Income Mobility, Mortality and Health in the US

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Chapter 1

The Problem: Health Disparities and Stratification

Socioeconomic inequality in health and mortality is one of the persistent regularities observed across societies since at least the turn of the 20th century. All countries, even those with high economic prosperity and human development, show systematic inequalities in mortality and morbidity. These inequalities are not small. They ranges from 5 to 10 years of life expectancy at birth, and from 10 to 20 years of disability-free life expectancy (Commission on Social Determinants of Health 2008). In a recent paper, Chetty et al. (2016) used administrative data to show that the gap in life expectancy between the 1% richest and poorest 1% of individuals in the U.S. was about 14 years. These disparities persist across multiple measures of individual social and economic status (e.g., education, income, wealth, occupation) and there is evidence they have recently widened in high-income countries (Mackenbach 2012).

These social disparities in health are usually explained by the characteristics of the social stratification system. The key argument behind the theory of *fundamental causes* of health disparities (Link and Phelan 1995; Phelan et al. 2010) is that inequality in health is caused ultimately by social stratification processes, not the exposure to proximal risk factors (e.g., smoking). Individual socioeconomic status would provide *flexible resources* (e.g., power, prestige, money, social connections) that can be used to avoid or minimize the consequences of diseases. These resources are contingent, changing and continuing to exist, reproducing the association between socioeconomic status and health over time. To explain health disparities, thus, is necessary to identify features of the stratification system that influence health status. According to Grusky (2014), for instance, three main elements define any stratification system: (1) mobility mechanisms that *sort* individuals into social strata, (2) allocation rules that distribute resources to social strata, and (3) social processes that provide more value to some resources than other. Correspondingly, health inequalities might be understood as a function of (1) social mobility and the differences of people across social strata, (2) resource distribution or differences in access to material and non-material

resources across social strata, and (3) the value of those resources to avoid health problems (Mackenbach 2012). All these processes and mechanisms might vary over time and between geographic areas, giving rise to differences in the magnitude of health inequalities across regions, nations, and periods. The first mechanism of stratification (i.e., sorting) involves processes such as residential segregation or health selection. Individuals in poor health early in life, for example, might be less likely to experience upward mobility and more likely to experience both downward mobility and have poor health.[1] The remaining two mechanisms of stratification are related to the effect of allocation of resources (e.g., income) on health and its interaction with characteristics of the prevailing epidemiological regime.

Mechanisms that produce health disparities, however, are not limited to sorting, differential resource abundance, and heterogeneity of resource productivity. It has also been argued that factors such as the distribution of income in the population (i.e., income inequality), the relative position of individuals according to income, and income mobility might have a *contextual* impact on individual health and mortality. In the next section, I briefly discuss the literature on income inequality and health. Although this project is about the link between social mobility and health, it is worth reviewing the mechanisms behind the relationship between income inequality and health to gain conceptual clarity and establish common ground with that rich research tradition. I focus mainly on theoretical and conceptual issues rather than methodological problems. Then, I link that discussion to the main topic of this dissertation, income mobility and health.

1.1 Income Inequality and Health

Income inequality is, by definition, an ecological variable (i.e., aggregate) that describes the scale of income differences across a population. The *income inequality hypothesis* states that a *contextual* factor such as income inequality affects individual's health. The link between income inequality and health, however, could be generated by different mechanisms or processes. Evans et al. (2004) provide a useful typology that summarizes why one might observe a correlation between income inequality and health:

1. *Mechanical* effects. If individual economic status is associated with health, then an increase in economic inequality will lead to increase in inequality in health. This effect, however, is not due to material factors only. *Scarcity* may impose a cognitive *bandwidth tax* that interferes with decision making and long-term planning, increasing the adoption of risky behaviors (Mullainathan and Shafir 2013).

2. *Relational* or slope effects alter the magnitude of the relationship between economic status and health, and change the relationship between income inequality and health even when

[1] The only requirement for health selection is that at some point in the life course of individuals' resources depends on previous health status (Palloni et al. 2009). There are good reasons to expect selection processes. Leigh et al. (2009) note that poor health can have serious consequences on educational outcomes, employment, and marriage opportunities. It should not be surprising that reproduction of income inequality occurs, at least in part, through an attainment process: poor health of low-income children translates into lower levels of educational attainment and, as a result, lower earnings during adulthood.

the distribution of income remains constant. These effects can be entirely exogenous (e.g., improving access to health care) and attenuate the link between income and diseases.

3. *Functional form* effects are related to the shape of the relationship between economic status and health. For instance, if the relationship is concave, the average health in society will improve as the average income increases and inequality of income decreases.

4. Finally, Evans et al. (2004) suggest the importance of *externality* effects. These effects occur when a contextual condition triggers individual processes that are detrimental to health. For instance, living in a social context of high inequality might intensify the feeling of relative deprivation among low-income persons, leading to higher levels of violent crime, even if individual economic status has no relationship to the outcome of interest. Thus, changes in other people's income can modify customs, ideals, politics, and the behavior of those whose own income remains unchanged.[2]

In order to formalize the relations conjectured by the *income inequality hypothesis*, I use the notation proposed by Wagstaff and van Doorslaer (2000). At the individual level, we can express the *income inequality hypothesis* as:

$$h_i = f_i(y_i, I_c) \tag{1.1}$$

where h_i is the health of individual i, y_i is individual's income, I_c is a measure of income inequality at the community level[3], and f_i is a function through which inputs are transformed into health. This formula represents independent effects of the terms included in the (unspecified) functional form f_i. It is possible to define a case where it is not the community's inequality that matters but the national level of inequality (let's say p), in which case we replace I_c by I_p (see, for instance, Zheng 2012).

At the community level, the expression is:

$$h_c = f_c(y_c, I_c) \tag{1.2}$$

where c is an index for the level of aggregation (e.g., census tract, county, state). Again, I_c can be replaced with I_p (income inequality at the national level or a larger geographic unit). Using this aggregate model, however, it would be difficult to distinguish between some of the effects discussed by Evans et al. (2004). Aggregate analyses, nevertheless, should not be discarded by invoking the

[2]Differences in health between two communities might be due to characteristics of the individuals living in those communities (*composition effect* and sorting), exposure to local factors that affects individuals' health (e.g., smoke from factory, community resources), or social influence (individuals who live in proximity are more likely to assimilate the same type of behaviors, Christakis and Fowler 2007). It is likely that these three processes are simultaneously affecting health outcomes.

[3]Community can refer to a county, state, a region, or any area smaller than a county (e.g., Census tract).

ecological fallacy or other inference difficulties.[4] These analyses can provide clues on how contextual effects observed at the individual level aggregate at the community and population level. Given the complexity of the individual and contextual effects of stratification processes on health, aggregation might not be linear and straightforward.[5] Pickett and Wilkinson (2015), for instance, note that effects of income inequality are stronger in large areas because in that context income inequality serves as a measure of the scale of social stratification. Income inequality in small areas is affected by the degree of residential segregation. Higher segregation would increase the inequality between areas, but also increase homogeneity within them (Chen and Gotway Crawford 2012).

It has been argued that income inequality affects the social fabric, reducing both social capital and mutual trust, and that the resulting stresses and lack of public investments harm the health of both wealthy and poor (although one group may be more affected than the other, Wilkinson 1992; Kawachi et al. 1997). Income inequality increases social distances, accentuating social class or status differences, and strengthening causal processes whereby social class marks individuals throughout life. Differences produce negative emotions, such as shame and distrust, that could translate into poorer health by psycho-neuro-endocrine mechanisms and induce behaviors such as smoking (Wilkinson and Pickett 2009). In addition, social comparison to higher-ranking friends and neighbors might cause stress and ill-health among people who are poorer than their reference group (Wilkinson 1992; Marmot 2004). The so-called *relative deprivation hypothesis* assumes that negative effects outweigh the benefits of having richer neighbors who may pay for better neighborhood amenities. Another explanation suggests that the diffusion of innovations (knowledge or technology) among the most disadvantaged groups is slower when income inequality is high, increasing health disparities by improving the health of the wealthy when these innovations begin and benefiting the poorer only after long lags (Phelan et al. 2010).

Another approach to explain the link between income inequality and health is a neo-material reinterpretation proposed by Lynch et al. (2004). According to these authors, structural causes of inequalities are the root of health disparities. Income inequality would be only a manifestation of a cluster of *neo-material* conditions that affect population health (Lynch et al. 2004). Thus, the association between income inequality and health would be contingent and reflect people's lack of resources, both at the individual and community level. This interpretation is an explicit recognition that the social, political and economic processes that generate income inequality influence personal resources and also have an impact on public resources such as schooling, health care, social welfare, and working conditions (Lynch et al. 2004).[6] Thus, according to this perspective, one would have to account for material factors at the community level to estimate the independent effect of inequality on health.

[4]Trends at multiple levels of analysis may or may not be similar and their causes may or may not be the same (Snijders et al. 2011).

[5]Health often reflects cumulative exposures over a long period of time. Thus, health at adult ages can be partly determined by exposure to biological and social factors early in life so that the roots of health inequalities may well lie in inequalities experienced back then.

[6]Gilens (2012) provides a political interpretation of these processes and notes that income inequality increases the political influence of the wealthy. If their policy preferences restrict public goods that provide health benefits (e.g., education, sanitation, policing), the health of the poor may be harmed.

1.2 Income Mobility and Health

Can these hypotheses be extended to income mobility? If so, what consequences and patterns should be expected? Unlike income inequality, mobility can be measured both at the individual level (inter and intragenerational socioeconomic changes over the life course) and aggregate level (earnings elasticity, rank-rank slope).[7] Thus, unlike income inequality, income mobility is not always an ecological or contextual variable. There is a large body of research on individual mobility on health and mortality. The work by Sorokin (1959) and the *mobility effects* literature, for example, suggests at least three individual pathways through which social mobility might affect health at the individual level. These mechanisms are associated with the *dissociative*, *falling from grace*, and *acculturation* hypotheses.

The *dissociative hypothesis* holds that mobility will have a negative causal effect on well-being, psychological distress, and health. Any changes in social class (up or down) could create chronic strain and generate a permanent negative impact on mental health and well-being. The *falling from grace* hypothesis predicts that *only* downward intragenerational class mobility is detrimental to health, specially in social and historical contexts that foster high social expectations for economic success (**?**). Downward mobility often entails an involuntary loss of an achieved status, and signals a failure to live up to social expectations of individual success. Because downward mobility is more likely to be negative and involuntary, it should have negative consequences for health, independently of the reduction of material conditions. Finally, the *acculturation* hypothesis (Blau 1956) argues that mobility is neither a cause nor consequence of health, but that socially mobile individuals come to experience similar levels of health as those who share their social class of destination. Under this hypothesis, social mobility would be unrelated to distress and health (after controlling for income or social class).

The focus of this dissertation, however, is *not* on the health consequences of individual experiences of socioeconomic mobility but on the *contextual* impact of a place's income mobility on the health of individuals who experience such mobility regime. As in the case of income inequality, one can think on income mobility as a contextual factor that causally affects health and health disparities. Recent evidence suggests that social mobility has a positive relationship with health and mortality eve n after adjusting for income at the community and individual level, and income inequality (**?**Venkataramani et al. 2015, 2016). That is, at a given level of inequality, people living in communities with a more flexible stratification regime have lower mortality and better health. Theoretically, it is possible to think on societies with different levels of inequality and social mobility provided that both characteristics of the stratification regime, although related, are produced by different mechanisms. To empirically disentangle the (independent) effect of income inequality and mobility, however, the relationship between both aspects of the stratification system should be moderate (or not too strong), so that we can observe communities being simultaneously characterized by unequal income distribution and flexible mobility regimes, and vice-versa. Evidence on the *Great Gatsby Curve*, according to which countries with higher income inequality score low in social mobility, is mixed and sensitive to the mobility measures used (Winship 2015; **?**). However, the relationship between income inequality and opportunity, even when considering the largest

[7] See Torche (2015) for a review of mobility measures.

estimates, is far from perfect, providing empirical grounds to estimate the independent effect of income mobility and inequality.

