# 16. Early childhood origins of modern social class health disparities

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## BACKGROUND

There is a widespread consensus among historical demographers that socioeconomic differentials in health and mortality are a rather recent phenomenon in the history of homo sapiens. In a classic study, Hollingsworth (1977) argued persuasively that, as early as the 17th century, mortality differentials between the wealthiest and the destitute in England were either minuscule or the reverse of what they are now. More recent historical accounts of the demography of pre-industrial populations suggest that the emergence of mortality inequalities as a fixture of modern populations dates back at the earliest to the period when mortality began a secular decline (Antonovsky, 1967; Bengtsson and Dribe, 2011). Their initial manifestations roughly coincide with the beginning of a period of sustained gains in human height after 1800 (Fogel, 2012). Necessary conditions to support disparities surge throughout the 19th and first half of the 20th century, because of inequities in the social distribution of improvements in nutritional status and dissemination of new scientific knowledge. They were strengthened in the last quarter of the 19th century as a result of unequally allocated benefits from the diffusion of germ theory and massive public health works for sewage water purification and distribution. A new regime emerges after 1930 with the widespread dissemination of new medical technologies that drastically reduced the burden of infectious and parasitic diseases. Following a short-lived period of contraction, health disparities increased and then mutated to become characteristic of modern epidemiological regimes that followed massive reductions in mortality.

It is indeed paradoxical that the most significant achievement in human history, the conquest of infectious diseases, became a new vehicle for the establishment of one of the most disfiguring features of modern societies, the gaping social class disparities in health status and length of life. Their receding importance, and the consequent improvements in survival to older ages, opened the gates to chronic illnesses, sensitive to an entirely new array of determinants that are also unequally distributed across social classes, including new exposures and new behaviors. Disparities associated with chronic illness would have grown because of larger fractions of the population reaching post-reproductive senescence (Medawar, 2019). Because fatality rates associated with post-reproductive diseases are tied to access to modern medical care, there is plenty of room for health and mortality disparities to persist and grow in any stratified societies with high life expectancy. We will argue below that modern health and mortality inequalities are powered by a unique set of pathways, the product of a peculiar combination of modern epidemiological regimes and social class systems. These pathways are by no means the only ones that account for modern disparities and, in some populations, may not even be the most important. Their uniqueness is rooted in that (i) they are only viable in populations characterized by modern stratification and epidemiological systems and (ii) can only be manifested over long periods of time, throughout the life cycle of individuals. Our goal

is to define a framework that includes these pathways as determinants of modern social class health and mortality disparities.

The chapter is organized in six main sections. The first section describes the uniqueness of modern epidemiological and social stratification systems that combine to produce a modern regime of health and mortality disparities. The second section briefly reviews recent estimates of mortality differentials by education in the US and Europe and confirms that, over the past 20 years or so, health and mortality disparities have become a systemic feature of advanced societies. The third section defines a causal model that includes early direct and indirect conditions as explanatory factors, highlights the importance of early health and early acquisition of capabilities and skills, and points to the potential role for self-replication of disparities across successive generations. In the fourth section we quantify the contribution of direct and indirect pathways to mortality disparities associated with obesity (and metabolic diseases) and smoking. The fifth section briefly discusses the intergenerational transmission of health disparities and the last section summarizes and concludes.

## EPIDEMIOLOGICAL REGIMES, SOCIAL CLASSES, AND HEALTH DISPARITIES

The literature in epidemiology and population health has long since adhered to the idea that human populations have undergone multiple epidemiological transitions. In the standard rendition (Omran, 1971), there are three stages culminating in a shift from a regime of infectious and parasitic diseases to one dominated by chronic conditions. In more recent versions, a new epidemiological regime is added to represent populations in which illnesses affecting the very old become salient (emerging chronic illnesses, cognitive and neurological disorders) (Olshansky and Ault, 1986). Next, we focus on three dimensions of epidemiological regimes that are not explicit in the original epidemiological transition framework.

### Unequal Allocation of Early Conditions and of Opportunities of Class Accession

In contrast to pre-modern regimes, adult health and mortality in modern epidemiological regimes can be associated with exposures whose manifestations may take a lifetime to be observed. The chain of events that leads from an initial risk of exposure to a final health outcome may take a very long time to unfold, could be heavily influenced by initial conditions experienced early in life, and may be modified to variable extents by subsequent events. Some infectious and parasitic diseases, such as rheumatic heart fever, HPV, respiratory TB, and some forms of malaria (plasmodium vivax), may also have delayed effects. But this is not typical of most infectious and parasitic diseases prevailing during the period 1750–1930. In modern epidemiological regimes, very low levels of infant and child mortality translate into large increases of the probability of surviving to critical ages after which adult delayed effects of early exposures may be observable. When child mortality levels are of the order of 200 per thousand, less than 45% of the population survives to age 60, in contrast to 95% in cohorts born after 1930. Because the quality of early exposures across a population is not random but strongly influenced by parental social class, they may have impacts on health and mortality disparities far exceeding those they had in preceding epidemiological regimes. This

is reinforced in modern social stratification systems characterized by significant correlations between parental and offspring social class.

Early exposures may alter processes of cell growth and differentiation in early embryonic life, fetal organogenesis, and epigenetic dysregulation, all related to maternal and parental exposures (including nutritional challenges and life stressors). In our framework, this source of disparities brings together two strands of empirical research that, until recently, grew disconnected from each other, namely, the analysis of life course determinants and the developmental origins of health and disease (DOHaD) (Gluckman, 2006; Gluckman, Buklijas, and Hanson, 2016; Gluckman and Hanson, 2004).

