with gradual increases in health. The data clearly shows this is true for education. When presented with evidence that education and health correlate, many think this must be entirely driven by education at the bottom: learning to write and read must matter but beyond that, why would education help? But indeed the data show (roughly) linear increases in health with each additional year of school, without diminishing marginal returns (Cutler and Lleras-Muney 2008, Montez 2012). The health returns to income are also always positive, albeit diminishing (Deaton 2010). So Marmot argues we should help everyone.

Current economic thinking, and common sense, would suggest that we want to tailor programs to aid those who need them the most - thus our typical "target the needy" approach. This gets us the biggest "bang for the buck." Universal programs provide goods to many who are not in need of support (or less in need). Conventional wisdom has it that by providing programs to everyone, we crowd-out the private market and simply substitute private provision with public provision. In other words, we waste money on people that need no help, lowering program success rates and increasing the cost of the program to tax payers. For example, health insurance expansions in the US often make individuals that already have health insurance eligible for public insurance. An estimated 20 to 50% switch to public insurance,

increasing the cost of the public program but not insurance rates for the population as a whole (Cutler and Gruber 1996, Gruber and Simon 2008).

In many contexts, however, the evidence suggest that in our urge to exclude the unworthy, we actually exclude most of the individuals we want to reach. Again, health insurance expansions in the US provide a case in point. Medicaid expansions (and many other public programs in the US) have infamously low take-up rates among the targeted recipients.² For example prior to the ACA only 10% of those made eligible for health insurance through Children Health Insurance Program enrolled (Gruber 2013). Moreover Gruber and Simon (2008) report that efforts to limit crowd-out led to even lower take-up rates. In other words, Medicaid expansions enrolled too many individuals it did not target (errors of inclusion) and almost no individuals that it targeted (errors of exclusion). This suggests that the cost of targeting is high.

A related argument pertains to recent efforts to give individuals in developing countries access to innovations that might improve their health, such as bed nets, nutritional supplements, or water chlorination filters. The claim is that free distribution results in waste: if households do not value free products, they will not use them and they will therefore not benefit from them. Prices serve as a screening device to identify those in need. But recent randomized trials suggest otherwise. Cohen and Dupas (2010) show that in Kenya cost-sharing did not reduce waste, but it did reduce the number of people who got the nets. Another recent randomized trial in Kenya to promote the use of water purifying technologies finds that “although errors of inclusion are low under cost sharing, cost sharing generates many errors of exclusion relative to free treatment.” (Dupas et al. 2016).

Partly for targeting reasons, the US pursues a policy of in-kind help for the poor, rather than cash-transfers (Currie and Gahvari 2008). The logic is that if money is offered to the poor, everyone will claim to be poor; but only those who are hungry will line up for free soup. But the disabled, those working multiple jobs, and many other hungry individuals might actually miss the soup line. In addition to being potentially ineffective in terms of reaching the intended population, provision of in-kind transfers is more expensive than simple cash transfers. The use of targeting mechanisms is itself costly for the government (administratively) and for individuals by design, in that it imposes hassle costs. It also generates deadweight losses because it

² Although this is less true for recent Medicaid eligibility increases associated with the ACA.

imposes a particular consumption bundle. For example Jacoby (1997) estimates that a Jamaican program to provide free lunch to children in school cost 400 to provide but was only valued by recipients at 158. All together, it is unclear that the benefits of targeting outstrip its costs.

While I won't suggest that all support programs need to be universal and that targeting is always wasteful, US programs are too concerned with exclusion, and not concerned enough with inclusion. The US welfare system as a whole transfers much less to its poor than do welfare systems in other rich countries. Although pre-tax poverty rates in the US are not out of line with poverty rates in other countries, other countries have much higher transfers and much lower post-transfer poverty. In fact, child poverty rates in the US today exceed 20% - nearly the highest in the developed world - and they are only diminished by a few percentage points through transfers. A recent study that carefully assesses the evolution of post-transfer family incomes in the US concludes "government redistribution has offset only a small fraction of the increase in pre-tax inequality. Even after taxes and transfers, there has been close to zero growth for working-age adults in the bottom 50% of the distribution since 1980" (Piketty, Saez and Zucman 2016).

Our current targeting approach uses strict cut-off rules: people are either poor and eligible for help, or they are not. But as Marmot notes, this distinction is arbitrary and is not supported by the data. Individuals right above and right below the federal poverty line don't look that different. This observation has led to a large amount of empirical research that exploits these discontinuities in treatment to establish causal treatment effects using Regression Discontinuity (RD) designs. (For a review giving many examples of anti-poverty programs evaluated this way, see Van der Klaauw, 2008.) This discussion suggests that programs should be designed using "sliding scales" like the Earned Income Tax, where the benefits are largest at the bottom and decrease progressively, rather than end abruptly. This design, in addition to serving a larger deserving population, also possibly generates fewer disincentives.