1.2.1 Mechanisms

What mechanisms would explain the link between health and mortality and income mobility at the community level? Communities with high-income mobility may share properties that help individuals and families manage resources available to them, improve resilience to confront adverse conditions and, ultimately, reduce individual exposure to health risks irrespective of a community's income levels and aggregate income inequality.[8] For instance, credible prospects for upward income mobility might encourage people to invest more in their health (i.e., higher expected returns to health investments, Venkataramani et al. 2015). Also, optimism about the quality of one's future may raise the desire to achieve good health, independently of the possibility of obtaining greater income by doing so (Venkataramani et al. 2015). A pessimistic interpretation of this mechanism holds that a rigid stratification system fosters individual hopelessness, weakens aspirations and, more generally, diminishes the value of adoption of attitudes and behaviors that promote good health (**?**). According to this explanation, not only actual scarcity might impose a cognitive *bandwidth tax* as Mullainathan and Shafir (2013) note, but expectations and outlooks of economic success could trigger the adoption of unhealthy behaviors through changes in decision making and long-term planning (e.g., time discounting).[9] Both positive and negative interpretations are not necessarily symmetric and generate different predictions. The increase of expected returns of health investments would suggest that high mobility regimes generate benefits for everybody (albeit with decreasing returns, see Grossman 1972), whereas hopelessness implies that the extent of mobility should only produce benefits for individuals at the lower end of the income hierarchy.

These lines of argument borrow from Grossman's model on health capital (Grossman 1972; **?**). In his seminal paper, *On the Concept of Health Capital and the Demand for Health*, Grossman defines health as a capital and investment good that increases the number of healthy days to work and earn income. Thus, how people allocate resources between health capital and other goods/services would depend on the prices of health care, wages and their productivity in the production of health. For instance, increasing wage rates (shifting the marginal efficiency of investment) would rise the returns from healthy days and increase the optimal health stock. The final result of an increase of income, however, might be ambiguous because of the trade-off between higher returns of health (healthy days are more valuable) and a higher opportunity costs associated with the production of health (time spent exercising). The level of education, on the other hand, is assumed to improve the efficiency to produce health investments (better knowledge of harmful effects of smoking, ability to follow medical instructions). Thus, having low expectations about socioeconomic prospects would discourage health investments (lower return of healthy days), or decrease investments in

[8] Income mobility could also be just a proxy for a host of community traits which could impact health.

[9] Time discounting characterizes how individuals' preference for a reward decreases with the delay to its receipt. In general, most people tend to prefer smaller, immediate rewards to larger ones available after a delay. It is also important to differentiate *time discounting* from *time preference*. The latter describes whether and how people consider events in the past, present and future when making decisions, while time discounting captures the degree to which people devalue rewards with every additional unit of delay (**?**).

education with subsequent reductions of health production efficiency.

These conditions also imply inter-generational effects. The perception of opportunities in a community may influence how parents socialize children and favor (discourage) the adoption of positive outlooks and the value of skill acquisition that eventually affect health status (**?**). Thus, early upbringing and socialization, the formation of skills associated with significant returns to extra years of healthy living, and the adoption of attitudes and behaviors that lower exposure to health risks and strengthen resiliency, would impact health throughout the life course. If early cognitive and non-cognitive skills matter for labor market success, income and wages, they may also matter for adult health not only because socioeconomic success breeds good health but because the traits responsible for success also generate adherence to low-risk behaviors.[10]

To formalize these ideas, one can write an expression using the notation proposed by Wagstaff and van Doorslaer (2000):

$$h_i = f_i(y_i, m_i, M_c, I_c) \tag{1.3}$$

where m_i correspondents to a measure of individual (intergenerational) mobility, M_c a measure of income mobility at the community level, y_i the individual income, and I_c inequality at the community level. As in the case of income inequality one can distinguish processes affecting *directly* individual behavior and delayed effects due to early socialization and intergenerational transmission.

The relationship between m_i and M_c deserves a comment. The hypothesized individual *mobility effects* tend to go in the opposite direction than those expected of contextual effects: while individual mobility could increase instability and stress, contextual mobility would improve expectations and outlooks. It is not hard to imagine that contextual mobility might also increase insecurity and stress, particularly among those at the top of the income distribution. However, those better off are able to protect themselves against the anticipated threat of falling in the economic ladder, and the stress to which they might be exposed can be reduced or offset by the benefits of being in a relatively high status. On the other hand, a contextual effect of social mobility through changes in *perceptions* of the mobility regime, time discounting, and eventually the adoption of behaviors, might be influenced by experiences of individual mobility.

It is also plausible to expect that *intragenerational mobility* has a similar impact on decision making and long-term planning as *intergenerational mobility*. Living in areas with more intergenerational mobility implies a higher chance of experiencing intragenerational mobility. This brings up the question of how persons define their perceptions about the mobility regime and how resistant these perceptions are over time. Hence, when the key factor being investigated is the *perception of the mobility regime*, the distinction between contextual and individual effects, intra and inter mobility, becomes fuzzy. In addition, we should expect lagged effects because it can take time to

[10]This also opens the possibility that health affects income mobility (feedback): the deterioration of health in a community might stiffen the stratification regime due to health selection processes discussed before. I will examine this question in Section xx.

align individuals' perceptions with actual changes in the stratification regime.

The link between M_c and I_c also deserves a comment. First, the contextual conditions generated by income inequality might be a direct cause of income rigidity. Nepotistic relations, for instance, are stronger in communities with high-income inequality and can inhibit social mobility (**?**). In this case, social mobility would be a mediator of the link between inequality and heath. Under this assumption, the estimation of the independent contextual effect of mobility requires adjusting for income inequality to avoid confounding. The reverse is also possible: changes in income mobility affect individuals' income, and by extension, the income distribution.

Let us imagine a counterfactual scenario where there are several communities with a given income inequality (measured by the Gini coefficient or any other statistic to characterize the income distribution). Those communities would also have a steady state income mobility. Suppose there is an intervention that improves access quality quality among those with fewer resources. We would expect that, in the long run, those changes would affect communities' income and its distribution. Changes in income would improve health, changes in the income distribution should also improve health (income inequality hypothesis). Perceptions of the mobility regime, in turn, will take some time to update. Once they are in line with actual socioeconomic opportunities both due to individual experiences of mobility or those of significant others (i.e., family, friends, neighbors), decision making and long-term planning will reduce the adoption of unhealthy behaviors. Contagion and social influence might also multiply the consequences of these processes (Christakis and Fowler 2007). Suppose we compare these communities with others that are identical but where the intervention did not take place. In this case, we can estimate the total *causal* effect of social mobility on health. By adjusting for income, we estimate the part of the effect that is not due to income (functional specification issues aside). In this *ideal* scenario, adjusting additionally for income inequality would imply *controlling* for an intermediate factor (i.e., post-treatment bias). The residual difference would represent the remaining effect of income mobility that was not due to changes in income levels and income inequality, but to *individual and/or contextual* effects of income mobility, attributable, at least in part, to the behavioral mechanisms described above. This simple example allows comparing an ideal setup with the actual models and data I would use.

1.2.2 General Hypotheses

To finish this section, I outline some conjectures that can be extracted from the previous discussion on income mobility and health:

1. A positive relationship between community income mobility and health/mortality at the individual and aggregate is expected, after adjusting for individual income, community income, income inequality and other individual and community confounders.[11]
2. Income mobility should be related to risk behaviors such as smoking and diet habits at the individual and aggregate level, after adjusting for individual income, community income, income inequality and other individual and community confounders.

[11] It is also important to avoid post-treatment bias and overfitting.

3. Benign consequences of the flexibility of a stratification regime on health should be higher among those at the bottom of the income distribution, and lower for those at the top. Those at the bottom have more to gain from a highly mobile environment than those at the top (i.e., expectations of economic success should have a higher impact on the investment in skills necessary to improve socioeconomic status). Conversely, the risk of downward mobility might have potentially harmful effects (e.g., stress), especially for those better off, although those negative consequences can be reduced or offset by the benefits of being in a relatively high status.

4. Income mobility should buffer the negative consequences of income inequality and relative deprivation. Thus, we can expect that differences in income mobility would be more consequential under highly unequal income distributions than in more equitable communities (i.e., income mobility as moderator of income inequality). This would depend, however, on the nature of income inequality and social mobility regimes. In some cases, inequality arises from the growth of the share of income to the very top of the income distribution (as in the case of the U.S.), but in other cases, inequality is due to increases in the population size at the lower income ranks. Social mobility might also occur at different levels of the income distribution.[12] In this context, exploring the variability of the relationship between income mobility and health outcomes across geographical areas becomes relevant.

5. At the aggregate level, we should expect a negative relationship between income social mobility and health disparities. As in the case of inequality and health, there is no theoretical reason to focus only on average health changes by social mobility and disregard changes in health disparities or variance (see, for instance, Neckerman and Torche 2007 and Truesdale and Jencks 2016).

6. Given the theoretical mechanisms proposed for the contextual effect of social mobility, one might expect a consistent relationship between income mobility and characteristics such as hopelessness or positivism (expectations and outlooks), after adjusting for family background variables, income, income inequality, and other confounders.

[12] Unless one is able to formally define and empirically identify *some* of the theoretically relevant configurations, we cannot aspire to formulate precise predictions, models or estimation. This is the motivation behind the idea of formulating a generative theoretical model of the impact of income mobility on health.

Chapter 2

Income Mobility, Income Inequality and Mortality in the U.S.

Over the last ten years, there has been a steady increase of empirical evidence documenting large gaps in life expectancy at birth by geography in the U.S. (Murray et al. 2006; Ezzati et al. 2008). However, inequalities by geography (state, counties) are not accounted for by differences in access to medical care, places' infrastructure or community characteristics, ethnic composition or, surprisingly, places' income (National Academy of Sciences 2015). This is remarkable in view of the fact that recent research shows that there are massive contrasts in adult mortality by income across U.S. counties. In fact, the best performing counties in the U.S. have levels of life expectancy that are about *20 years larger than the poorest performer*. Moreover, adult disparities seem to be expanding over time as the difference in life expectancy at age 40 between the richest and poorest quartiles of the income distribution of U.S. counties grew from 9 years to about 11 among men and from 5.2 years to 6.6 years among women (Chetty et al. 2016). These gaps are non-trivial and represent 25% of life expectancy at age 40 among men and 13% among women. Based on this evidence, it would be reasonable to expect that most, if not all, U.S. geographic disparities vanish after accounting for place's income. But that's not the case. Factors other than income seem to matter as much or more.

Steady or growing disparities in longevity by geography and by markers such as education and income present a unique challenge. They are at odds with expectations about the role and influence of modern medicine and health care as well as with universally accepted norms of fairness. This may explain the large amount of research dedicated to find the root causes of these disparities and to translate such knowledge into interventions directed at reducing them. An important part of this effort has been allocated to understanding the role of a place's income inequality. A large body of literature documents the existence of a positive association between levels of aggregate income inequality and poor health and mortality, particularly among individuals in the upper and lower part of the income distribution for countries, small areas, and individuals (Pickett and Wilkinson

2015; Subramanian and Kawachi 2004; Wagstaff and van Doorslaer 2000; **?**; **?**; Kawachi et al. 1997; Wilkinson 1992; **?**; Wilkinson and Pickett 2009). Emphasis on the potential role of income inequality has been buttressed by recent evidence documenting a steady increase in the U.S. income inequality (**?**), a fact that makes plausible the idea that recent increases in mortality disparities by geography could indeed be only partially rooted in income inequality.

In this paper, we suggest that an understudied factor, *income mobility*, could also play a significant role. We argue that communities with low income mobility may host conditions that diminish opportunities for individuals' advancement and lifetime achievement, discourage forward looking strategies and careful planning, and weaken individuals' motivation to adhere to behaviors that minimize accumulation of exposure to health risks and could contribute to excess mortality across a broad spectrum of ages and causes of death. Although both income inequality and income mobility are aggregate dimensions of a stratification system, they are quite distinct and should have different implications and impacts. Individuals in communities characterized by comparable levels of income and income inequality but faced with opposite lifetime income mobility prospects may be exposed during formative years to different learning experiences, preferences, and behavioral strategies that ultimately shape health behaviors and lifelong exposure to health risks.

While the association between income inequality and health has been studied as part of a 20-year old literature, recent work suggests that its contribution to the explanation of disparities in longevity may be quite small (Murray et al. 2006; Ezzati et al. 2008). In contrast, the health consequences of income mobility have been rarely studied, a surprising fact in light of growing empirical evidence of a long-term decline in intergenerational social and income mobility in the U.S. among the birth cohorts currently experiencing increased mortality disparities (**?**Hout et al. 1993; Hout 1988, 1984). This research landscape is changing and in a series of very recent papers, a group of researchers began exploring the association between a place's income mobility and health behaviors, self-reported health and mortality (Venkataramani et al. 2015, 2016; **?**).