**Unequal Allocation of Early Resources that Minimize Health Risks and Optimize Social Class Accession**

Another peculiarity of modern epidemiological regimes is that they are shaped by conditions highly dependent on individual preferences, choices, and behaviors, some of which are rooted in early formative years and modified by socialization processes strongly influenced by family and class of origin. This, by itself, is enough to promote social class health inequalities. However, the mechanism is buttressed because modern stratification systems are founded on selective social class accession rules that favor individuals with skills and traits that happen to be associated with characteristics that minimize exposure to chronic illnesses. The net result is that those born in favorable social class positions are more likely to develop traits that simultaneously minimize health risks and favor their own social class standing. There will be a correlation between traits acquired early in life that influence both individuals' opportunities of social class accession and their exposure to health risks. In an offspring generation, these traits are the outcome of genetic, epigenetic, and sociocultural inheritance from parents, family of origin, and school experiences. An important body of empirical research on some aspects of this source of disparities refers to it as health selection.

**Replication of Disparities: Heritability of Health-related Traits and Social Class**

In societies founded on a social class system, there are multiple mechanisms that generate a correlation between parental and offspring social class. Because some of these mechanisms are associated with early health conditions and trait formation and their interrelations, a regime of health and mortality disparities prevailing at one time will depend on its past, has a momentum of its own, and may persist for at least one generation, even after the conditions that originated it weaken or vanish. Thus, a modern epidemiological regime facilitates the intergenerational transmission of health disparities.

There is a body of very rich research on the aforementioned sources of modern health disparities. But this research strand has grown separately from DOHaD and our contribution is to integrate them into a single framework that draws from past work in epidemiology and population health (Kuh and Ben-Shlomo, 2004), health and labor economics (Cunha and Heckman, 2007; Grossman, 1972, 2000), and the literature on sociocultural heredity (Bonduriansky and Day, 2018; Cavalli-Sforza and Feldman, 1981). Emphasis on these sources of disparities (and associated mechanisms) is neither meant to ignore direct, causal, impacts (see below) nor serve as a distraction from the importance of social class as a "fundamental" determinant (Link and Phelan, 1995). Health disparities are an inevitable product of stratified social systems, not

just the result of weakly related components that can be understood separately from each other. Our objective is to highlight mechanisms that are unique in modern stratified social systems and that happen to coexist with a modern epidemiological regime.

## HEALTH AND MORTALITY INEQUALITIES: HOW LARGE ARE THEY?

Analysis of health and mortality inequalities has been carried out on multiple health outcomes and using a broad array of metrics of individuals socioeconomic ranking. Indicators based on mortality rates have been the workhorse in this literature whereas social class and socioeconomic ranking are traditionally assessed with occupation categories, occupational prestige scores, income, wealth, educational attainment, self-assessments, and combinations thereof. There is agreement, however, that estimates of educational disparities are the most robust to multiple pitfalls, are moderately comparable across populations and over time, and are most abundantly documented in the literature.

### Magnitude of Recent Mortality Disparities by Education in the US and Europe

In the online Appendix to this chapter,[2] we compute harmonized estimates of life expectancy at age 45 and associated relative mortality risks. These calculations include data from the US and selected European countries and refer to birth cohorts and cross-sections whose mortality experiences reflect conditions experienced after 1950 approximately. These estimates lead to two main inferences.

US adult mortality educational disparities reflected in life expectancy at age 45, E(45), are relatively large, they hover in the range 3.0 to 6.0 years and, on average, suggest that those with lowest education (less than high school) experience residual lifetimes 10–20% shorter than those with more than high school education. These disparities are ubiquitous across the life span, graduated by education categories and, finally, increasing over time and across birth cohorts.

A review of recent estimates of mortality disparities by education in multiple countries in the EU point to inequalities in life expectancy and relative mortality risks at age 45 of strikingly similar magnitude to those found in the US, they also point to an increasing trend in the most recent periods and, finally, are ubiquitous although variable across countries.

### Causes of Death Responsible for Mortality Disparities

A detailed accounting of disparities by causes of deaths is beyond the scope of the chapter. However, there is strong empirical evidence showing that the main causes of death in modern mortality regimes are also responsible for the bulk of education mortality differentials (Glei, Lee, and Weinstein, 2020). These include ischemic heart disease (CHD), hemorrhagic and ischemic stroke, cancers (breast, bronchial and lung, bladder, liver), Type 2 diabetes and, finally, accidents, homicides and suicides (AHS) (Mackenbach, 2006). With the exception of AHS, these are all chronic conditions whose causal roots are multifactorial and have been associated with environmental exposures of various kinds and individuals' life styles. Although this remains to be investigated more thoroughly, there are two features of modern

individual life styles that may ultimately account for a significant fraction of cause-specific mortality disparities: obesity and smoking persistence.[3]

Smoking is known to be a behavior that starts very early in life and is responsive to parental, kin, and peer influences. Obesity, on the other hand, is a condition influenced by a host of allelic variants, maternal obesity before and during pregnancy, infant and childhood diet, adolescent food preferences, and physical activity. Note that in the US at least, smoking accounts for most of socioeconomic disparities in mortality from smoking-related diseases. In addition, smoking alone contributes to about 38% of cardiovascular disease mortality and 34% of all-cause mortality. Thus, a low bound estimate of the contribution of smoking to overall mortality differentials is about 21% (Glei, Lee, and Weinstein, 2020). A similar, if not a higher, figure must apply to obesity and, as a consequence, it may well be that jointly these two conditions could account for over 40% of observed disparities. Because disparities in smoking and obesity prevalence have been widening over time, their contribution to overall disparities must be increasing also. In short, identifying the role that early conditions may have as triggers of smoking adherence and behaviors that induce obesity, may go a long way in explaining mortality disparities documented before.