Some have argued in favor of universalism because universal programs are more likely to be politically viable (Skocpol 1991). I don't think this is clear, because even programs like Social Security, which almost all eventually have access to, are under constant political pressure. Marmot's view is different: policies need to be more inclusive because there are potential health benefits not just to the poorest and least educated but to almost all individuals.

But then one should ask why universalism came to be accepted in some countries but not others. In the US, racial and ethnic considerations are important in thinking about redistribution. Alesina et al. (1999) show that “voters choose lower public goods when a significant fraction of tax revenues collected on one ethnic group are used to provide public goods shared with other ethnic groups.” Residential segregation by income has increased (Reardon and Bischoff 2011). This has potentially important implications for the provision of local public goods and services (such as education and hospitals, or clean water and parks) that might improve health (Durlauf 1996). It also means that individuals are less aware of the plight of those who are not in the immediate spheres, and possibly creates lower political consensus on what policies are to be pursued.

This line of reasoning suggests that achieving Marmot’s objectives requires political considerations. The population must first hold the belief that the policies are desirable, not just because they are perceived as being effective, but because their perceived outcomes are seen to be fair. This requires persuasion.

“We know what to do to make a difference”

Noting the large and unacceptable differences in health and longevity, Marmot prompts us to action. He claims that we know what to do to ameliorate these inequities. In this section, I comment on his specific proposals, and suggest that, in fact, we do not know. Or better put, the evidence on the causal effect these policy proposals will have on health is not as clear as Marmot leads us to believe. I focus my comments here on a few policies that I am familiar with (education, redistribution, early childhood and neighborhoods) and ignore some important ones (such as preventive healthcare).

Education for all?

I have spent a significant portion of my academic career investigating the correlation between education and health. My own early research would support Marmot’s policy recommendation to invest more in education. However, recent research has made me question my original findings, and led me to a more nuanced view of the role of education.

In Lleras-Muney (2005) I asked if differences in mortality by education levels were causal by investigating whether those who were forced to go to school because of compulsory schooling laws lived longer lives as a result. The design exploited changes in compulsory schooling that took place in the US between 1915 and 1940. I found that the effect of

education on mortality, which this natural experiment measures, was large, and larger than the implied effect of education measured by OLS, with one more year of schooling resulting in more than one year of extra life. I went on to further investigate mechanisms to better understand why education led to better health.

However, since the 2005 article appeared, many other papers have used similar designs in other countries with much more mixed results. In my view, the most convincing of these efforts is the study by Clark and Royer (2013) of the effects of changes in compulsory schooling in the UK (Marmot's home), which increased the school leaving age from 14 to 15 in 1947, and then to 16 in 1972. The authors found that the reform successfully increased the average education of the population by 0.46 and 0.3 years—a much larger increase than in the US, in which years of education increased by only 0.05 years (one more year of school for 1 in 20 kids, compared to one more year for every other child in the 1947 UK case). Yet there was no significant difference in the mortality of the affected UK cohorts thereafter.

The stark difference in the findings suggests that strong caution is needed when promoting pro-education policies as a means of improving health. Even if we could rationalize the difference in the findings (for instance, as stemming from the fact that in the US only a few were affected and they came disproportionately from the lower end of the distribution), the results from the British study provide a compelling example of a massive education reform that had no measurable impact on health.

Of course, we could also argue that the methodology of one or both of the studies is flawed. But a fair number of similar studies using compulsory schooling as a “natural experiment” have found a significant amount of heterogeneity in the estimated impact of education on mortality.³ Even when the effects are positive, there is heterogeneity across groups—a common finding, for instance, is that effects are larger for men than they are for women.

³ Albouy and Lequien (2009) find no effects in France, Meghir et al. (2012) find no effects in Sweden, van Kippersluis et al. (2009) find effects in the Netherlands but they are smaller in magnitude than in the US. A recent study pooling several European countries by Gathmann et al (2015) documents this heterogeneity as well. There are some important differences across studies. For instance the timing of when effects are measured differs possibly explaining small effects in some studies--this matters because at younger ages mortality rates are low.

Why would there be heterogeneity in the effect of education? I have spent a lot of time reflecting on this question. The returns to schooling were at an all time low in the 1970s (at least in the US, see Goldin and Katz 2008). So perhaps this explains why more education did not translate into more health for the post-WWII cohorts in the UK. In the US at the turn of the century, high school education led to greater access to white collar jobs, which are safer on average than blue collar jobs or jobs in agriculture. But the difference in the healthiness of occupations has probably declined over time, as work-safety regulations have been implemented. Another possibility is that in Europe the existence of universal health insurance and broad safety nets diminishes the importance of education as a determinant of health. There are many other possible reasons, like differences in the quality of schooling, more extensively discussed elsewhere. (See, e.g., Cutler and Lleras-Muney, 2014.)