The goal of this paper is to extend this emerging area of study. First, we propose potential pathways through which income mobility may influence individuals' health and mortality. These pathways are distinct from, albeit related to, those associated with income and income inequality, operate independently of these, greatly overlap with pathways that enhance adult labor market success, and could potentially have powerful impacts on health and mortality disparities across socioeconomic and race groups. Second, we examine selected and well-defined hypotheses about the association between income mobility and adult mortality using county-level data from the U.S. Third, we compare the magnitude of associations between income mobility and mortality and income inequality and mortality. Finally, we estimate age, race/ethnic, and cause-of-death specific patterns of these associations, and compute potential losses/gains of years of adult life resulting from shifts in aggregate income mobility to assess how consistent they are with our hypotheses.

Our results suggest that places with higher levels of income mobility also experience lower adult mortality risks and that these impacts are larger than those attributable to a place's income inequality. The age pattern of effects contains a peak in young to middle adulthood and becomes attenuated at very old ages. The association is similar for males and females but stronger in the African American population. Finally, we find that the excess mortality associated with lower in-

come mobility is largely a result of the influence of communicable diseases, accidents and injuries.

2.1 The relation between place's income mobility and mortality

In this section, we briefly discuss potential causal mechanisms that could generate an association between place's income mobility and health. First, we need to define precisely what we are not doing. We examine the relation between place's income mobility and health/mortality, that is, the linkage between a contextual characteristic (place's income mobility) and an individual (aggregate) trait such as health and mortality.[1]. That is, we are not assessing the relation between individuals' lifetime income mobility experiences and their adult mortality – a problem studied in a large and distinguished body of research (Chandola et al. 2003; Illsley 1955; Fox et al. 1982; Blane et al. 1999, 1993)[2]. What we attempt here is demonstrate that there is an association between an *aggregate* property of the stratification system, on one hand, and individual experiences, on the other. It is, of course, possible that individuals' experiences of occupation or SES mobility are also influenced by the prevailing aggregate regime of income mobility. Indeed, these experiences may be one of many other pathways through which aggregate income mobility and individual mortality are related. However, in this paper, we are interested in the *total* effects of places' income mobility on individual health and mortality and are not concerned with the precise empirical identification of mediating pathways.

We argue that an association between places' income mobility and mortality could exist if communities with higher income mobility host social and economic environments that reduce mortality risks relative to communities with lower income mobility, *independently of the income level and income inequality*. Individuals and groups who occupy the most vulnerable and exposed social positions within unequal communities may be comparatively better off when they face advantageous income mobility prospects than when they do not. Just as individuals who command lower incomes in communities with more equitable income distributions may experience better health than individuals with similar incomes in societies with higher income inequality, so too could individuals and groups that occupy lower ranked positions in societies with higher income mobility enjoy better health than counterparts in societies with more rigid stratification systems.

In theory, communities could be classified in different combinations of income mobility and inequality, where each combination is characterized by a health and mortality profile. Indeed, in our data we actually observe communities (counties) simultaneously characterized by unequal income distribution and flexible mobility regimes or by generous income distributions and high levels of social rigidity.[3] The standard conjecture is that indicators of mortality and health will be

[1]Throughout the paper we use the term *effects* to refer to the magnitude and sign of standardized or non-standardized regression coefficients measuring the strength of the association between two variables and do not presume the existence of a proven causal relationship.

[2]The bulk of this literature is concerned with the long run impact of early occupational (career) shifts or the short run effects of late occupational (career) shifts. This work is based on empirical research that focuses on patterns of relationships between individuals occupation (or SES status broadly conceived) at an early point in their adult life and subsequent older adult health and mortality.

[3]Figure S2 in the *Methodological Supplement* shows a heat-map of the number of counties by income inequality

more beneficial in communities with less inequality than in those with high inequality. The new conjecture is that at a given level of income inequality, better health and mortality conditions will be experienced by members of communities with higher income mobility.

We propose four pathways that might produce a link between income mobility and mortality:

1. *Residential mobility, adult health and mortality*: As stated above, an association between aggregate income mobility and individual health and mortality may be the outcome of a composition effect, namely, places with higher income mobility contain a population composition biased toward individuals who experience mobility (and their health and mortality consequences). In this case, the association between the aggregate property of the stratification system and individual experiences of health and mortality reflects the influence of individual residential mobility patterns (and associated selection processes).

2. *Individual early experiences*: A large body of literature on health and mortality disparities demonstrates that SES (income, education) health and mortality gradients are pervasive, persistent and, as of recent, increasing everywhere in high-income countries (**??**). Further, there is evidence that early conditions and upbringing of individuals matter greatly for adult health and mortality disparities (Palloni et al. 2009; **?**). Thus, some of the health differentials between men in low and high ranking positions initially attributable to chronic stress among those in subordinate positions (**??**) may be rooted in antecedent health conditions sculpted early in life (**?**). If early conditions are influential for SES health and mortality disparities, they may also be influential as vehicles that establish relations between income inequality, income mobility, and adult health and mortality.

   During early stages of socialization individuals experience sensitive and critical windows for the acquisition of cognitive and non-cognitive abilities that are the foundation of skills acquired later in life (**????**). Some of these traits involve the development of outlooks and attitudes that influence investments in skill acquisition and health, including propensities to adhere health-related behaviors. Thus, behaviors critically associated with modern chronic illnesses, such as smoking uptake and desistance, alcohol consumption, substance abuse, choices of diet and physical activity, are in part determined by capabilities sculpted early in life. Early avoidance of unhealthy behaviors has large payoffs in adulthood because these behaviors are closely related and reinforce each other, the physiological and psychological damage they produce are accumulated over time, and they all are strongly non-reversible. Early adoption of healthy behaviors is facilitated by socialization that emphasizes robust future outlooks, self-confidence, and self-reliance, beliefs in the neutrality and fairness of social reward allocation systems, hopefulness and optimism, and incentives to succeed. These are all traits that reduce time discounting so that the addition of one year of healthy life in the future of an individual is endowed with significant rewards and returns (Grossman 2000, 1972).

   We know from empirical research that negative affect, chronic stress, subordination, bleak future outlooks associated with poverty lead to increases in time discounting (**?**). Higher

and mobility deciles. As can be seen, the correlation between income mobility is only moderate and most of deciles have have a have countie

time preferences favors resistance to the adoption of behaviors that may yield immediate rewards but are health-damaging and discourage those that have a more distant and elusive pay-off but are health-preserving (**??**).

This mechanism alludes to influences that shape environments during critical stages of individuals' upbringing. The conjecture is that a places' income mobility regime is powerful enough to shape those environments. But so is an individual's ancestral income mobility experiences, particularly parental and possibly grand parental mobility. Strictly speaking these are two very different mechanisms that can be properly identified only if we simultaneously observe both the influences of a place's aggregate income mobility and individuals familial income mobility experiences.

3. *Community endowments*: Communities with low-income mobility may distort opportunities and incentives, reinforce unequal allocation of favorable traits, undervalue public institutions that contribute to the formation of skills with a high wage premium and, many of them, support non-meritocratic reward allocation strategies. These community properties directly influence the suite of opportunities available to individuals and shapes the way parents socialize children and favor (discourage) the adoption of positive outlooks and the value of skill acquisition. Rigid or weak income mobility fosters individual hopelessness, despair, mistrust, disbelief in a level playing field for all, weaken aspirations and, more generally, diminish the value of adoption of attitudes and behaviors that promote good health.

2.1.1 Hypotheses

Based on the mechanisms discussed above, we define five testable hypotheses about the aggregate association between income mobility and mortality using county-level data. The hypotheses discussed below assume that the individual mechanisms described above can be identified using county-level data or, alternatively, that aggregate patterns might reflect a subset of them. That is a strong assumption as inferences at the county level cannot necessarily be translated to individuals (i.e., ecological fallacy). Although we acknowledge this limitation, we think it is worth examining in detail the associations between income mobility and mortality at the aggregate level as a first descriptive effort to explore the potential consequences of income mobility for health.

1. We expect that association between a place's income mobility to which individuals are exposed early in life and their health and mortality as adults should be stronger in stages of the life cycle that reflect the mechanisms described above. That is, exposure during formative years (between ages 5 and 20) to a particular income mobility regime would have stronger effects on mortality in age groups 20-59 and then after age 60 (i.e., long-term consequences of individual early experiences).

2. Causes of deaths contributing to excess mortality among individuals in low income mobility places should be associated with traits, preferences and behaviors that are sculpted early in life. Thus, for example, income mobility should have a larger impact on mortality due to chronic illnesses associated with smoking and diet among older adults and those associated

with alcohol and drug use among younger adults, including suicides, homicides and other forms of violence.

3. Deleterious effects of a rigid income mobility regime should be stronger among individuals who occupy low to low-middle income ranks than among those located in more favorable positions in the income ranking. Similarly, the effects should be larger among African Americans and other minorities that have been traditionally discriminated against and have access to a much-reduced set of opportunities relative to other groups in the population, even in places with generous mobility regimes.

4. Gender differences should be small if men and women are subject to similar expectations regarding their social and economic success. In contrast, in communities where families expect less from their daughters than from their sons and, more generally, whenever investments in sons exceed those in daughters, there should be stronger effects of a place income mobility among males than among females.[4]

5. Finally, income mobility effects would be stronger in places with higher income inequality, that is in places where the health costs of income rigidity, particularly among those in the lower income ranks, are higher.

2.2 Data

We use a large data set that results from merging two separate data bases. The first is the Health Inequality Project Data (HIPD) created by Chetty and colleagues (Chetty et al. 2016) that contain information on income from tax records for the period 1999 and 2014 by US counties and commuting zones. The HIPD also include statistics of the income distributions and two indicators of income mobility derived from measures of the association between incomes of children born between 1971 and 1993 and their parents' income. First, we use the index of relative mobility (IRM), rank-rank slope, or the correlation between a child's income rank in her birth cohort income distribution and parents' income rank in their parents' income distribution (i.e., Spearman's correlation).[5] The relative income mobility indicator ranges between -1 and 1, and larger values correspond to lower income mobility (higher rank-rank correlation between parents' and child's income).

We also estimate models using absolute upward mobility score or "the mean rank (in the national income distribution) of children whose parents are at the 25^{th} percentile of the national parent income distribution" (Chetty et al. 2014, p. 7).[6] Absolute upward income mobility ranges

[4] In the absence of suitable measures of gender preference or standards regarding gender's investment differentials, this hypothesis can only be crudely assessed.

[5] Rank-rank slopes have proved to be quite robust across specifications and highly suitable for comparisons across areas (Chetty et al. 2014). *Canonical* measures of relative mobility, such as inter-generational income elasticity (of child income relative to parents' income) tend to be more sensitive to changes in inequality across generations.

[6] Although at the national level both the relative and absolute measure of mobility provide similar information, when studying small areas a child's rank in the national income distribution would be an absolute outcome because income a given area has little impact on the national distribution. We use a *permanent-resident* version of income

from 0 to 1 and higher values correspond to large income mobility. To facilitate interpretation we multiply the upward mobility score by -1 so that the meaning and expected association of relative and absolute income mobility with mortality are the same (i.e., increases in income mobility and inequality is expected to rise mortality risk and vice-versa).[7] Finally, we use the Gini Index (GI) as an indicator of income inequality.[8]

The second database we employ is the CDC mortality records by age and causes of death for U.S. counties during the period 2000-2014. This includes detailed mortality statistics (death and population counts by age and cause of death) for periods of time that better match the period of reference of the income mobility indicator in the HIPD. We compute mortality rates by five year age groups starting at age 0. This ensures close correspondence to the observed income mobility experiences and minimizes the influence of systematic biases (see below). In addition, we are able to compute mortality by causes of deaths and thus examine conjectures about patterns of their aggregate associations with income mobility.

After merging the two data bases, we are able to include a total of 2846 counties, about 91% of all counties in 2000 (see Figure S1 in **??**).[9] We build different data modules tailored to the particular hypothesis we examine. Our more general model and analysis requires death counts aggregated by county and age group (about 50,000 records). We then add more complexity by *disaggregating* the data by race/ethnic group and cause of death (about 200,000 records).

2.3 Model Estimation

2.3.1 General model and estimation

We seek to identify patterns of association between income mobility and mortality by county. First, we use death counts by county and age group as dependent variable. We fit Poisson models to the age group specific observed counts by gender with (mid-year) population as offset. The most general model pools the death counts for all years (2000-2014), ethnicity/race, and causes of death available in the CDC data. Second, we include random effects for age groups as well as for state and county to capture unstructured associations of the death counts. Finally, we adjust for over-dispersion by adding a random effect at the observation level.[10] The general model is specified as follows:

mobility measures, that is, parents who stay in the same counties between 1996-2012. Note that children who grow up in a county may have outmigrated as adults.