We end this section by noting that it is nothing short of remarkable that highly heterogeneous populations that differ in their social class stratification, central government organization, medical health care systems, and the reach of social programs enacted to level the playing field, all exhibit roughly similar levels and trends in mortality disparities and comparable contributions of causes of death. Below, we argue that this is the result of conditions prevailing in all stratified social systems that share a modern epidemiological regime.

## A CAUSAL MODEL FOR HEALTH DISPARITIES

We now introduce a model that identifies mechanisms responsible for modern social class disparities emphasizing direct and indirect pathways that originate in early exposures. We also discuss conditions required for these pathways to be efficient vehicles for the production and replication of social class disparities. The model represents chains of events over the life-course, highlights intergenerational links, and emphasizes the relevance of cognitive and non-cognitive skills (human capital). The variables in Figure 16.1 represent constructs measured at some point in the life course, while the arrows connecting them represent causal relationships.[4]

In high-income societies at least, the bulk of mortality disparities is attributable to chronic illnesses (Mackenbach, 2006). At least part of the observed association between social class and adult health and mortality associated with chronic conditions have roots in processes that start early in life and influence both social class accession and determinants of health. In Figure 16.1, we distinguish between parents' social class and childhood environments (including pregnancy and maternal conditions). We use a rather broad definition of childhood environment or early life conditions and include factors that can jeopardize, impose constraints on, or enhance children's early development. For example, the quantity and quality of maternal care, environments experienced at home (parental education, family income, welfare, stress, diet and physical activity, alcohol and tobacco exposure), and school experiences. We also include exogenous events that may alter children's life trajectories, such as wars, famines, or social contexts such as neighborhood poverty, violence, and crime. For the sake of simplicity,

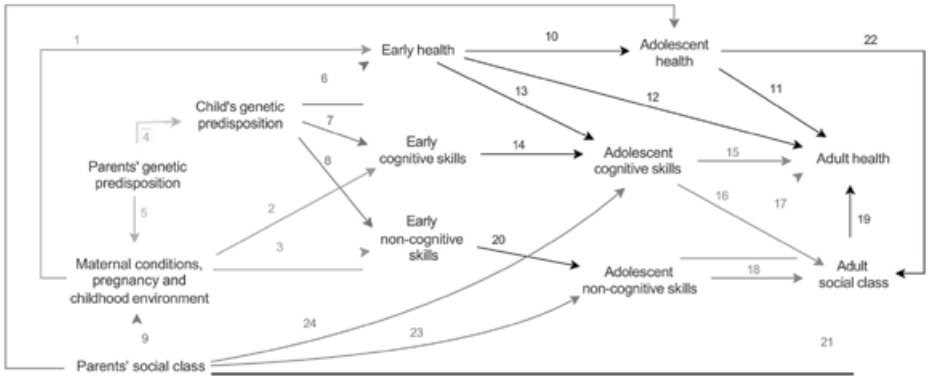


*Figure 16.1 Life-course causal model for health disparities*

the model ignores the timing of events and duration of exposures (in-utero, throughout infancy or childhood, or during adolescence), even though some of their impact is highly sensitive to these dimensions.

Figure 16.1 includes two different sets of pathways, direct and indirect. We describe each in turn.

**Direct Pathways that Originate in Early Conditions**

There is abundant empirical evidence showing that adverse environments during the fetal period, infancy, and early childhood increase the adult risk of chronic illnesses such as cancers, and metabolic and cardiovascular diseases (see pathway 1 in Figure 16.1) (Gluckman, Hanson, and Beedle, 2007). The mechanism depends on parental factors that influence preconception exposures, in-utero, and post-natal conditions associated with nutritional challenges and stress response. Some of these involve epigenetic modifications, others include alteration of cell specialization and growth. In all cases, they may have a long reach depending on the timing of exposures and individuals' resilience.

Parental circumstances and early life play a significant role, since adversity that occurs during these critical periods can permanently set health trajectories in ways that equivalent exposures experienced outside of those periods do not. Maternal conditions prior to gestation can affect offspring early health, and influence cognitive and non-cognitive skills independently from adversity experienced during gestational, infancy and childhood periods. Additionally, there is considerable evidence suggesting that parents' genetic predisposition to deleterious health conditions influences offspring risks of adverse health and undermines cognitive and non-cognitive trajectories. Aside from parental circumstances or genetics, adversity encountered while in the womb and during early infancy will compound the effects of parental circumstances prior to gestation.

Table 16.1 is a highly stylized summary of the nature of sources, mechanisms and health outcomes related to exposures experienced preconception, in-utero and during infancy and early childhood. The table is maximally simplified as, among other things, it only highlights a very selected set of direct pathways leading from early exposure to adult delayed effects.

*Table 16.1 Classes of direct pathways related to obesity and T2D*

| **Source** | **Child outcome** | **Adult outcome** | **Mechanisms** | *References* |
|---|---|---|---|---|
| Maternal prepregnancy BMI, gestational weight gain, gestational diabetes | Child obesity<br>Metabolic dysregulation | Obesity<br>T2D | Genetic<br>Epigenetic<br>Physiological | Oken et al., 2009<br>Simeone et al., 2015 |
| Fetal caloric nutritional restriction | Child obesity<br>Organosesis irregularities<br>Metabolic dysregulation | Obesity<br>T2D<br>CHD<br>HBP<br>Kidney diseases<br>COPD | Homeostasis<br>Epigenetic<br>Fetal programming<br>PAR | Barker et al., 1989<br>Gluckman, Hanson and Beedle, 2007 |
| Fetal nutrient imbalance | Adipogenesis irregularities<br>Metabolic dysfunction | Obesity<br>T2D | Epigenetic | Gernand et al., 2016<br>Gertler and Gracner, 2021<br>Patti, 2013 |
| Maternal, fetal, infant stress | HPA dysregulation | Obesity<br>T2D<br>Cognitive and non-cognitive limitations | Epigenetic<br>Physiological | Entringer, Buss, and Wadhwa, 2015<br>Kuzawa and Quinn, 2009<br>Thayer and Kuzawa, 2015 |
| Maternal smoking | HPA dysregulation | Obesity<br>T2D<br>Respiratory disease<br>Cognitive and non-cognitive limitations | Epigenetic<br>Physiological | Al-Amri et al., 2021<br>Mattsson et al., 2013 |

They share one property: they are all associated with adult conditions that account for an important fraction of chronic conditions associated with modern health and mortality differentials, namely, obesity, Type II diabetes (T2D), stroke and CHD.