Another set of studies looking at the question of whether more education leads to better health uses twins: identical twins share identical genetic endowments so within-twin comparisons hold these initial conditions fixed. There is much written about how to interpret the results from these studies, and about their external validity. But interestingly the findings also vary—some of the studies find no effect of education on health (Behrman et al. 2011, Amin et al. 2013) and some that more education leads to better health and lower mortality (Lundborg 2013; Lundborg et al., 2012).

The devil is in the details. That is, even if we accept that education can matter, it does not matter all the time, and not all policies that raise educational attainment will yield the expected health benefits, or should be implemented. Education is costly for individuals and their families, and to tax-payers, so blanket increases in education might not be cost-effective.

Income transfers?

Richer countries have higher life expectancies and richer individuals live longer lives. Like education, GDP and income at a point in time are both strong correlates of mortality, across populations and individuals. Income increases tend to have a stronger “protective” effect for those who are initially poor. Significantly, wealth and income gaps in the US today are the largest they’ve been in 100 years (Piketty 2014): if wealth and income cause longevity, then rich and poor will have substantially different longevity. Marmot proposes more redistribution of income to reduce the health gap between rich and poor.

The relationship between income and health has been the subject of a great deal of controversy. And not all the evidence supports Marmot's conclusions that there is strong evidence of a large and causal effect of income. In Cutler, Deaton and Lleras-Muney (2006) we give an overview of the many examples of this contradictory evidence. At the aggregate level, booms are associated with higher, not lower mortality. In Cutler et al. (2016) we provide some evidence that pollution and alcohol consumption increase substantially in good times, possibly explaining the puzzling findings. At the individual level, a number of studies find that, in the short term, income transfers can be detrimental to health and increase mortality. That's the finding of Snyder and Evans (2006), who study the effects of changes in pension-income resulting from administrative mistakes. They find that mortality was greater for those who received greater benefits. Smith (2005) and others (Adams et al 2003), using panel data, have found only weak correlations between increases in incomes and subsequent health improvements among adults in the short run.

In the short term, income does not substantially affect health, and might even harm it for some. It is considerably harder to establish how persistently high levels of income affect health over the long run. Work by Lindhal (2003) exploiting lottery winnings does suggest there is a large positive improvement in adult health that comes from larger incomes, but other work exploiting bequests finds very small effects (Meer et al 2003). More research on the causal effect of *permanent* income is needed.

One might expect that income would have its strongest effect on children. My own recent work addresses this question. We looked at the effects of childhood transfers, and the results support Marmot's proposal to help poor families. In Aizer et al. (2016) we collected individual-level records of thousands of women who applied for the "Mother's Pension" program, which provided cash to poor women with children whose husbands had died, were in prison, or had abandoned them. It operated between 1911 and 1935, after which it became Aid to Dependent Children (ADC). The transfers increased family income by an estimated 30 percent and lasted for about three years. We followed 16,000 boys whose mothers applied for the pension until they died. Boys whose mothers received the pension lived about 1.5 years longer, had about 0.3 years more of school and about 10% higher incomes as adults, compared to boys whose mother applied but were denied. This is strong support for Marmot's recommendation of income redistribution.

But it's far too simple a conclusion. Other studies provide equally compelling evidence that in some settings more resources do not

improve children's lot in life. Bleakley and Ferrie (2016) document that land lotteries that distributed land to white males in the 1850s in the state of Georgia had no effect on their children's lifetime outcomes, measured by literacy, education, income and wealth. Nor did the land distribution affect their grandchildren, despite the fact that this was a large wealth transfer. A recent study by Price and Song (2016) follows the recipients of the famous Gary Income Maintenance experiments, which took place in the US in the 1950s. In this randomized experiment poor households were randomly assigned to different income-guarantee levels: the government would top-off the families' income to make sure they were above the poverty level. Yet again the children of those benefitting from the programs did no better than the children of those who did not. They do not appear to have lived longer.

Why are the results so different? The MP program targeted the poorest households (single mom with young children under 14), whereas almost the entire population of Georgia was eligible to win the land lotteries. In the Gary Income Maintenance case studied by Price, those who received guaranteed income faced large marginal income tax rates: if they worked and made more money, the government would send a smaller check. This resulted in lower labor force participation among the beneficiaries, and as a result the actual increase in family income was small.

But other evidence suggests that income transfers can work. Other "quasi-income" transfers like food stamps targeted to poor households appear to lead to better health (Hoynes et al. 2016). Another policy that seems to work to help poor working-age families is to increase wages, possibly through the tax code, with programs like the Earned Income Tax Credit (EITC). Marmot correctly points out that, despite popular perceptions, the majority of the poor today in the US are in fact employed. However their wages and earnings are low, and have not increased over time. A recent review of the EITC reports that it helps lift millions of working families out of poverty, and appears to increase adult and child health as well as child education (Nichols and Rothstein 2016). However the EITC does not help the unemployed or those out of the labor force. It conditions money on employment.