[7]Results using absolute mobility are shown in the **??**. They are similar to the ones using relative income mobility. This is not surprising as the correlation between relative and absolute mobility scores is high (-0.70).

[8]Gini coefficient within bottom 99%.

[9]The counties included in our analyses correspond to the continental US only as we exclude Alaska and Hawaii to estimate reasonable spatial models.

[10]All random effects are assumed to be IID with mean 0 and variance σ^2_ε.

$$
\begin{aligned}
D_i &\sim \text{Poisson}(\mu_i) \\
\log(\mu_i) &= \log(\tau_i) + \alpha + \beta_{\text{m}}\text{M}_i + \beta_{\text{g}}\text{G}_i + \beta X_i \\
\alpha &= \alpha_c + \alpha_{\text{state[i]}} + \alpha_{\text{county[i]}} + \alpha_{\text{obs[i]}} + \alpha_{\text{age[i]}} \\
\beta_{\text{m}} &= \beta_{\text{Mob}} + \beta_{\text{Mob}_{\text{age[i]}}} \\
\beta_{\text{g}} &= \beta_{\text{Gini}} + \beta_{\text{Gini}_{\text{age[i]}}}
\end{aligned} \tag{2.1}
$$

where D_i is the number of deaths by county and age, M_i is the income mobility measure for county i, G_i the Gini coefficient, and $\log(\tau_i)$ is the logarithm of the exposure for county i (i.e., log of mid-population). X_i represents of a set of covariates we adjust for.[11] In this model, the coefficients for income inequality (G) and income mobility (M) vary by age only, and $\alpha_{\text{obs[i]}}$ represents the adjustment for overdispersion.

SEbastian: La definicion de simbolos es ambigua. Di represnts bot cunt i and age x ...so porque no cambiarlo a DIx?

A few caveats are in order. First, in contrast to models with fixed effects using dummy variables for age groups (and corresponding interactions terms), the approach above allows for shifts in the estimates of age effects (and their corresponding confidence intervals) so that they are close to each other (partial pooling) where necessary. This is particularly important when, as happens in the CDC death statistics, information is sparse or when observed variation in the counts originates in noise not signal. Fitting this type of multilevel model results in an important advantage, namely, it yields more reliable estimates for small groups and facilitate multiple comparisons (**??**).

Second, we extend the general model above by exploring interactions and expanding the data set by race/ethnic group and cause of death. Given the small number of categories we examine (four race/ethnicity and cause of death groups), we estimate the model in Equation 2.1 separately for each race/ethnic/cause of death group.

Finally, and most importantly, to circumvent shortcomings inherent to standard maximum likelihood estimates (MLE), we adopt a Bayesian approach and estimate the models with multiple nested and crossed random effects. All models are estimated with the integrated nested Laplace approximation (INLA, Rue et al. 2009), as this method does not require the use of simulation to sample from a posterior distribution. This facilitates the estimation relative to MCMC-based approaches.[12]

To implement the Bayesian approach, we perform prior sensitivity analysis.[13] We start using the `R-INLA` default priors[14]. We explore different specifications for Penalized Complexity (PC) pri-

[11] Computing the exponential of $\alpha + \beta_{\text{m}}\text{M}_i + \beta_{\text{g}}\text{G}_i + \beta X_i$ yields the estimate of the mortality rate per county and age because $\log(\lambda_i) = \log(\mu_i) - \log(\tau_i)$.

[12] For an applied introduction see Blangiardo and Cameletti (2015); Wang et al. (2018) and Zuur (2017).

[13] For more details see the *Prior sensitivity analysis* section in the **??**.

[14] `R-INLA` uses a precision measure for the variance of the posterior distribution parameters defined as $\tau_\varepsilon = \frac{1}{\sigma^2_\varepsilon}$. The

ors which are designed to be weakly informative (for more details see **?**).[15] Fortunately, estimates associated with income mobility and inequality are insensitive to the different prior specifications we examined.

2.3.2 Model variants

We estimate six variants of the general model. The first is a baseline model that contains standardized income relative mobility (IRM), standardized income inequality (GI), centered log of the average household income and log of population at the county level as well as age, state, county, and observation level random-effects. Higher order models include income mobility and inequality age varying coefficients, interactions between income inequality and mobility, average income and corresponding interaction with IRM, and additional adjustments such as standardized income segregation, proportion of African-American (log), proportion of Hispanic (log), unemployment rate (log), proportion of people uninsured (standardized) and medicare expenses (standardized). All the variables were centered (see Table S1 in the **??** for descriptives of the variables we used).

Covariate adjustment could change estimates of the association between income mobility and mortality because they may capture unmeasured factors that confound the relationship of interest, but also because they reflect elements along a causal chain linking income mobility to health. Including covariates from the second group would amount to over-controlling and we intentionally sought to avoid this. For example, we do not include measures of health behaviors as covariates as it is likely that one of the pathways that relates income mobility and mortality includes changes in health behavior.[16]

Finally, we estimate models that consider spatial autocorrelation. Because county-level mortality data are area level information, spatial dependency is taken into account through neighborhoods structure. Neighbors are defined as the areas (counties) which share borders with it (first-order neighbors) or which share borders with it and with its first-order neighbors (second-order neighbors) (Blangiardo and Cameletti 2015). We use the parameterization proposed by Riebler et al. (2016).

---

default prior distribution for a fixed parameter is a normal distribution with mean 0 and precision $\tau = 0.001$, that is equivalent to $\sigma = 31.62$. This diffuse prior is used for all fixed regression parameters, except for the intercept in which case the precision is 0, that is, the corresponding sigma is large. The parameterization of random effects `R-INLA` uses a log gamma distribution for the priors of $log(\tau)$ with shape $a = 1$ and inverse scale $b = 0.00005$.

[15] PC priors require that we specify some scaling. To calibrate the scaling of the random effects prior, we set U and p to different values so that $Pr(\sigma_u > U) < p$. The values we use for U and p, respectively, are PC(1,.10), PC(10, .10), PC(10, .0.1). In the first case, for instance, we calibrate the prior so that the probability that the standard deviation of the random effect is greater than 1 is lower than .10. Our main results are based on the PC prior that ensured better fit (i.e.,, *deviance information criterion* (DIC) and *Watanabe Akaike information criterion* (WAIC)), that is, PC(1, .10).

[16] Goodness of fit of the covariate adjustment model using cross-validation is shown in the section *Goodness of Fit* in the **??**.

2.3.3 Converting estimates of parameters into estimates of adult life expectancy

To estimate effects of income mobility and income inequality on a summary measure of mortality, namely life expectancy at several ages, we use the parameter estimates of the model especified in Equation 2.1 and compute predicted values of mortality rates for each of 18 age groups. We then employ standard demographic procedures to construct all columns of a life table generated by the predicted rates. Finally, we compute life expectancy at various ages. We repeat these calculations for multiple scenarios defined by assigning values to the indicators of income inequality and mobility. With these estimates on hand we are able to compare expected values of life expectancy at different ages in places with high and low-income mobility or inequality. We use differences between predicted values as measures of the impact of each dimension of the stratification system.

2.4 Results

2.4.1 Average Association

Tables 2.1 (female) and 2.2 (male) display estimates of parameters corresponding to six models in which the dependent variable is death counts by county and age between 2000 and 2014 (51,606 records from 2867 counties). The coefficient β_{Mob} and β_{Gini} (see Equation 2.1) represent the *average association* between income relative mobility (IRM) and Gini index (GI) respectively. To simplify the description of results, we focus only on the effects of IRM, GI and corresponding interaction effects.

In the simplest baseline model (no age-varying coefficients) of each table, the standardized estimates of IRM and GI are .07 and .02 for females and .07 and .03 for males, respectively. Exponentiating these coefficients results in proportionate increases in mortality rates given a one unit increase (one standard deviation) in IRM and GI when all other variables are held equal to their average values. All effects are in the predicted direction, and IRM credibility intervals (CI) are all positive, the contrasts between males and females are minor and, as expected, the coefficient for county's income is several times higher than those of IRM and GI.

The second model (age-varying coefficient) allows the IRM and GI to vary by age-group and both the *deviance information criterion* (DIC) and *Watanabe Akaike information criterion* (WAIC) improve considerably. The third model includes the interaction between IRM and GI. Although positive as expected, the interaction effect is close to zero and imprecise. Thus, our data show no evidence that the association of IRM with mortality is stronger when income inequality is sharper. The fourth model includes the interaction between IRM and county's income. The corresponding effects are positive albeit small, suggesting that, contrary to expectations, the harmful effects of IRM on mortality gets larger in places with higher, not lower, income.

The fifth model adjusts for a set of covariates that stand for confounding factors, The fit of this model is better, but the magnitude of IRM coefficients changes only slightly. Figure 2.1 displays the posterior distribution of the exponentiated mobility and Gini coefficients by gender. The figure

shows that mortality rates among females are 1.09 and 1.01 higher in places with lower income mobility and higher income inequality respectively. Among males the excess mortality are 1.07 and 1.02, respectively. Thus, the deleterious impact of income mobility on mortality is always larger than the effect of income inequality.

The final model adds a structured term to account for spatial autocorrelation. Although this model increases the goodness-of-fit it modifies the coefficients of interest slightly and does not change previous inferences.

2.4.2 Effects by age

Average effects of income mobility and inequality in Table 2.1 and 2.2 are not completely informative since they refer to changes in average mortality rates assuming that each age group has the same weight. To circumvent this we estimate models with age-varying effects (see above) Tables 2.1 and 2.2 confirm two regularities. First, and as mentioned before, the fit of the model (assessed by DIC and WAIC) improves considerably after adding the random-coefficient terms for age. Second, the variability of IRM and GI coefficients by age-group is non-trivial and confirms the idea that effects of income mobility are age-patterned.

Figure 2.2 show (exponentiated) estimates and CIs of β_m and β_g from Equation 2.1 by sampling posterior distributions of Model *Covariates* in Tables 2.1 and 2.2. These can be interpreted as age-specific mortality ratios associated with shifts of one standard deviation of IRM and GI. In the case of women, the IRM curve suggests a higher impact at younger ages – possibly a reflection of parental conditions – a peak during early adulthood (25-44) and a gradual tapering off at older ages. The shifts due to income mobility are always larger than those for income inequality and reach their peak at later ages. For males, the IRM curve is less pronounced and flatter. Ratios are relatively stable until late adulthood and although the differences between effects of income mobility and inequality ratios are in the same direction as among women, their magnitudes are smaller.

To show the impact of shifts in income mobility and inequality using an easily understood metric, we estimate differences in life expectancy at various ages associated with changes in one standard deviation of income mobility (income inequality). To do this we use Model *Covariates* (Tables 2.1 and 2.2) to predict individual mortality rates by age using. We then predict counterfactual mortality rates by computing the following quantities:

$$\begin{aligned} \alpha &= \alpha_c + \alpha_{\text{age[i]}} \\ \beta_{\text{m}} &= \beta_{\text{Mob}} + \beta_{\text{Mob}_{\text{age[i]}}} \\ \beta_{\text{g}} &= \beta_{\text{Gini}} + \beta_{\text{Gini}_{\text{age[i]}}} \end{aligned} \tag{2.2}$$

The values of covariates other than income mobility and income inequality are always set to their mean (i.e., zero). Once we obtain the set of predicted mortality rates by age, we estimate life expectancy using standard life tables for five-year age groups (0-4, 5-9, ..., 75-84, 85+). We

assume that the average person years lived by those dying within an interval ($_na_x$) is 0.5 for all the age groups, except the first one where we use 0.3. For the last age-group, we compute $_na_x$ as the reciprocal of the mortality rate ($\frac{1}{m_x}$).[17]

Figure 2.3 displays curves of the magnitude of predicted (absolute) changes in life expectancy by age implied by increases in IRM (decreases in income mobility) and GI (increases in income inequality) equivalent to one standard deviation. The graphs are sensitive to both differences in levels of mortality and the magnitude of effects by age. The largest (absolute) life expectancy losses is at age 0 ($E(0)$) but these gradually decrease with age. Although there are no significant gender contrasts in the age patterns of losses, the magnitude of differences due to changes in IRM and GI is, as before, slightly lower for men than for women and, also as verified before, the absolute differences are consistently higher for income mobility than for income inequality. The fact that the absolute magnitude of differences or losses in life expectancy is larger at age 0 should not be surprising since, unless the sign of the estimated effects varies by age, the age-specific effects will accumulate over time. Since life expectancy at birth reflects the sum total of effects throughout the life course, the magnitude of the expected impacts will be higher at age 0. Furthermore, because life expectancy at birth is disproportionately influenced by changes in mortality before age 5, minor differences in effects between very young and adult age groups may be over-represented changes of life expectancy at age 0. An alternative way to assess the impact of shifts in income mobility and inequality is to compute the magnitude of the *relative* changes. These quantities are plotted in Figure 2.4 and, as expected, they show an increasing trend by age, particularly at older adult ages, where the size of life expectancy declines rapidly.