### Indirect Pathways that Originate in Early Conditions

Early conditions may influence the acquisition of early skills and capabilities (paths 2, 3). Maternal behaviors right before and during pregnancy, fetal environments, conditions in infancy and early childhood, can affect processes of cell growth and specialization and, through them, have an impact on connectivity of brain tissue, on which the development of cognitive and non-cognitive capabilities depends. As a result, these early experiences may have a long reach.

First, in modern stratification systems, educational attainment strongly affects the allocation of individuals in social classes. It happens to be the case that early cognitive (and some non-cognitive) skills will partially determine adolescent cognitive skills (path 14), adult education and, through it, an individual's social class (path 16).

Second, cognitive and non-cognitive abilities are strongly and independently associated with a range of factors that influence health outcomes (path 15). Most importantly, among these, are those involved in the formation of individuals' health behaviors on which the timing

and duration of exposure to risks of chronic illness depends. They also regulate preferences and choices that may interfere with individuals' ability to access resources that enhance resistance and recovery from disease. As a result, early conditions and experiences may constrain the repertoire of skills an individual will command, derail education trajectories and, simultaneously, reduce the array of resources available to minimize exposures or improve resistance to and recovery from illness. Thus, the link between early health and cognitive and non-cognitive skills (path 13) and the effect these have on both adult health (paths 15, 17) and social class (paths 16, 18) is a pathway for health and mortality disparities. In past empirical research this has been referred to as indirect health-selection. There is a robust body of empirical evidence showing that this pathway could be partly responsible for the education (or any social class) health gradient (Manor, Matthews, and Power, 2003; Power and Jefferis, 2002; Stern, 1983; West, 1991).[5]

**Other Causal Pathways**

Although our interest revolves around direct and indirect pathways rooted in early conditions, modern health disparities have other sources. Most importantly, membership in a social class by itself constrains access to resources that minimize exposures to risks and augment resistance and recovery (path 19). These effects do not operate through childhood environment and early health, genetic predispositions, or cognitive/non-cognitive skills. Their impact is causally direct in the sense that it stems from differential access to resources available to a social class. These include wealth, income, education, all of which facilitate purchase in open markets of medical care, favorable residential settings, health insurance, and health information (Cutler and Lleras-Muney, 2010). Some of these resources might influence the formation of adult attitudes, preferences, tastes, choices, or behaviors that minimize risk exposures. Research on educational mortality disparities attribute some of the effects to resources endowed to those who attain some level of accreditation by virtue of the accreditation itself (Halpern-Manners et al., 2020). Thus, educational attainment facilitates acquisition of personal traits and capabilities such as self-efficacy (Mirowsky and Ross, 2005) implicated in the adoption of beneficial health behaviors (paths 23, 24). This is different from the alternative explanation we favor here, in which the association between education and mortality is a result of complex pathways that produce both low mortality risks and high educational attainment. Although empirical evidence supports both approaches, the empirical evidence is partial, as it only applies to some subpopulations, and incomplete, as the models tested do not include all the relevant pathways.

**Manifestation of Pathways**

As it stands, the causal model we formulated above is incomplete and should be extended in two directions. First, health and mortality differentials are the result of mechanisms that minimize exposure to illness (E) and maximize individual capabilities of resistance (R) to and recovery (Re) from illness over the life-course. Any indicator of health status or stock we care to use is invariably the joint result of exposure, resistance, and recovery (ERRe). But since each dimension of the triplet ERRe may be the result of partially independent causal processes, using a shortcut to bundle them into a single outcome will necessarily obscure important mechanisms. For example, the impact of unequal access to modern medical care, an increasingly important factor accounting for modern disparities, is only relevant to the extent

that it affects the R or Re dimensions. This, in turn, may be a function of pathways involving early health and cognitive and non-cognitive capabilities.

Second, the explanatory power of a mechanism generating disparities is a function of the precise nature of the dominant epidemiological regime. As a result, health and mortality disparities of similar magnitudes in different populations or historical periods could be the result of different processes. Any general model of health disparities should include parameters that regulate changes in the nature of gradients as a function of shifts in epidemiological regimes. A formal rendition of the model in Figure 16.1 could be as follows:

$$H_i(t) = hE_{it}^{\varepsilon} * Z_{1,i(t-k)}^{\sigma_{1,H}} * Z_{2,i(t-k)}^{\sigma_{2,H}} * Z_{3,i(t-k)}^{\sigma_{3,H}} \tag{16.1}$$

$$E_i(t) = eF_{i(t-k)}^{\phi} * W_{1,i(t-k)}^{\theta_{1,H}} * W_{2,i(t-k)}^{\theta_{2,H}} * Z_{3,i(t-k)}^{\theta_{3,H}} \tag{16.2}$$

where H and E are health status and education attainment, the Zs and Ws are vectors of early health conditions (1), cognitive (2) and non-cognitive (3) traits, all fixed at time t − k and sharing at least some common elements. F is a vector of family characteristics, h and e are additive effects of exogenous factors (including random errors) and, finally, Greek letters are elasticities which are implicit functions of characteristics of the prevailing epidemiological regimes. For example, a shift from a regime dominated by infectious diseases to one dominated by chronic illness is manifested in an increase first of σ1,H and then of σ2,H and σ3,H. Similarly, in modern stratification systems the magnitudes of the θ's elasticities should be larger than in stratification systems that constrain social class mobility. Modern populations that are combinations of different epidemiological and stratification systems will be characterized by different values of σs and θs.