Evidence on the effect of conditional cash transfers (CCTs) across countries in the developing world shows great heterogeneity as well. CCT programs appear to increase school attendance and healthcare use in the short run, not surprisingly since these behaviors are required for the cash to be transferred. But evidence of their effect on "final outcomes" is much less uniform. Based on studies across several countries, many of which are from randomized interventions, Fiszbein and Schady (2009) in their review conclude "CCTs appear to have had

a modest impact on years of schooling completed by adults; they reduced the incidence of low child height for age only in some countries and only among some populations; and they resulted in modest improvements in cognitive development among very young children, but had no discernible effect on learning outcomes for children who benefited from CCT programs while they were of school age.”

These comparisons suggest that how the income is provided matters. There is indeed a great debate today about whether transfers to the poor should be conditional or unconditional on a set of behaviors, such as children’s school attendance or parental work; or be given in cash or in kind. The evidence also points to important differences in short and long term indicators of success. Overall there is much to be learned about how to best design these programs.

Marmot is also rightly concerned with high unemployment rates among youth: “I have described this youth unemployment as a public health time bomb.” There is indeed evidence that graduating in a recession leads to declines in employment and incomes that last for many years (Oreopolous et al, 2012). And cohorts that graduate in recessions have much worse health and higher mortality later in life (Cutler, Huang and Lleras-Muney 2016).

But how to help the young? Youth training programs have some benefits, but there is little consensus on the effectiveness of these programs, particularly over the long run. A recent meta-analysis of 200 training programs around the world by Card, Kluve and Weber (2015) suggests substantial heterogeneity in their labor market impacts: effects are small for youths and older workers. They also differ by type of program: some programs, like direct government employment, have negative effects; others, like job finding assistance, have positive impacts. And training has modest, but possibly increasing effects over time—though there is no evaluation examining impacts of any type of intervention beyond three years. There is also no evidence on their health effects (Barnow and Smith 2015).

Whether temporary cash transfers are sufficient to undo the negative effects of sustained unemployment early in ones’ career is also unclear, though we find some support for this claim. (In Cutler et al 2016 we find that the negative health effects of graduating in bad times are smaller in countries with large transfers, though we cannot claim this is causal.) But I know of no direct evidence showing that generous unemployment insurance tempers the health consequences of unemployment. And individuals graduating in bad times would not

be eligible for these protections, since they require prior employment as a condition for eligibility.

Similarly difficult questions arise concerning displaced workers. There is evidence they suffer substantial income and health losses (Sullivan and von Wachter 2009). But in this case it is even harder to point to potential interventions to help mature adults whose skills have become obsolete—training programs appear to have smaller effects on older workers, though they do seem to benefit the long term unemployed (Card, Kluve and Weber 2015). Marmot proposes “policies that create jobs”—I think all economists wish they knew what these are.

Early circumstances and neighborhoods?

Marmot proposes expanding early childhood interventions. There is high quality causal evidence to support the claim that early childhood interventions boost lifetime health. This evidence comes from studying the long-term impacts of randomized programs, like the Perry School and the Abecedarian programs that Heckman has studied extensively (Heckman 2006, Heckman et al. 2010). For instance, the Abecedarian Program, an intervention that gave disadvantaged children cognitive and social stimulation between birth and age 5, resulted in improved health in adulthood (after age 30) based on biometric data (Campbell et al. 2014). Head Start, the modern equivalent of the Abecedarian program, also appears to have long term health benefits, though this evidence comes from observational studies (Deming 2009, Ludwig and Miller 2007). Lastly, evidence from randomized trials of monkeys in captivity also provides strong support for large long-term health effects of adverse childhood circumstances (Conti et al. 2012).

But here again the details matter. Interventions later in life, during school years, appear to have much smaller effects (Reynolds et al 2011), though this might depend on the type of intervention: Heller et al (2016) find very large returns for behavioral interventions among adolescent boys from disadvantaged neighborhoods in Chicago. The benefits of Head Start vary substantially across children, depending, for example, on the quality of alternative forms of care that children receive instead of Head Start (Kline and Walters 2016). Program effects also differ depending on the type of services offered by providers and their quality (Walters 2015). For instance, a study by Baker, Gruber, and Milligan (2015) finds that the introduction of a universal day care program in Canada resulted in *worse* outcomes for affected children, most likely because the expansion was done rapidly and the quality of the care was not high. So when and who is targeted, as well as what exactly is given, matters for the size of the effects.

A similarly cautious conclusion must be drawn regarding neighborhood improvements. First, it is not clear how this is to be achieved. I know of no experimental evidence that has manipulated neighborhoods to improve social cohesion. But there is experimental evidence on the effects of moving to better neighborhoods, from the Moving to Opportunity intervention. It targeted poor families and offered them vouchers to move to richer neighborhoods. The effects on children were very mixed. Girls appeared to benefit somewhat, while boys did worse (Kling, Liebman and Katz, 2007). Recent evidence re-examining the experiment shows that the results depend heavily on the age of the children at the time of the move. Children who move to a better location do better in the long run. But adolescent boys do not deal well with moving (Chetty et al. 2016).