2.4.3 Effects by Race/Ethnicity

Do the effects uncovered before vary by race and/or ethnic group? Figure 2.5 displays the posterior distribution of mortality ratios using the model *Covariates* of Table 2.2 and 2.1 estimated separately for the following race/ethnic groups: Non-Hispanic Whites, African Americans, Hispanics, and Other. The figures reveal that IRM exerts larger influence than GI and in the expected direction in all groups. The figure also shows that contrasts between the impact of income mobility and income inequality are smallest for African American (males and females) and largest for Non-Hispanic Whites and Others. In addition, note that the uncertainty of estimates is always largest for Other and smallest for non-Hispanic Whites. Finally, while non-Hispanic White females mortality is more sensitive than male mortality to income mobility the same is not the case in the Other groups.

Figure 2.6 displays curves with the predicted changes in life expectancy by age implied by a standard deviation upward shift in IRM and GI for African Americans and Non-Hispanic Whites. Therwe are clear differences between African Americans males and Non-Hispanic Whites males in life expectancy: at $E(0)$ African Americans decrease about 1.5 years in life expectancy versus 0.6 years among Whites. Females' differences associated with a shift in IMR are smaller and difficult to assess precisely. Shifts in GI also have a harmful effect for African Americans, although considerably smaller (about 0.6 years of decrease in life expectancy at $E0$) than effects of IMR

[17] For details on these calculations see the code in the repository: `https://github.com/sdaza/dissertation/tree/master/ch02`

(between 1 and 1.5 years). Again, differences in impact are assessed using *relative* changes (see Figure 2.7). These quantities confirm differences between Whites and African Americans and reveal tha the gaps between effects of GI and IRM increase with age.

2.4.4 Effects by causes of death

We now examine patterns of association between income mobility and mortality by broad groups of causes of death. To simplify estimation we classify the total 39 selected causes of death adopted by the CDC into four broad groups of causes: communicable diseases, non-communicable diseases, injuries (including accidents, suicides, homicides), and residual causes.[18]

We estimate the *Covariates* model in Table 2.1 and 2.2, but do so separately by four cause death as defined before. Figure 2.8 displays the posterior distribution of mortality ratios $\exp(\beta_m)$ and $\exp(\beta_g)$ (see Equation 2.1). The effects are uniformly in the expected direction, they are always larger for income mobility than for income inequality, and behave similarly for males and females. The largest ratios are associated with communicable diseases, a group that includes HIV, other STD-related deaths, and respiratory TB as major contributors, as well as more diffuse illnesses such as influenza, pneumonia, and bronchitis. These effects, however, are also the least certain, e.g., their posterior distributions have large variances. Our initial conjecture is that we should see larger contrasts in causes of deaths involving consumption of substances (particularly reflected in injuries, a group that includes suicides, accidents, and homicides) as well as those associated with high risk behaviors (e.g. STD's). As comnfirmed by the Figure this is in fact the case. Some contrasts in non-communicable diseases are also expected (e.g. smoking related causes, T2D) but we cannot discern patterns with more fine tuned grained groups of causes of deaths due to small number of events.

Overall, although the pattern of results for causes of deaths are concordant with the hypothesis formulated at the outset, they do not supply a platform for strong inferences. First, we have scarce power to estimate simultaneously age and causes of death effects which would be required for a rigorous test of the hypothesis implicating causes of death. Second, alternative explanations could be invoked to account for the observed patterns (e.g. excess deaths due to STD's may be due to unmeasured excess poverty in places with low income mobility) and these cannot be easily discarded.

2.5 Discussion

The results of these analysis are mixed. First, there is little doubt that the gross impact of a place's income is significantly larger than those associated with either income mobility or income inequality (Chetty et al. 2016). Thus, US geographic disparities are reduced but not eliminated after accounting for income mobility (and income inequality). However,our findings also show that the

[18] See Table S6 in **??** for details on the coding schema we use.

association between mortality and income mobility is uniformly stronger than that between income inequality and mortality. This empirical evidence alone should support the case for income mobility as a relevant mortality determinant, perhaps more relevant that income inequality

Second, and contrary to our expectations, the income gradient of beneficial effects of higher income mobility is positive as places with higher income tend to experience larger mortality reductions as income mobility increases. In contrast, and concordant with our expectation, the effects of income mobility are larger among disadvantaged and discriminated groups, such as African American males and other minorities (Hispanics). Third, as expected if there are no gender differentials in parental investments on offspring, we observe no persistent and marked gender differentials among non Hispanic Whites, although effects among women are slightly higher than among men.

Finally, the analyses by causes of deaths reveal patterns that are largely consistent with the hypothesis. In particular, causes of deaths are highly sensitive to income mobility are strongly associated with high risk behaviors. Yet, this evidence is too coarse to discard alternative explanations and firmly establish the role of income mobility.

Undoubtedly the tools we use here to test conjectures about the role of income mobility are blunt. But they are in no case blunter than those utilized to produce evidence on which the whole edifice about the relation between mortality and income inequality has been built over many years

Among the limitations of our analysis, we should mention the nature of our data. Despite its richness, it has several shortcomings. The most important is the potential dislocation between exposure to an income mobility regime during formative years and adult mortality experiences. The CDC mortality information does not refer people originating in different places but to people who die in those places. Therefore, the associations we observe might be the result of selective migration over the life-course and not of exposures to a given mobility regime during critical ages. In other words, the effects of income mobility or inequality may be contaminated by characteristics that distinguish migrants from non-migrants. The only way to circumvent this is to use individual longitudinal data that provides enough information to either directly assess or to neutralize the effects of migration selection processes.

A second shortcoming is that even in the absence of residential migration, the measures of a place's income mobility does not map tightly to mortality experiences of interest. Thus, for example, a place's income mobility assessed for generations born in 1980 ought to be relevant for youth mortality in years (approximately) 1995-2010 and to older adult mortality for years 2040 and later. Lack of correspondence between income mobility and mortality is not problematic in a stationary regime, e.g., when a place's income mobility at time t stays the same for a generation or so. The closer a place's income mobility is to a stationary regime, the stronger will be our inferences.

It is important to note that some of the mechanisms linking mobility and mortality operate at the individual level and, therefore, it is problematic to infer them from patterns observed in aggregate data (e.g., ecological fallacy). Ours is only an initial attempt to explore key conjectures that establish the fundamental role of income mobility

To make further progress we must proceed in two different directions. First, we should identify precisely the mechanisms linking adult health and mortality to both a place's income mobility (aggregate property) as well as to the actual income mobility experienced by the parental and the great parental generations (family level property). Both may exert influences on early formative environments and the adoption of health behaviors. In particular, the latter is likely to be influenced by and act jointly with a place's aggregate income mobility to modify early upbringing and socialization, the formation of future outlooks, and the adoption of attitudes and behaviors that minimize exposure to health risks. It may be the case that growing up in a community with a rigid stratification system discourages individuals in less advantageous positions and facilitates adoption of behaviors that provide immediate rewards but are highly noxious, difficult to abandon, and bearers of large effects that take a long time to manifest. Yet, an individual's family mobility experience could be equally influential and may even offset deleterious effects stemming from a place's income mobility.

Second, we should focus on individual outcomes at several stages in the life course. Although death is a fairly definitive state and mortality rates can be assessed with little difficulty, we need observation of a chain of intermediate outcomes spread over individuals' life course. If place and familial income mobility turn out to be important, counteracting the deleterious effects of unfavorable income mobility regimes and adverse mobility experiences will require changes that are no different from those advocated by economists to increase human capital. All of them require modifying early childhood environments (**?**). If early cognitive and non-cognitive skills matter for labor market success, income and wages, they may also matter for adult health not only because socioeconomic success breeds good health but because these traits are the same that determine health behaviors and associated lifelong exposures. While one cannot alter a place's income mobility overnight anymore than one can shift its income distribution, timely changes in parental and child educational programs may go a long way to shield individuals and families from negative backlashes of rigid mobility regimes.

2.6 Tables and Figures

Table 2.1: County Level Poisson Models Relative Mobility
PC prior $= Pr(\sigma > 1) < 0.10$, Women, CDC 2000-2014

	Baseline	Varying-Coefficient	Relative mobility x Gini	Relative mobility x Income	Covariates	Spatial
Constant	−5.81	−5.82	−5.82	−5.82	−5.82	−5.82
	[−6.69; −4.93]	[−6.71; −4.93]	[−6.72; −4.93]	[−6.73; −4.90]	[−6.73; −4.92]	[−6.72; −4.92]
Income relative mobility	0.07	0.10	0.10	0.10	0.09	0.08
	[0.06; 0.07]	[0.07; 0.13]	[0.07; 0.13]	[0.07; 0.13]	[0.06; 0.12]	[0.05; 0.11]
Gini	0.02	0.01	0.01	0.01	0.01	0.02
	[0.01; 0.02]	[−0.00; 0.03]	[−0.00; 0.03]	[−0.00; 0.03]	[−0.00; 0.03]	[−0.00; 0.04]
Log income	−0.38	−0.37	−0.37	−0.36	−0.28	−0.27
	[−0.40; −0.35]	[−0.39; −0.34]	[−0.39; −0.34]	[−0.38; −0.33]	[−0.31; −0.25]	[−0.30; −0.24]
Relative mobility x Gini			0.01			
			[0.00; 0.01]			
Relative mobility x Log income				0.04		
				[0.02; 0.06]		
Random Effects						
SD observations	0.13	0.10	0.10	0.10	0.10	0.10
	[0.12; 0.13]	[0.10; 0.10]	[0.10; 0.10]	[0.10; 0.10]	[0.10; 0.10]	[0.10; 0.10]
SD age group	1.87	1.90	1.92	1.96	1.93	1.91
	[1.45; 2.55]	[1.42; 2.54]	[1.42; 2.62]	[1.42; 2.60]	[1.41; 2.51]	[1.41; 2.56]
SD counties	0.10	0.10	0.10	0.10	0.09	0.12
	[0.10; 0.10]	[0.09; 0.10]	[0.09; 0.10]	[0.09; 0.10]	[0.09; 0.09]	[0.11; 0.12]
Phi counties						1.12
						[1.09; 1.17]
SD states	0.07	0.07	0.07	0.07	0.06	0.05
	[0.06; 0.09]	[0.06; 0.09]	[0.06; 0.09]	[0.06; 0.09]	[0.05; 0.08]	[0.03; 0.06]
SD mobility by age		0.06	0.06	0.06	0.06	0.06
		[0.05; 0.09]	[0.04; 0.09]	[0.05; 0.09]	[0.05; 0.09]	[0.05; 0.09]
SD gini by age		0.04	0.04	0.04	0.04	0.04
		[0.03; 0.05]	[0.03; 0.05]	[0.03; 0.05]	[0.03; 0.05]	[0.03; 0.05]
DIC	365579	364194	364196	364194	364133	364053
WAIC	363118	362578	362581	362574	362506	362443

Note: Selected coefficients (mean of marginal posterior distribution). Poisson model with offset = `log(population)`. 95% credibility intervals.