A final but important feature is that the size of ε, the direct causal effect of E, is likely to be a function not only of epidemiological and stratification regimes, but also of some of the early inputs included in vector Zs and Ws. For example, the executive capacity with which highly educated people may be endowed is likely to be dependent of early non-cognitive capabilities, such as self-reliance and perseverance.

## ILLUSTRATIONS OF DIRECT AND INDIRECT PATHWAYS: OBESITY, TYPE II DIABETES (T2D) AND SMOKING

In this section we review empirical estimates of the contribution to education mortality disparities that could be associated with two of the multiple causal pathways described before. First, we show that a direct pathway from early conditions influences adult risks of obesity and metabolic diseases and can account for a significant fraction of the education mortality gradient. Second, we illustrate the impact of one (out of many) indirect pathways involving early non-cognitive traits related to smoking behavior and estimate the magnitude of mortality disparities associated with it. In both cases, we only focus on key results and leave details of statistical analyses to the online Appendix.

Education disparities in the prevalence of obesity are quite large and vary widely across populations. In the US older adult population (50 and above), about 30% of those with less than HS education are obese versus 19% among those with more than HS (see online Appendix).

These gross disparities in obesity conceal heterogeneity associated with population ancestry. This is due to recent migrant flows from West Africa and North-Central America, in the case of US and Canada, and from West and East Africa as well as South Asia, in the case of some Western European populations. We emphasize this distinction because direct pathways that increase obesity risks differ by population ancestry. In fact, first generation migrants from Asia, Africa or Latin America to North America or Europe are the subpopulations that face the largest contrasts between ancestral and modern environment and are thus at highest risk of manifesting health outcomes predicted by the Predictive Adaptive Response (PAR) conjecture (Gluckman, 2006). These populations are concentrated in the lower social classes and are simultaneously exposed to a whole array of social mobility obstacles. Consequently, it is in these populations that the direct mechanisms described above will operate maximally to produce social class disparities. Instead, the local, native ancestry subpopulations are more likely to avoid PAR and experience instead risks associated with maternal health status, maternal nutrition, and early exposure to familial obesogenic environments. Below, we quantify the relevance of direct mechanisms associated with obesity only among local populations. Consequently, the resulting estimates must be lower bounds of the target quantities.

We use the Health and Retirement Survey (HRS) and estimate the contribution of an indicator of adverse early conditions to observed education mortality differentials associated with adult obesity and T2D (paths 1, 12 and 1, 10, 22 in the model). We use the elderly adult population of the HRS and compute the difference in life expectancy between those with no high school degree (LHS) and those with some college (MHS). We then estimate the impact of an indicator of adverse early conditions on the risk of old adult obesity, separately by education category. In addition, we calculate the relative risk of T2D among the obese and non-obese and, finally, compute the relative risk of mortality associated with T2D. Under some assumptions spelled out in the online Appendix, these computations yield proper estimates of the fraction of differences in life expectancy at age 45 between those with no high school and those with more than high school education that is attributable to the effects of poor early conditions on adult obesity and T2D.

Education mortality disparities in the HRS survey are stark. In the sample followed since 1992, those with LHS experience mortality risks twice as high (2.08) relative to those with MHS. In terms of life expectancy at age 45, E(45), this translates into a disparity of about 4.1 years of residual life, very close to the estimates from independent sources described before and for a similar period of time. The issue of interest to us is this: how much of this difference can be attributed to early conditions? Since this is too broad a question, we narrow it down further and ask: how much of the difference can be attributed to the effects that early exposures have on obesity and of a single chronic illness associated with obesity, namely, T2D?

The sequence of required steps to arrive at suitable estimates is fully described in the online Appendix. Here we only review key results. First, we estimate that those with the poorest values on the indicator of early conditions who were alive in 2006, experience an excess probability of becoming obese before 2017 that is 1.72 times as high relative to those with better values of the same indicator. Because all variables contributing to the indicator of early conditions are set before completing education, it is legitimate to assess its association with educational attainment. It turns out that about 78% of those with LHS are classified in the worst category of the early conditions indicators versus 38% among those with MHS.

Second, according to HRS data, being obese increases the risk of T2D almost twofold. If we conservatively assume that this relative risk does not vary with early conditions (or education)

and, in addition, that the excess mortality risks associated with T2D do not vary with early conditions (or education) and obesity status, we can compute mortality risks by early conditions in each education group. We then convert these relative risks into a metric of life expectancy at age 45, E(45).[6]

Figure 16.2 displays values of life expectancy at age 45, E(45), under three alternative scenarios labeled MINobesity, HRSobesity, and MAXobesity. These scenarios correspond to the initial levels of obesity prevalence in HRS. The scenario HRSobesity utilizes observed values in 2006 whereas MINobesity and MAXobesity reduce (increase) the observed values by 15% respectively. The Y-axis is for values of E(45). The X-axis is for alternative values of the relative risks of obesity associated with adverse early conditions (lower values correspond to higher risks). Because estimates are sensitive to initial levels of adult prevalence in HRS, the figure includes three lines whose points were computed using different assumptions about the age pattern of obesity prevalence in the 2006 HRS. The highest line assumes levels of obesity prevalence 10% lower than observed in 2006 HRS. Those defining the middle line assume a prevalence identical to that observed in HRS. Finally, those in the lower line assume levels of prevalence 10% higher than observed. In addition, the figure includes two horizontal lines. The lower one is drawn at the value of E(45) observed among those with LHS. The highest horizontal line is drawn at the value of E(45) observed among those with MHS. The difference between these two horizontal lines is about 4.1 years, the original estimate of education adult mortality disparity. Points along the three non-horizontal lines represent values of E(45) that would be attained by the LHS group if everything except differential allocation of early conditions were the same across education groups. One way to read the figure is to focus on the difference between any point in any of the three lines and the top horizontal line: this