But of all of Marmot's proposals it is childhood interventions affecting cognitive and social development that have the strongest empirical support.

Rank and the workplace

Marmot has a large body of work examining the relationship between rank and health among British civil servants. This important and influential research has tracked a large number of individuals for over 25 years. Using high quality data it has found substantial correlations between rank (as measured by occupation) and mortality later on. It has also tracked health and socio-economic outcomes of over a long period. Marmot interprets the evidence as providing strong support that low rank causes poor health.

The theory that low rank causes low health has extensive support coming from fascinating animal studies. These studies document that lower ranked animals (typically primates or rats who have clearly established social hierarchies) have higher levels of stress-related hormones, such as cortisol. Researchers also observe that low ranking is associated with low control over food, mates, and physical safety - and unpredictable and uncontrollable events have been shown experimentally to lead to elevated stress hormones. And studies also show that when levels of stress-related hormones are repeatedly elevated, disease is more likely to ensue, because of reduced immune function, among other possible mechanisms (Sapolsky 2004).

More recent evidence comes from experiments with monkeys. Snyder-Mackler *et al.* (2016) *experimentally* manipulated the social rank of macaques in captivity and followed them for two years. Social rank in this study predicts immune regulation and response to infection at the cellular level: immune cells from lower ranking members were less

able to fight infectious disease in the lab. Together the studies provide a powerful and compelling story as to why and how low rank leads to poor health.

But it is unclear to what extent one can extrapolate these findings to human societies. Peer groups and rank are much harder to observe in large human societies. Individuals belong to multiple groups and opt out of groups where they have low rank. While the Whitehall studies are quite powerful, Marmot nonetheless overreaches a bit in their interpretation.

As with twin studies the population under study in Whitehall is highly selected and many important environmental influences are “controlled for.” This is very good for some purposes: e.g., we can more easily isolate other factors that matter. But the external validity of the conclusions is questionable. The population that serves in Whitehall is not representative because individuals self-select into the service. Whitehall is also a unique work environment.

Another limitation of these studies is that individuals start at a given rank within the civil service—and this is not randomly determined or independent of one’s history. Case and Paxson (2009) find that those from high SES families and in good health enter at higher ranks. This is not surprising: education is a large predictor of rank at entry in institutions with meritocratic hiring practices such as Whitehall. Case and Paxson (2009) and Eloviano et al (2011) also find that poor health in childhood predicts entry rank in Whitehall II, independently of family SES.

Moreover individuals within Whitehall advance in rank for some reason, possibly related to their own socio-economic conditions, their education, their family background and yes, their health. Indeed Case and Paxson (2009) find that current health status (and family background) predicts increases in rank within Whitehall. But current rank does not in fact predict future health.⁴

The evidence suggests caution. While the results from Whitehall show compelling associations between rank and health, they are insufficient to conclude that rank is the main causal mechanism at play. Because there is evidence of selection. Marmot claims that rank remains predictive of health later in life, even after controlling for education; and dismisses the selection argument. But there is too

⁴ Eloviano et al (2011) don’t find exactly the same. They find that conditional on SES at entry, further changes in health are mostly predicted by SES, rather than the converse.

much unexplained variation in health, even after controlling for the many factors in the Whitehall study: the fact that education and other measurable factors reduce the apparent effect of rank suggests that unobservable factors could entirely nullify the effect.

In Marmot's defense, identifying the causal effect of long-term conditions on health is a very difficult task. The effects of stress and rank are cumulative and only emerge after sustained and repeated stress exposure (at least in animals). There is substantially more and better causal evidence on the short-term effects of various factors. We also have now substantial evidence of the causal long-term impact of shocks, like in utero deprivation. But estimating the causal effect of factors that persist over many years (e.g., "breathing bad air from birth to age 18"; "living in stressful or violent households or neighborhoods"; "being in a bad job") is much more difficult. Given the strong association we observe more research on the causal long-term effects of rank and permanent stress should be undertaken.

Other recent studies also suggest important effects of repeated exposure. For example Case and Deaton (2017) document that since around 2000, the mortality rates of white adults in the US, particularly those with low education, have been rising. More recent cohorts are getting sicker earlier in life, and they are "going downhill faster" (their health is deteriorating faster with age). They conclude that "The data are consistent with long-run processes influencing outcomes, rather than contemporaneous shocks affecting health." They do not identify the causes of these long run processes. Proving causality of this type in humans is extremely challenging and an areas where research is needed.

Sin Taxes?

Throughout the book Marmot champions cigarette and alcohol taxes as both effective and desirable. In this respect he aligns with many economists. However I disagree with his enthusiasm on both factual and ethical bases.