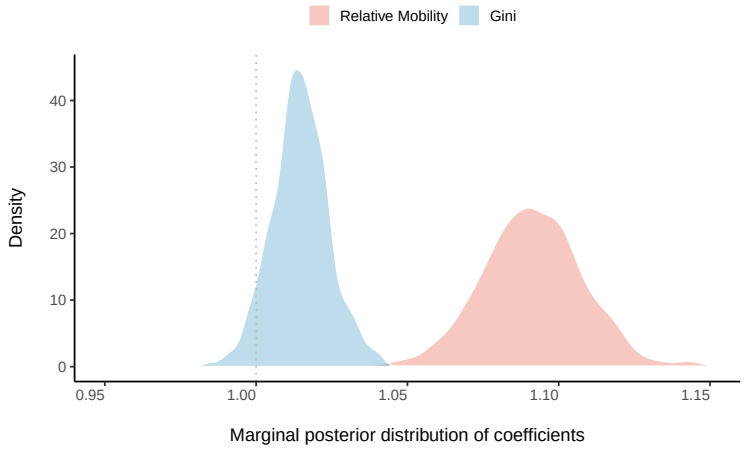
Table 2.2: County Level Poisson Models Relative Mobility
PC Prior = $Pr(\sigma > 1) < 0.10$, Men, CDC 2000-2014

	Baseline	Varying-Coefficient	Relative mobility x Gini	Relative mobility x Income	Covariates	Spatial
Constant	−5.31	−5.32	−5.32	−5.31	−5.31	−5.31
	[−6.16; −4.46]	[−6.18; −4.46]	[−6.16; −4.48]	[−6.17; −4.45]	[−6.19; −4.44]	[−6.18; −4.44]
Income relative mobility	0.07	0.08	0.08	0.09	0.07	0.06
	[0.06; 0.08]	[0.06; 0.10]	[0.06; 0.10]	[0.07; 0.11]	[0.05; 0.09]	[0.04; 0.08]
Gini	0.03	0.03	0.03	0.03	0.02	0.02
	[0.02; 0.03]	[0.01; 0.05]	[0.01; 0.05]	[0.01; 0.05]	[0.01; 0.04]	[0.01; 0.04]
Log income	−0.37	−0.37	−0.37	−0.35	−0.23	−0.20
	[−0.40; −0.34]	[−0.40; −0.34]	[−0.40; −0.34]	[−0.38; −0.32]	[−0.26; −0.19]	[−0.23; −0.17]
Relative mobility x Gini			0.01			
			[0.00; 0.01]			
Relative mobility x Log income				0.07		
				[0.05; 0.10]		
Random Effects						
SD observations	0.14	0.12	0.12	0.12	0.12	0.12
	[0.14; 0.14]	[0.12; 0.13]	[0.12; 0.13]	[0.12; 0.13]	[0.12; 0.13]	[0.12; 0.13]
SD age group	1.83	1.85	1.83	1.83	1.87	1.83
	[1.38; 2.47]	[1.38; 2.49]	[1.37; 2.54]	[1.40; 2.44]	[1.30; 2.49]	[1.31; 2.48]
SD counties	0.11	0.11	0.11	0.11	0.10	0.13
	[0.11; 0.12]	[0.11; 0.12]	[0.11; 0.12]	[0.11; 0.12]	[0.10; 0.10]	[0.12; 0.14]
Phi counties						1.17
						[1.09; 1.25]
SD states	0.07	0.07	0.07	0.07	0.06	0.05
	[0.06; 0.09]	[0.06; 0.09]	[0.05; 0.09]	[0.06; 0.09]	[0.05; 0.08]	[0.04; 0.07]
SD mobility by age		0.04	0.04	0.04	0.04	0.04
		[0.03; 0.06]	[0.03; 0.06]	[0.03; 0.06]	[0.03; 0.06]	[0.03; 0.06]
SD gini by age		0.04	0.04	0.04	0.04	0.04
		[0.02; 0.05]	[0.02; 0.05]	[0.02; 0.05]	[0.03; 0.05]	[0.03; 0.05]
DIC	392522	391921	391926	391916	391854	391808
WAIC	389480	389792	389801	389786	389701	389663

Note: Selected coefficients (mean of marginal posterior distribution). Poisson model with offset = `log(population)`. 95% credibility intervals.

Figure 2.1: Posterior Distribution of $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ by Gender
Model *Covariates* in Tables 2.1 and 2.2

(a) Women



(b) Men

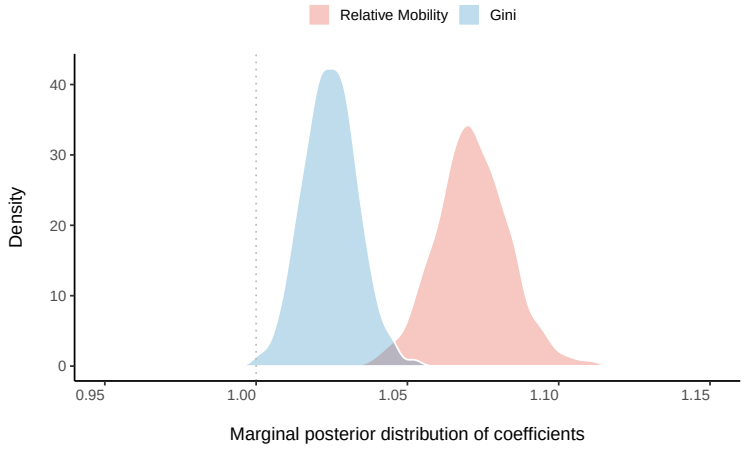
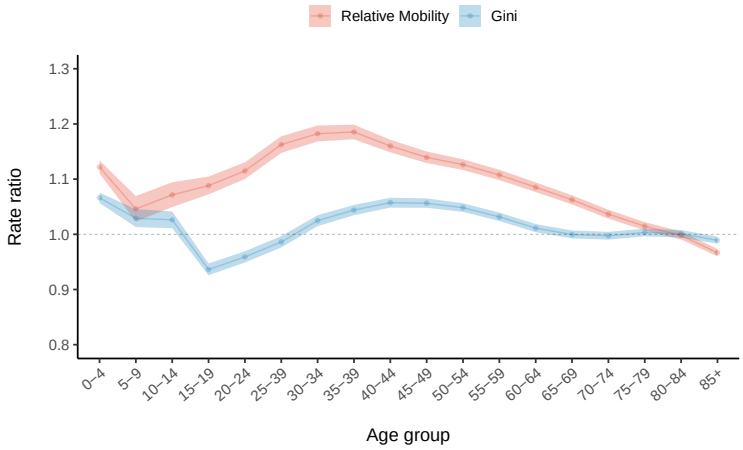


Figure 2.2: 95% Credibility Interval Posterior Distribution of $exp(\beta_m)$ and $exp(\beta_g)$ (Equation 2.1) by Age Group Model *Covariates* in Tables 2.1 and 2.2

(a) Women



(b) Men

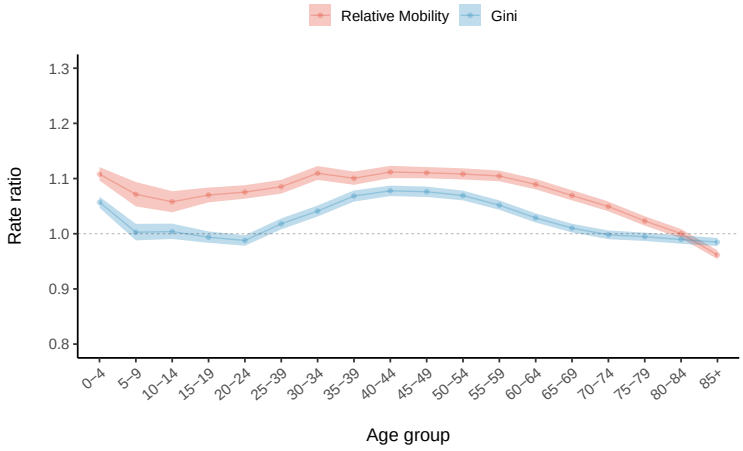
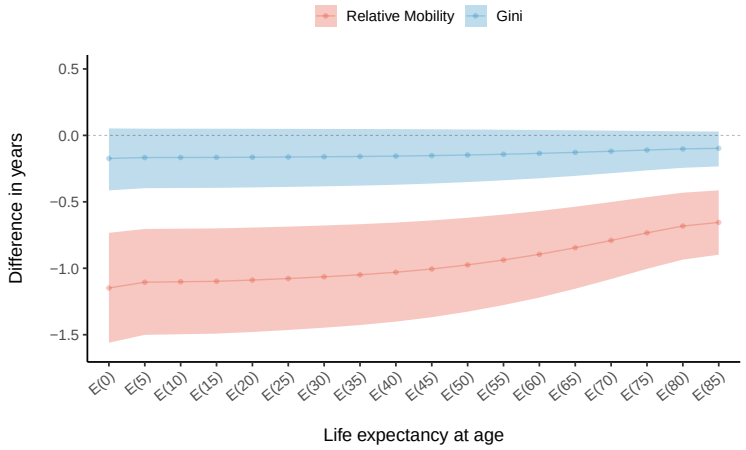


Figure 2.3: 95% Credibility Interval of Predicted LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 2.1 and 2.2

(a) Women



(b) Men

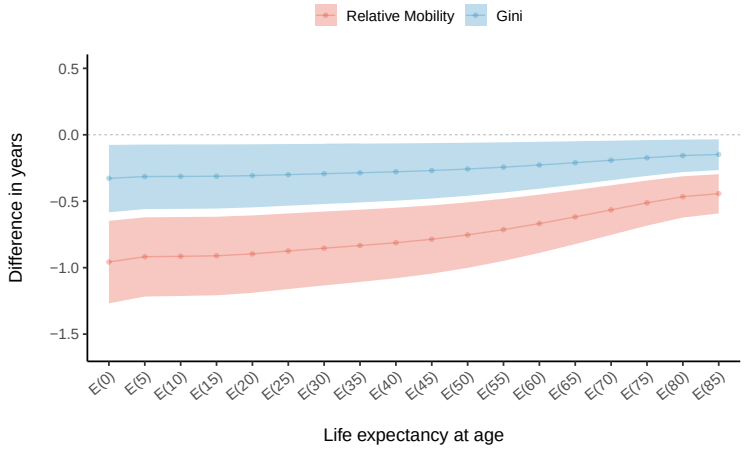
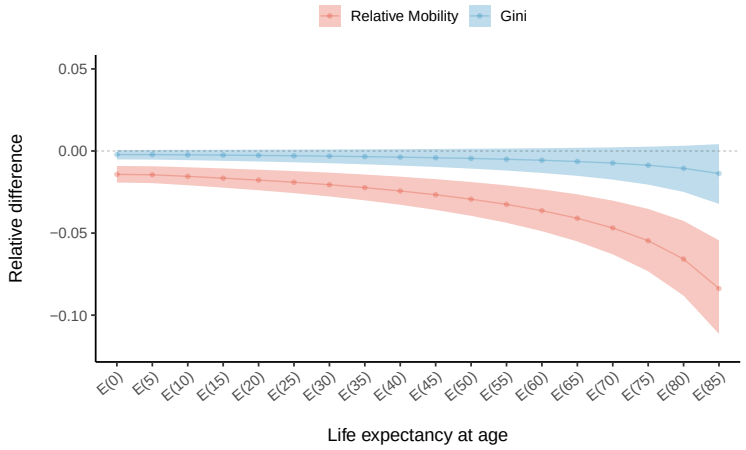


Figure 2.4: 95% Credibility Interval of Predicted Relative LE Differences by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 2.1 and 2.2

(a) Women



(b) Men

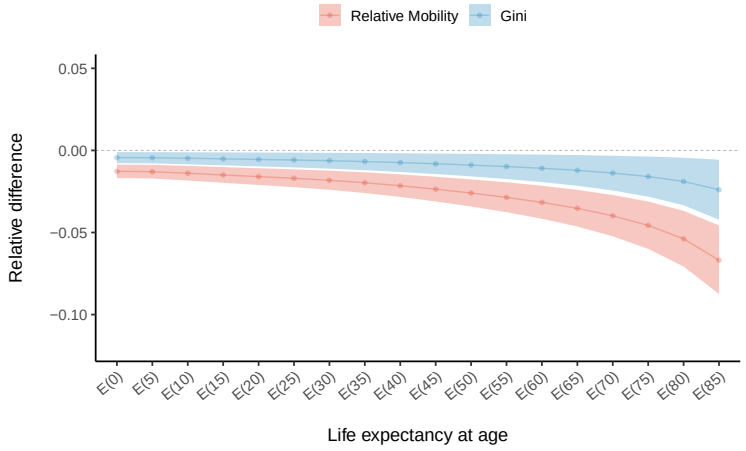
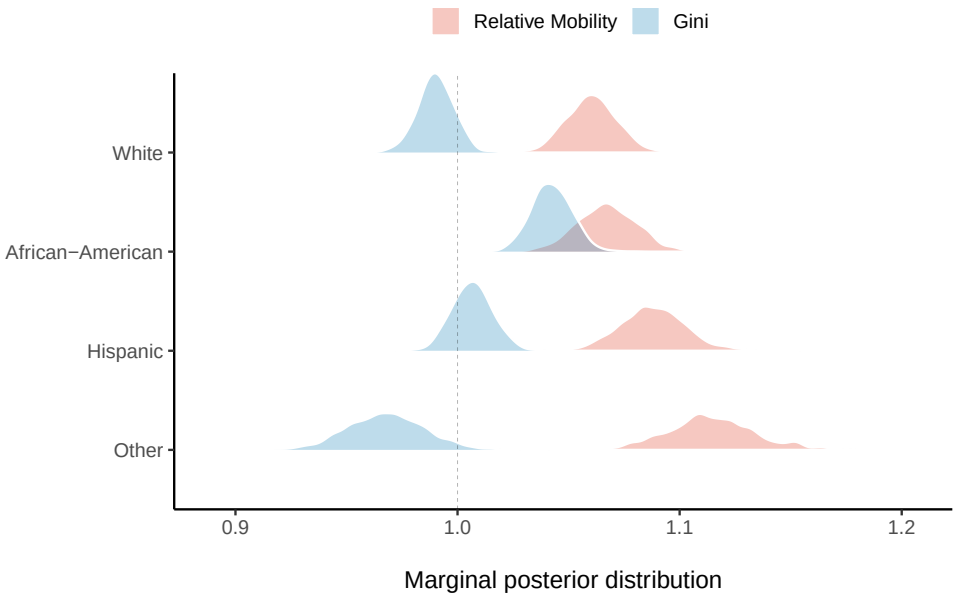