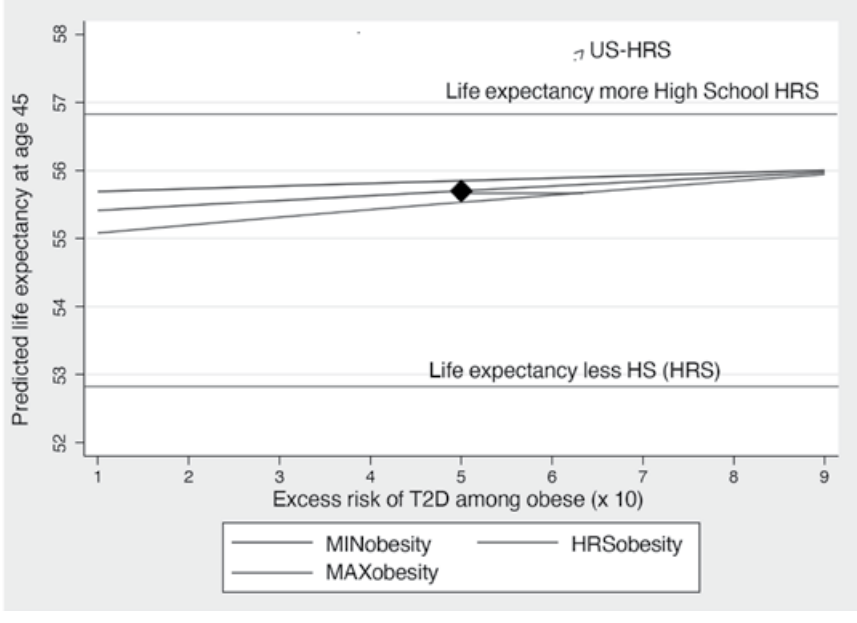


*Figure 16.2* *Predicted life expectancy at age 45, E(45), associated with T2D excess mortality risks (data from US HRS)*

difference is equivalent to the education disparity attributable to effects of early conditions operating via obesity and T2D. In the case when we use the observed prevalence of obesity in HRS (diamond point), this difference is approximately years (56.9 − 55.5) or, equivalently, about 34% (1.4/4.1) of the observed education disparity (difference between the two horizontal lines). This is not a small number. Furthermore, because of the assumptions we made before to simplify computations, the actual contribution of early conditions to adult mortality disparities might be even more sizable.

**Indirect Pathways: Smoking**

This section describes the influence that indirect pathways originating in early conditions may have on sustaining social class disparities. We focus on paths 3, 20, 16 and 3, 20, 18. These are pathways which involve exposure to early conditions that result in preferences and behaviors that influence both educational attainment and health conditions.

Educational contrasts in smoking behavior are even starker than those for obesity. In the US, the fraction of adults 25 and older who are current smokers is of the order of .35 among those with LHS and .16 among those with MHS (Cornelius et al., 2020) or odds of the order of 3. In the UK the figures are .22 and .10 respectively. A 2000 report on smoking in Europe shows that differences among adults older than 20 are of similar order of magnitude though they vary broadly by countries: from odds ratios as high as 2.5 in Norway to values below 1 in Portugal (Cavelaars, 2000).

Past research on the relations between early upbringing, smoking, and mortality has unveiled abundant and robust evidence for two regularities:

i. Smoking and adult mortality: The linkage between smoking and mortality is well established and, despite some remaining uncertainty regarding magnitudes, we know that smoking significantly increases the risk of CHD, stroke, and cancer to several sites (breast and lung and upper respiratory tract), the three most powerful causes of adult mortality in modern populations (CDC, 2021).
ii. Smoking and early upbringing: It is known that smoking is a behavior adopted early, difficult to abandon, and that its effects are felt only after long latency periods. For the most part, tobacco addiction is acquired during early adolescence, is influenced by parental, sibling, and peer smoking, and its initiation and persistence are tightly associated with individuals' future outlooks and time preferences, risk aversion, self-control, and discipline. Some of these factors also contribute to sort out who graduates from high school and who drops out as well as those who are successful in the labor market and those who are not (Simmons-Morton et al., 2001).
iii. Education and early upbringing: Educational attainment is a prime example of a mortality determinant that is highly sensitive to cognitive and non-cognitive traits. The connection between cognitive traits and education is well-established and so is the importance of non-cognitive traits such as industriousness, discipline, and perseverance (Cunha and Heckman, 2007). At least some of these traits are involved in the formation of individual preferences, tastes, and choices that also influence health-related behaviors, and these, in turn, shape the individual exposure–resistance–recovery triplet. Protective health behaviors, such as avoidance of smoking, moderate or no alcohol consumption, healthy diets, and physical activity are more likely to be adopted by individuals who have strong future

outlooks and low discount rates of the future. These same time preferences influence decisions about schooling continuation, training, and acquisition of skills.

From the above, we conjecture that it is possible that at least part of the association between education and adult health and mortality is due to the fact that both educational attainment and adult health status are influenced by time preferences set early in life (paths 3, 20, 17).[7] Individual time preferences are just one in an otherwise much larger bundle of health and education-related cognitive and non-cognitive traits that are partially or totally transferred from parents to offspring. They are all part of the sociocultural contribution to individual phenotypes (paths 2, 3) to which also contribute genes and epigenes (path 4) and non-family environments (Cavalli-Sforza and Feldman, 1981). Just how large can the contribution of indirect pathways be?