While there is evidence that excise taxes lower cigarette and alcohol consumption, the effect is small. The demand for cigarettes among adults is inelastic: Gallet and List (2003) report that across 368 studies, the median (short run and long run) elasticity of demand with respect to price is roughly -0.4. Although the value varies across studies and populations, most studies find it to be below one (with the exception of teens). The same is true for alcohol and illegal drugs, which also have inelastic demands. (The median price elasticity for alcohol across 91 studies was -0.5, Gallet 2007)

Of course this is not surprising because these goods are addictive, and have no adequate substitutes—individuals tend to consume them regardless of price. This is the very reason why these goods have been taxed since time immemorial: they provide a predictable source of revenue for the government, unlike excise taxes on other goods. So although sin taxes have some effect on consumption, the effect is small. Thus if we wish to use taxes to regulate unhealthy consumption, taxes must be set at very high levels.

But taxes on these goods are regressive, particularly if consumers fail to adjust their consumption. Smoking rates are much higher among adults that are low educated and poor; only 10% of college graduates smoke, compared to 30% of high school drop-outs. And 32% of those below the poverty line smoke, compared to 20% of those above it (Center for Disease Control 2015). The same is true of those consuming opioid pain killers illegally (Case and Deaton 2015). By increasing taxes on poor or uneducated consumers, we do not necessarily induce them to stop consuming—we simply make them poorer. At the extreme (as in the case of some illegal drugs), high prices lead to crime and further social ills.

Consumption of these goods is highly social and subject to important social and contextual influences. We eat and drink with friends and at parties; we smoke when others are smoking; we use pain killers when situations are difficult to handle. These observations, along with our improving understanding of how the brain works, leads to different conclusions as to how best to address consumption of goods that have adverse consequences.

As Fudenberg and Levine (2006) note “many sorts of decision problems should be viewed as a game between a sequence of short run impulse selves and a long-run patient self”, a view most famously expounded in Kahneman’s book “Thinking Fast and Slow”. The rational self responds to taxes and prices. But the consumption of addictive goods is significantly driven by irrational impulses. Thus prices matter somewhat because the rational long term decider in us has some say—but they matter only a little because our impulsive self overrides these decisions in the short term.

It is therefore essential to understand what controls short-term impulses. As Berheim and Rangel (2004) explain, what matters for kicking addiction is the removal of the cues in the environment that trigger consumption and recidivism. Most successful programs that deal with alcoholism and drug addiction require rehab: a drastic change in habits and social networks. They do not rely on information

provision or price changes. Most addicts do in fact know addiction is bad, want to stop consuming, but can't, despite high prices and steep social (and sometimes legal) consequences. Instead new social support systems and changes in the environment are the cornerstone of successful programs like Alcoholic Anonymous.

The study of alcohol and cigarette consumption highlight limitations of the traditional economics model of health production, in the spirit of Grossman's classic paper (1972) and Becker and Murphy's seminal rational addiction model (1988). In addition to assuming full rationality, this is a model of individual decision making. Although prices, information, income and preferences matter, these factors typically explain only a small portion of observed differences in health behaviors.

Social influences would seem to matter a lot. And a long literature has established strong correlations between friends and family members in their behaviors at a point in time (Durlauf and Young 2001). Moreover behaviors and peers evolve jointly (Christakis and Fowler 2007 and 2008). Peer effects in behaviors are extremely difficult to establish, particularly because individuals select their friends (see Heckman 2010 for a review). But recent papers exploiting random assignment into groups do support their importance. Carrel and colleagues show that fitness of one's (randomly assigned) mates affect one's fitness in the Air Force, as well as other outcomes (Carrell et al 2011). Most convincingly, work by Centola and colleagues provides strong support for the notion that network structure and the behavior of individuals in the network have strong influences on health behaviors. It documents that experimentally varied structure and composition of (online) networks affects health behaviors (Centola 2011, Zhang et al 2015).

But here I would cite Marmot himself, and others like Link and Phelan (1995), to propose that in order to fight excessive alcohol consumption, pain-killer abuse, illegal drug abuse and consumption of other bads, we must address circumstance. Case and Deaton (2015) characterize the recent increase in suicides, liver-related and drug-abuse related deaths as "deaths of despair." It does not matter how one fights one specific vice. If individuals are in pain they will find other ways to assuage it. Lack of education, income, social connections, or poor economic prospects surely do cause despair. Taxing sin will not eliminate it.

The causes of disease

Marmot's primary thesis is that low socio-economic status (SES) causes disease. Poverty, lack of education and/or lack of control make people

vulnerable to many diseases. And so if we lower SES disparities, then health disparities will disappear. Though Marmot interprets the large literature documenting correlations between SES and disease as one-way relationships, there is ample evidence that disease causes SES. Marmot claims this second channel is small or ignorable—but that has not been demonstrated. Marmot is more likely to be correct about the relative importance of SES as a contributor to total health in the case of developed economies, but even then his dismissal of the possibility that health affects socio-economic status is not warranted.