Figure 2.5: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ (Equation 2.1) by Race/Ethnicity and Gender

(a) Women



(b) Men

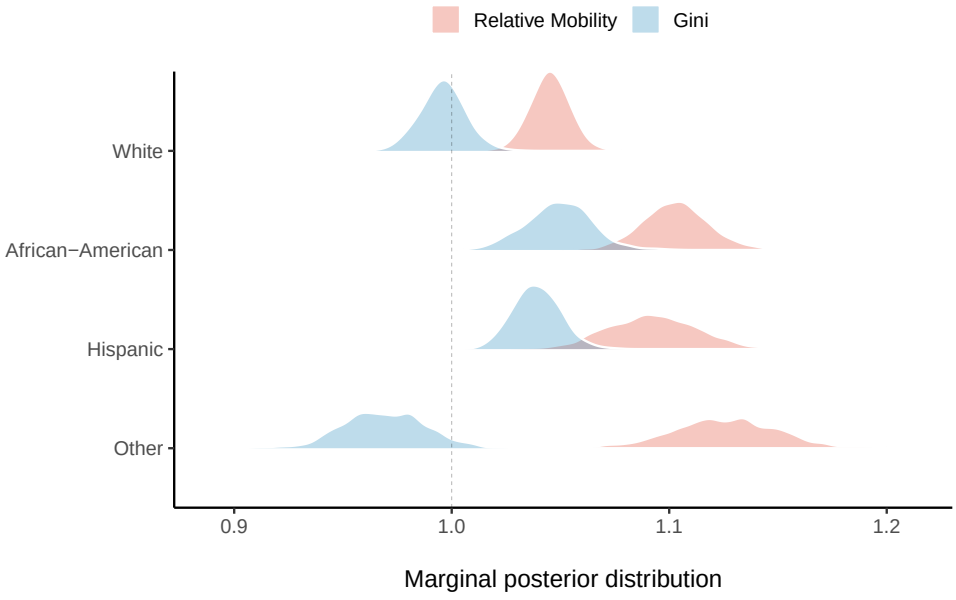
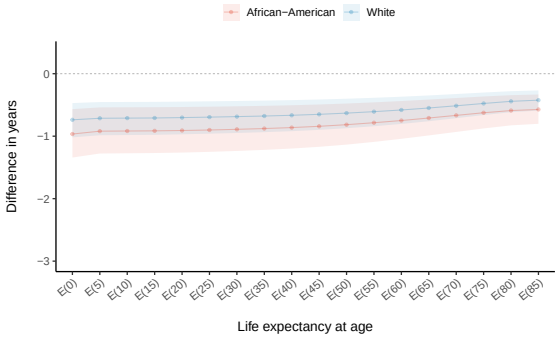
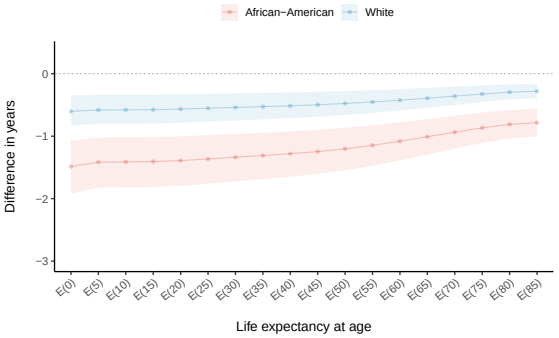


Figure 2.6: 95% Credibility Interval of Predicted LE Differences
by Age Group and Race/Ethnicity, Increase in One Standard Deviation

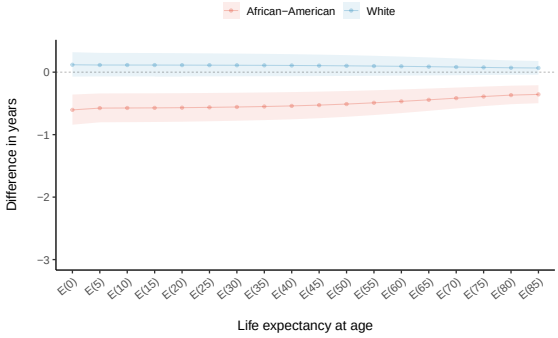
(a) Women - Relative Mobility



(b) Men - Relative Mobility



(c) Women - Gini



(d) Men - Gini

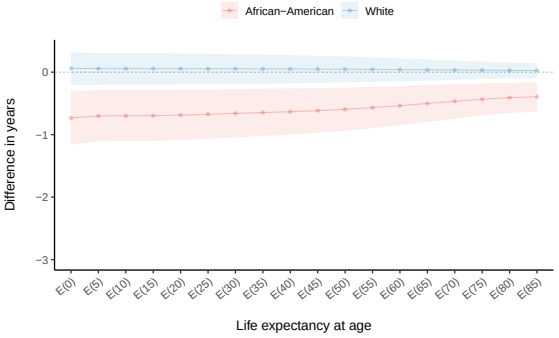
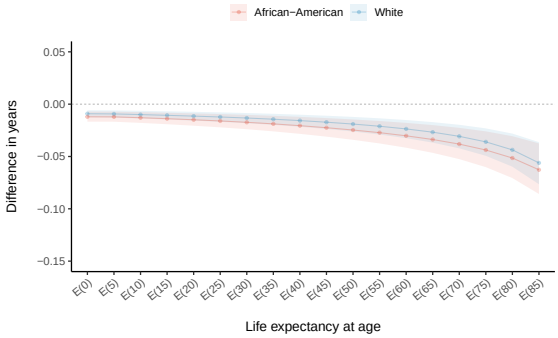
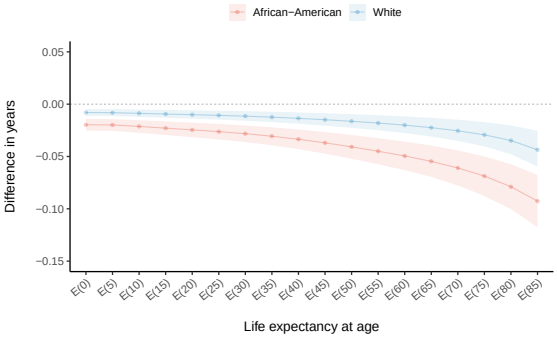


Figure 2.7: 95% Credibility Interval of Predicted LE Relative Differences by Age Group and Race/Ethnicity, Increase in One Standard Deviation

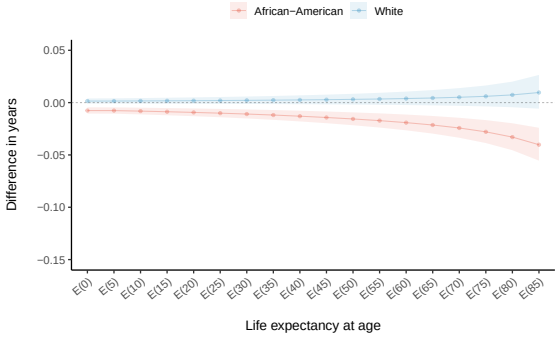
(a) Women - Relative Mobility



(b) Men - Relative Mobility



(c) Women - Gini



(d) Men - Gini

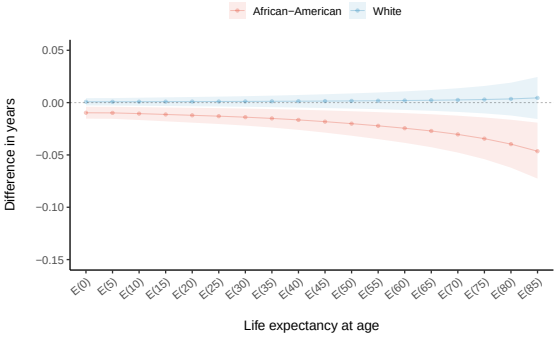
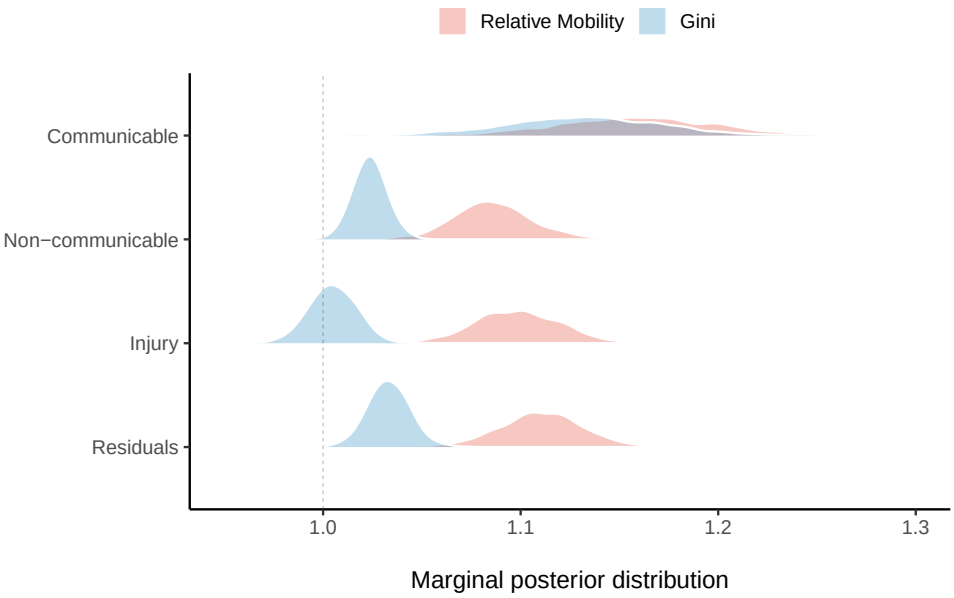
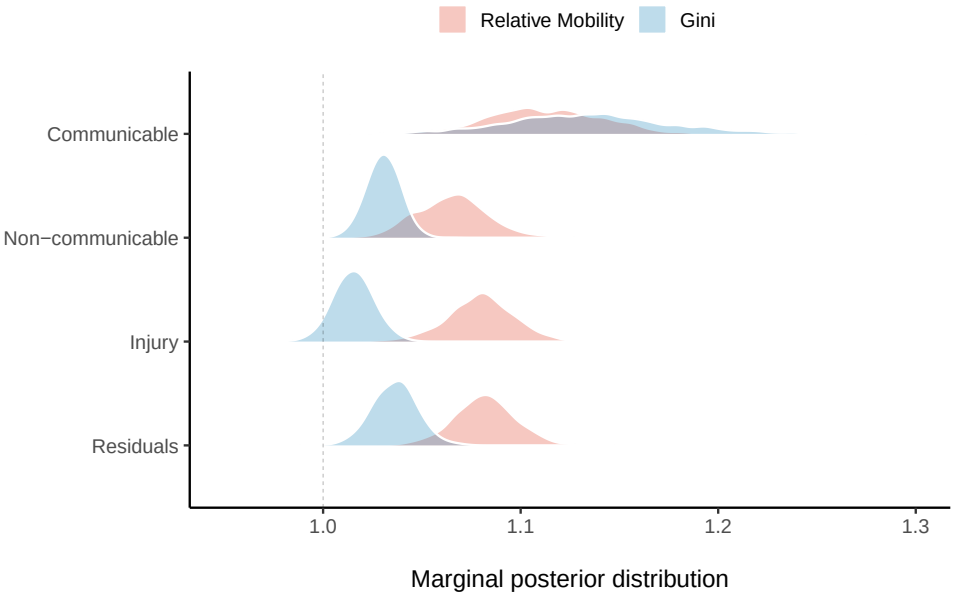


Figure 2.8: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta\text{Gini})$ by Cause of Death and Gender

(a) Women



(b) Men



Methodological Supplement

The code used to create the database and run the models and plots is available at: `https://github.com/sdaza/dissertation/tree/master/ch02`.