To offer an approximate answer to this question, we use the 1958 British Cohort and estimate the contribution a single early behavioral trait to adult health and mortality disparities associated with smoking. We choose a single trait, T, in the offspring generation, namely, having a diagnosis of experiencing behavioral problems or "externalizing behaviors" at ages 6 and 11. Estimates from a model estimated with NCDS data shows that this trait influences both offspring smoking and educational attainment. In addition, T is unequally distributed by offspring social class of origin, being much more prevalent in lower social classes (see online Appendix for details of the model and statistical analysis).

Life expectancy at age 50, E(50), in the UK is of the order of 31.70 and 28.90 years for high and low educated individuals, giving a total disparity of about 2.8 years. The question we pose is as follows: how much can this disparity be reduced if the impact of T on smoking is suppressed? To assign a numeric value to this quantity we use estimates of three parameters: effects of T on education, effects of T on adult smoking, and effects of education on smoking. We then perform simple simulations that combine these parameter values with alternative values of T's prevalence. To increase realism, we also choose a range of values of parameters centered on the point estimated in NCDS data. Combining values of T and of parameter estimates, we perform a total of 50 simulations and choose those that yield values of E(50) for each education group that differ by less than one year relative to those observed in the population. We then use the simulations thus selected and compute counterfactual values of E(50) for the lowest education group that would be observed if either the prevalence of T was set equal in both education groups or if the effects of T on smoking were set to zero (see the online Appendix for details about the model used, parameter estimates and simulations).

Figure 16.3 displays the fraction of reduction of education disparities in E(50) that would be obtained if either the effects of T on smoking were eliminated (triangles) or the prevalence of T were set equal in both education categories ("x"). These two quantities, (y-axis), are plotted against the absolute value (in years) of the difference between E(50) observed and predicted in each simulation that produced the quantities. Preferred simulations yielding errors below 5% are enclosed in a rectangle. These yield counterfactual reductions in the range .05–.15. This means that, under the estimated model and simulated data, one would expect that education disparities in E(50) would be reduced between 5% and 15%. As in the case of obesity studied before, these contributions are not small and suggest that observed education mortality disparities could feasibly be rooted in at least one indirect mechanism.

As before in the illustration with obesity, we are probably erring on the conservative side and downplay the role of indirect mechanism. First, there are determinants of smoking other

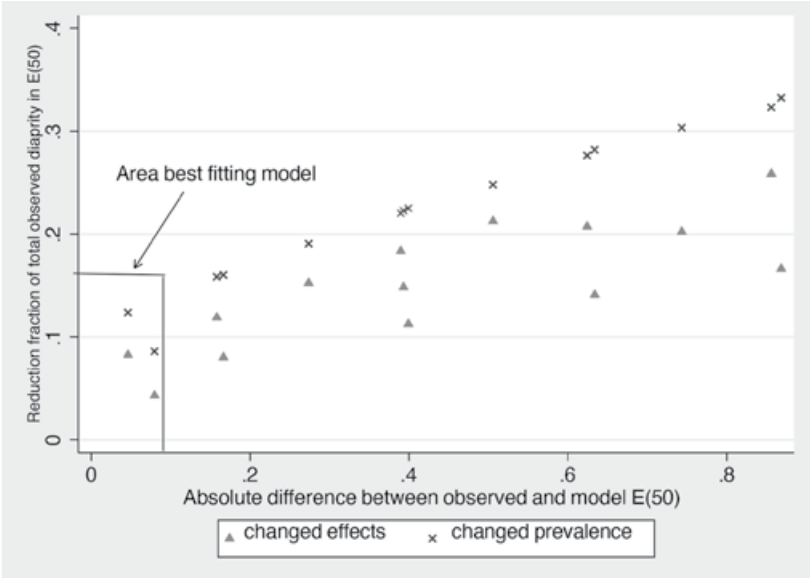


*Figure 16.3 Education differences in life expectancy at age 50, E(50): total and associated with smoking (data from the UK NCDS-1958)*

than early behavior problems and some of them may be sensitive to unequally distributed other early traits. Second, these estimates ignore the contribution of parental effects and heritability, including those associated with maternal smoking during pregnancy and with early family-shared environments. Third, the health impacts of smoking depend on a host of factors, including age of onset, dosage, regularity, and duration. It is unlikely that conditions that influence initiation are the same as those that affect desistance. Nor can smoking behavior be easily decoupled from other health-related behaviors, such as alcohol intake and diet, that have independent additive effects and are influenced by non-overlapping determinants.

## INTERGENERATIONAL TRANSMISSION OF DISPARITIES: DIRECT AND INDIRECT PATHWAYS

Both direct and indirect pathways rooted in early conditions may produce disparities in one generation and can also contribute to their self-replication in subsequent ones. Below we discuss different forms of heritability associated with obesity and metabolic disorders, on one hand, and smoking, on the other.