Disabilities cause poverty because they affect individuals' ability to study, work and earn a living—this was the main rationale for the provision of disability insurance in western countries. According to the social security administration, one in four individuals become disabled before they retire in the US today.⁵ Similarly the natural process of aging eventually leaves individuals with impairments that make it impossible for them to provide for themselves, and leads to poverty in old age, at least for those without accumulated wealth or families to support them. In developing countries the elderly have the highest poverty rates of any group (Schwartz 2003), and old-age pensions lower them substantially (Case and Deaton 1998). Increases in the generosity of old-age pensions have substantially reduced elderly poverty in the US since the 1960s (Englehard and Gruber 2006). I see the fact that disease and poverty increase with age as evidence that poor health causes poverty. Marmot does support old age pensions strongly but strangely does not consider the case of the elderly as providing evidence that poor health leads to poverty. (Marmot asserts that only economists think that health causes SES: “if someone comes across the social gradient in health and assumes that health leads to socio-economic position, rather than social circumstances lead to health, then he is an economist”).

Malaria, HIV, tuberculosis, and other diseases with large effects on individuals' ability to study and work are still prevalent in many countries (Strauss and Thomas 1998). Giving money or education to individuals in these countries would not eliminate the disease environment they face. Other policies, like malaria and worm eradication, might be more effective in reducing health gaps. Similar arguments could be made, for instance, about malnutrition—providing iron supplements or iodizing salt could be cheaper, better solutions.

These counterexamples all come from developing countries. But within developed countries is it really health that leads to income? Marmot claims not, but his conclusion is too simplistic. It is true that the

⁵ <https://www.ssa.gov/pubs/EN-05-10029.pdf>

eradication of most infectious disease and the improvement of nutrition have likely lowered the chances that poor health in childhood causes low SES in adulthood in rich countries. Evidence remains, however, that unlucky health events are predictive of lifetime socio-economic outcomes in rich countries. For example in utero exposure to the flu in Denmark results in lower adult earnings (Schwandt, 2017). Many papers indeed find that unlucky events in utero have long term effects in labor market outcomes (Almond and Currie 2011, Almond et al 2017).

Marmot might object that exposure to bad in utero events is likely tied to parental SES, so it is all about SES causing health. But this is not true. In Britain, Case et al. (2005) find that “Controlling for parental income, education and social class, children who experience poor health have significantly lower educational attainment, poorer health, and lower social status as adults.” Birthweight predict adult health and earnings, even among identical twins, and regardless of parental SES (Barker 1995, Black, Devereux and Salvanes, 2007). Recent evidence on the effects of the Clean Air Act of 1970 in the US finds that those exposed in utero to greater pollution had lower labor force participation and earnings at age 30, compared to children in the same counties after reductions occurred (Isen et al, forthcoming). Exposure to radiation fallout in utero results in lower education and adult earnings (Almond, Edlund and Palme, 2009, Black et al 2016)—exposure to fall-out depends on wind trajectory in these studies and is unlikely to depend on parental SES. In fact in the Black et al. study more educated individuals were more likely to be exposed. Moving into adulthood, Smith (1999) shows that income changes are not very predictive of health changes, but the onset of new illnesses result in lower labor force participation and earnings. Negative health shocks strongly predict retirement and reduced labor force participation (Smith 1999, 2005; Case and Deaton 2003).

It does not help Marmot’s ultimate aim to improve health and minimize health gaps (and other gaps in fundamental freedoms, a la Sen) to advocate a simple SES-to-disease view. That’s because many diseases are not caused by SES and would be best (most cost-effectively) cured by non-SES interventions; and also because some diseases have large SES consequences, and we would like to maximize both health and income (and minimize gaps in both). The optimal policy is likely to vary over time and place.

A couple of specific non-SES policies that Marmot does not highlight are worth mentioning. One is environmental regulation. As mentioned already, pollution causes short term disease and mortality, and lowers productivity in the workplace and performance in school, even at low

levels of exposure. And a few studies find long term consequences of exposure on cognition, education, incomes and disease in adulthood. The same is true about exposure to radiation, lead and other toxins found in the air and in the water (Currie 2013, Aizer et al 2016, Almond et al 2017); of course these results are not from randomizations, but experimental animal studies support the conclusion that these factors have both short and long term consequences on a large number of outcomes (e.g., Morgan et al 2011, Davis 2013). Moreover, exposure to toxins is higher among those from low SES, and they would disproportionately benefit from tightening environmental regulations. So greater regulation of pollutants is likely to maximize health and incomes, and minimize SES health gaps.

Obesity is another interesting case. Until very recently poverty was associated with under-nutrition, stunting and malnutrition, but today the poor are much more likely to be obese. The exact reasons for the reversal of this relationship are not well understood. But obesity does lead to disability and often limits work capacity and earnings. Obesity policy is a subject of extensive academic and policy debate. But it's not clear that policies that give money to the poor would be more (cost) effective than policies that directly attempt to promote healthy eating and exercise habits in school, at home, and in other social spheres. And it might turn out that obesity has other important causes (in addition to excessive fat, sugar or caloric consumption, or insufficient exercise regimes). Recent evidence suggests that low biodiversity of the intestinal flora is correlated with obesity (Ley et al 2006). In lab experiments, modification of this flora leads to obesity in rats (Ridaura et al 2013). Why obese individuals have less bio-diverse guts is not known, though it has been suggested it's caused by antibiotic use or the composition of food. Regardless these patterns suggest treatments for obesity, other than SES-based policies, may be more effective.