### Direct Pathways and Intergenerational Transmission

There are three sources of heritability of disparities associated with direct pathways increasing risks of obesity and metabolic disorders. The first source is via sociocultural transmission. In fact, parental and offspring diet and physical activity preferences, the two most important behavioral drivers of obesity, are closely related. To the extent that the phenotype is unequally

distributed by social classes in one generation, it will continue to be so in subsequent ones when paired with imperfect social class mobility.[8]

The second mechanism involves epigenetic changes. There are many animal studies that demonstrate that alteration of the macro and micronutrient composition of the maternal diet can exert powerful influences on the hypo (hyper) methylation and other types of chromatin changes. This, in turn, "programs" fetal body type and risks of metabolic disorders (Bateson and Gluckman, 2011). Although similar results have been obtained in human populations subjected to several types of nutritional constraints and stresses of the maternal diet or the balance of nutrients, the empirical evidence pertains to very selected populations and is difficult to generalize. The involvement of the epigenome is sensitive to deficiencies of some micronutrients that are themselves, or participate in the production of, methyl donors and imbalanced diet composition (e.g. carbohydrates excess). This suggests that inequities in allocation of resources that promote a balanced maternal diet will reinforce conditions to develop obesity and metabolic disorders. In addition, it is possible that the epigenetic marks that trigger obesity and metabolic disorder risk in the fetus, infant, and child can be reliably inherited across at least two generations. If this is the case, replication of disparities is enhanced even if the original source (unequal distribution of nutritional deficiencies) disappears (Patti, 2013; Pembrey, 2010; Pembrey et al., 2006).

### Indirect Pathways and Intergenerational Transmission

The most direct route through which indirect pathways may reproduce inequalities across generations is by increasing the risk of smoking via shared maternal and parental environments. First, although the mechanisms are unclear, there is some evidence that maternal smoking during pregnancy increases risks of addiction in the newborn (Blood-Siegfried and Rende, 2010; Niaura et al., 2001). If this is the case, smokers' offspring are exposed to a double risk: to become smokers as adults and to have diminished cognitive abilities due to in-utero induced damage.

Second, there is a large body of evidence supporting the idea that an individual's likelihood of smoking is higher among those whose parents or siblings smoke. This is a classic form of sociocultural inheritance of a health behavior. To the extent that smoking behavior is unequally distributed across classes, this route alone could induce a replication of disparities across generations in stratification regimes that have restricted social mobility. There is, however, another gateway which reinforces replication. This is that membership in social classes in which smoking is more prevalent may carry an additional risk associated with the transmission of non-cognitive factors, such as poor outlooks and heavy discount of the future, that reduce the opportunities for the acquisition of skills that promote social class mobility even in stratification systems with high mobility.[9]

## CONCLUSION AND DISCUSSION

We propose a framework that accounts for systemic and growing social class health and mortality disparities in modern populations. Within the proposed framework we elaborate a theoretical model that emphasizes direct and indirect pathways rooted in early exposures. The model has room to account for differential sensitivity of observed disparities to secular

shifts or moderate alterations of the epidemiological and stratification regimes in a population. Using the example of obesity (metabolic and cardiovascular illnesses) and smoking (cancers and cardiovascular illnesses), we showed that attention to the direct and indirect pathways as well as to their power of replication across generations, can augment our ability to account for current modern disparities and past trends. Although we do not show directly how the framework would strongly support actual empirical research, the examples of obesity and smoking leave enough clues about how to do so.

The chapter has several shortcomings. First, as is done in most of this literature, we ignore the importance of separating the three components that contribute to health status, namely the triplet ERRe. Doing so may simplify the descriptive task in a chapter like this, but it is not best practice in a research agenda on the subject. Second, we did not discuss in much detail the role of direct, causal, impacts of social class. This is because these have been thoroughly examined in most of this literature and we wished to highlight pathways that have been less addressed despite the fact that their importance may have increased over time. Third, we did not consider the potential influences that policy interventions, either local or national, may have and under what conditions they may do so. If, for example, the bulk of health and mortality disparities by education were traceable to differential obesity, would a policy intervention targeting inequities in access to modern medical care to deal with T2D have durable effects? And how large could these be? Would a policy targeting instead maternal nutrition and BMI be more effective in the long run? The model must be adapted to ensure that, just as it makes explicit that the power of some pathways is a function of epidemiological and stratification regimes, the reach of some of these interventions may be equally dependent on the power of those pathways. Finally, illustrations of the efficiency of direct and indirect pathways are based on only two data sets from two quite singular populations. Because of this, strong generalization of our findings is inappropriate but we believe they do serve to prove the point that these mechanisms can, under some conditions, be influential.

## NOTES

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2. https://www.e-elgar.com/textbooks/hoffmann
3. Alcohol intake plays a less important role but is also included as part of the array of behaviors that make up modern "life styles".
4. This is a simple graphical causal model, a directed acyclic graph (DAG), and is meant to be a heuristic tool, not a detailed causal model.
5. Perhaps the most influential theoretical work in this area is by Grossman (1972, 2000). More recent contributions are the work by Conti et al. (2010a, b), Hoffmann et al. (2018, 2019), Mackenbach (2012), Palloni (2006) and Palloni et al. (2009).
6. It bears repeating that the assumptions just made about independence between conditional risks of T2D and adult mortality among diabetics, on the one hand, and early conditions and education, on

the other, are violated in the HRS data. As a result of this violation, we will estimate lower bounds of the contribution of early conditions to disparities in adult mortality.

7. The mechanism is likely to be more complicated than this. Thus, some empirical evidence suggests that time preferences are themselves influenced by educational attainment (Becker and Mulligan, 1997; Oreopoulos and Salvanes, 2011; Perez-Arce, 2017).
8. It is important to note that food preferences at least have been shown to be influenced in-utero and involve changes in the fetal epigenome (Bateson and Gluckman, 2011; Bonduriansky and Day, 2018). This reinforces the sociocultural mechanism.
9. There is, of course, a final source for the replication of inequalities associated with indirect pathways and smoking, the genetic route. There is some empirical evidence of alleles that increase the risks of nicotine addiction. If these also have pleiotropic effects and influence levels of cognitive and non-cognitive capacities, a genetic source for the correlation between health status and social class would be established. We know of no direct empirical evidence suggesting the existence of such allelic variants.

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