Focusing policy on eradicating the most prevalent diseases, such as respiratory diseases and obesity, has some advantages over the SES-based policies Marmot advocates. First and foremost, this approach will receive political and financial support, because even though these diseases disproportionately affect the poor and uneducated, they also affect the rich and the educated. Secondly this approach is inclusive in that it benefits many individuals, and it achieves Marmot's goal to increase health *and* lower health gaps. As mentioned before, clean water, salt ionization, malaria eradication and other blanket anti-disease policies have increased life expectancy and lowered health gaps. Lastly a disease-based approach forces researchers to examine the multiple levels and mechanisms through which disease emerges, from biological to social to economics. This yields better insights as to how to fight disease. Increases in resources that are not accompanied

by knowledge on how to avoid and treat disease are unlikely to generate the greatest gains in life expectancy.

In summary, there is evidence that income and SES cause health; but there is also evidence of the converse. And there is also evidence that other factors, which are relatively independent of SES or individual characteristics, like the disease environment, matter. There is no good decomposition of the extent to which each of these three possibilities account for the observed correlation between health and SES. Moreover this decomposition is likely to vary greatly across contexts. Marmot's position that SES is the only (or the main) determinant of health, and the only explanation for the correlations we observe in the data, is too simplistic. Health is a complex process with many determinants—the best approaches to improve it are likely to vary greatly depending on the disease and the larger setting or context.

Moving forward

The evidence shows that there are individual socio-economic markers (e.g., education at age 25; entry rank in the workplace; income at age 40) that are great predictors of health and mortality after age 40. These health gaps are large and growing. In addition to compellingly documenting these health inequities, Marmot urges us to address these health gaps, and to adopt policies to redress them. He provides ethical reasons for intervention, and makes a compelling case for more, and more inclusive (universalist) policies. Although there is debate about what policies are best suited to address these differences, the fact that longevity around the world increased so dramatically in the last two centuries suggests we can improve health for all.

I do not fully agree with Marmot's interpretation of the evidence on the determinants of health. And I am much more cautious about interpreting the evidence on what works. But Marmot's proposals are sensible: If I had to gamble, based on current evidence, on how to address health *and* income gaps, his policies (early childhood education, redistribution, employment, prevention) would be on my short list—in part because these policies are likely to have many potential gains, in terms of income, health, and broader welfare. But the relative costs and benefits of each should be carefully considered against alternatives.

The evidence so far most strongly supports early education interventions, and possibly income transfers through the tax code. But even in these cases there is a lot of heterogeneity in the effect of

programs—this is true about almost all the interventions I have reviewed. What drives heterogeneity in treatment effects? Can this heterogeneity be characterized, modeled and used prospectively to better design programs and better target them? We need to make more careful recommendations that are based on a clearer understanding of why things work, when, and for whom. As Deaton (2010) argues “the analysis of projects needs to be refocused toward the investigation of potentially generalizable mechanisms that explain why and in what contexts projects can be expected to work.”

Another important observation is that the effects of interventions can differ substantially in the short and long term, often in surprising and unpredictable ways. Today in rich countries chronic diseases, occurring mostly among adults, are the main killers. SES and other conditions measured relatively early in life predict the onset of chronic diseases later in life. Chronic diseases are the result of slow cumulative processes, where exposure to certain factors over many years is important. Short-term insults, whose effects are not fully felt for many years, also matter. The evidence that Marmot presents, and evidence from recent studies, suggests it is becoming increasingly important to understand the long-term causal effects of *persistent* exposure to various factors like stress and how these effects evolve. And the same is true about the effects of interventions, whose dynamic effects are poorly understood.

More importantly, even with a clear understanding of what works, some key issues are political. Marmot does not spend much time considering why it is so difficult to maintain support for social insurance and redistribution in the US, among other countries. Even if we agreed on what works, it is very difficult to establish and maintain political support for these programs.

Despite my disagreements with Marmot, I admire the lucidity with which he exposit his arguments and the considerable effort it takes to assemble and interpret the evidence in a cohesive framework. And I found myself moved by the arguments and the descriptive evidence. Inequality within the US and in many other countries of the world today is at an all-time high. This can be measured in many ways, by looking at traditional income and wealth metrics, or by looking at social mobility. It can also be seen in the highly unequal levels of health and longevity within countries. Health is a form of wealth. Low health and low wealth constitute a double deprivation. How exactly these facts are linked is subject of debate. But it is clear that this is not a situation we can ignore. In this I support Marmot’s call to arms.

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