1.1 Descriptive Statistics County Level

Figure S1: County Coverage Income Mobility Measures (Colored)
2867 counties (91%)

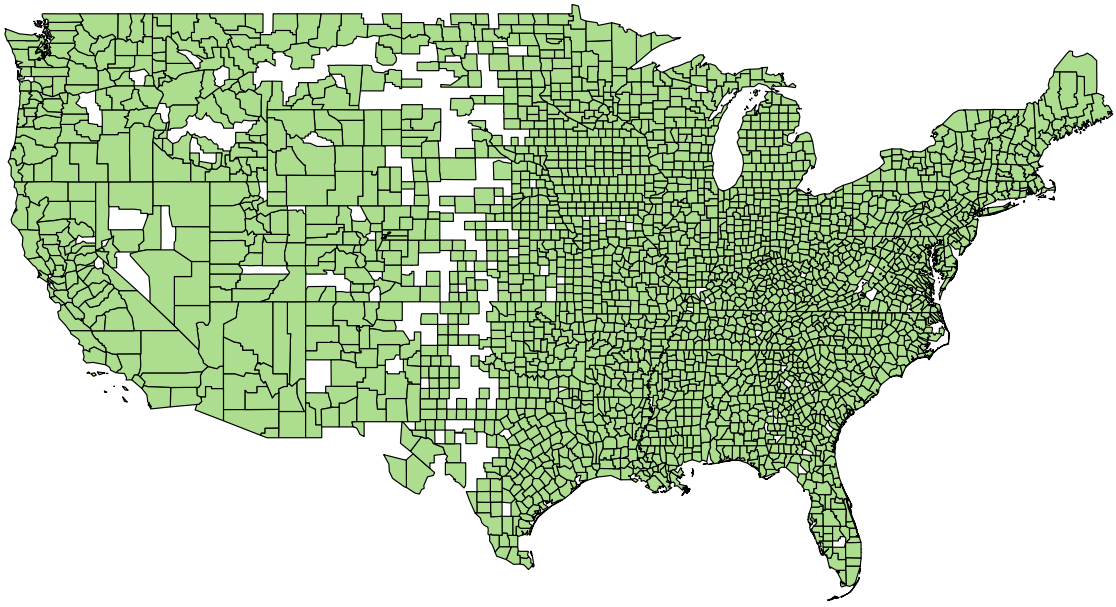
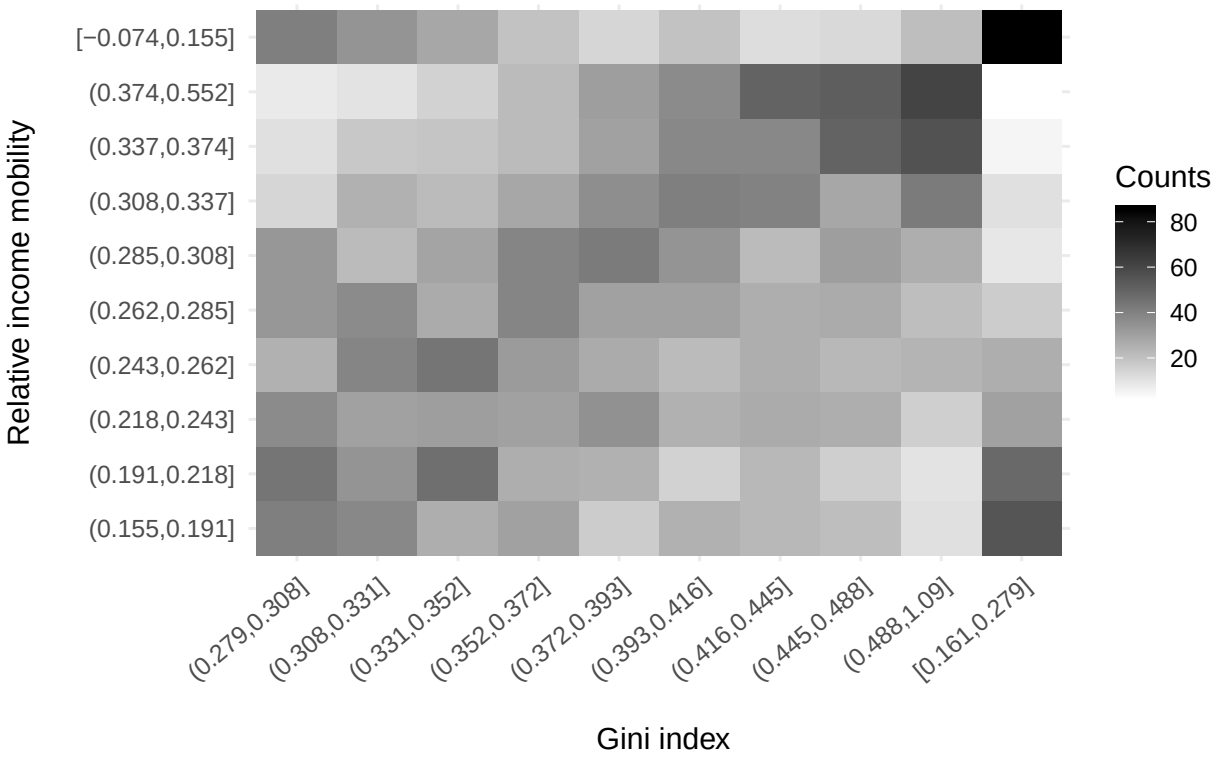


Table S1: Descriptive Statistics

Variable	N	Mean	Median	SD	Min	Max
Relative income mobility ($\times$100)	2867	26.43	26.23	8.59	-7.40	55.23
Absolute income mobility ($\times$100)	2867	47.33	46.84	6.08	23.17	68.32
Gini coefficient	2867	0.38	0.37	0.09	0.16	1.09
Population 2000	2867	97758.63	28274.00	304860.67	2837.00	9519338.00
Household income	2867	32963.43	32258.13	7060.38	13646.95	77942.65
Income segregation	2867	0.03	0.02	0.03	-0.00	0.18
Unemployment rate	2867	0.05	0.05	0.02	0.02	0.18
% Hispanic	2867	5.99	1.78	11.82	0.16	97.54
% African-American	2867	9.18	2.02	14.67	0.00	85.97
% Uninsured	2867	18.08	17.85	5.29	3.63	38.09
Medicare expenses	2867	9333.58	9233.24	1558.80	4265.11	18443.22

Note: Variables are in their original scale (i.e., before log transformation and standardization).

Figure S2: Overlap Income Mobility and Inequality, 2867 counties



1.2 Goodness of Fit

We examine the goodness of fit (GOF) of the model *Covariates* in Table 2.1 and 2.2 using the *leave-one-out* predictive measure *probability integral transform* (PIT) (Wang et al. 2018).

The probability integral transform (PIT) is defined as:

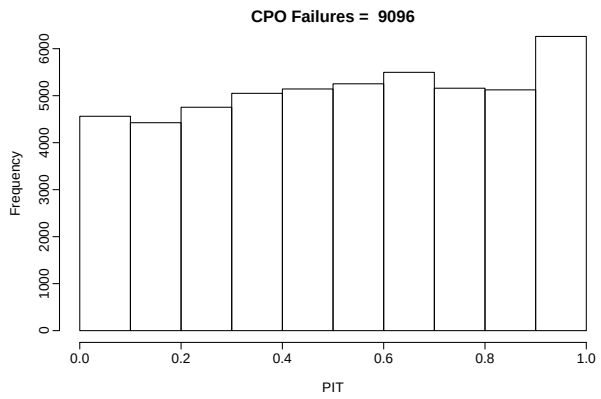
$$PIT_i = p(y_i^{new} \leq y_i | y_{-i})$$

where y_{-i} denotes the observations y with the i^{th} observation omitted. The only difference between PIT and the posterior predictive p-value is that PIT is computed based on y_{-i} rather than y. We would expect PIT statistics to be approximately uniformly distributed for a good model. Values of PIT close to zero or one would indicate observations which are much smaller or larger than expected. One advantage of the PIT relative to other measures such as the *conditional predictive ordinate* (CPO) is that the deviations have a direction.

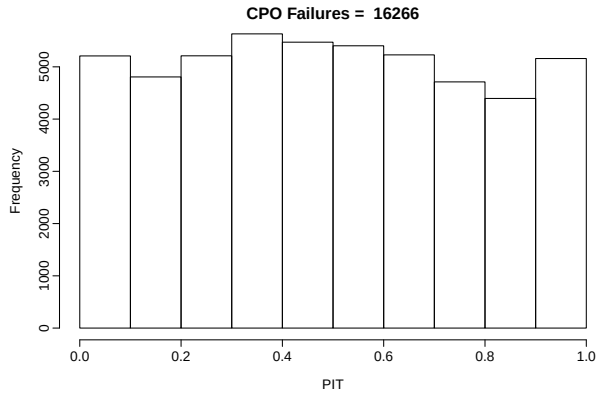
Figures S3 and S4 show the histogram and the uniform Q-Q plot of PITs for females and males. As can be seen, the distribution of the PITs is close to a uniform distribution, suggesting that the model reasonably fits the data.

Figure S3: PIT Distribution
Model *Covariates* in Tables 2.1 and 2.2

(a) Women



(b) Men

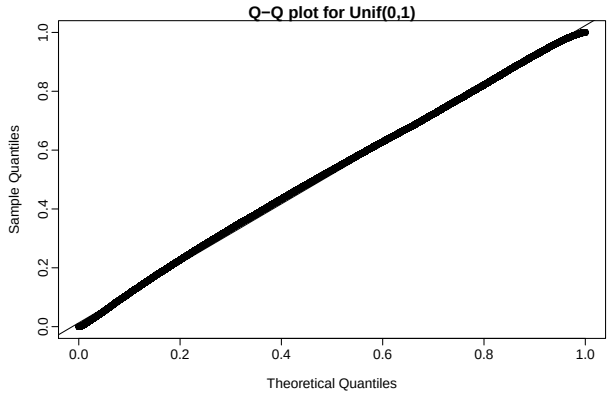


1.3 Prior sensitivity analysis

We perform prior sensitivity analysis. We start using the `R-INLA` default priors. `R-INLA` uses a precision measure for the variance of the posterior distribution parameters defined as $\tau_\varepsilon = \frac{1}{\sigma_\varepsilon^2}$. The default prior distribution for a fixed parameter is a normal distribution with mean 0 and precision $\tau = 0.001$, that is equivalent to $\sigma = 31.62$. We use this diffuse prior for all fixed regression parameters, except for the intercept in which case the precision is 0, that is, the corresponding sigma is large. For parameterization of random effects `R-INLA` uses a log gamma distribution for the priors of $log(\tau)$ with shape $a = 1$ and inverse scale $b = 0.00005$. Then, we explore different specifications for Penalized Complexity (PC) priors which are designed to be weakly informative (for more details see **?**). PC priors require we specify some scaling. To calibrate the scaling of the random effects prior, we set U and p to different values so that $Pr(\sigma_u > U) < p$. The values we use for U and p, respectively, are PC(1,.10), PC(10, .10), PC(10, .0.1). In the first case, for instance, we calibrate the prior so that probability that the standard deviation of the random effect is greater than 1 is lower than .10.

Figure S4: Q-Q Plot PIT
Model *Covariates* in Tables 2.1 and 2.2

(a) Women



(b) Men

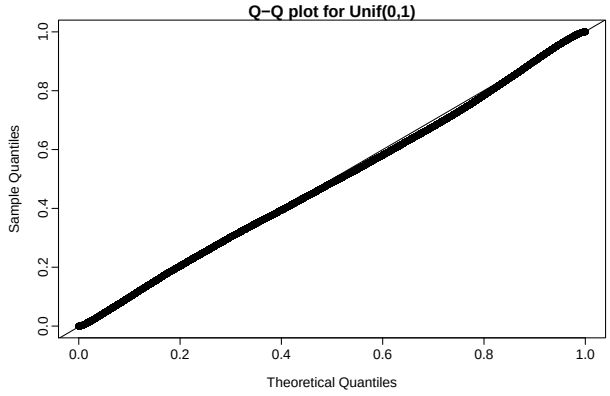


Table S2 and S3 show the results using different priors with a model equivalent to the *Covariates* model in Table 2.1 and 2.2 of the main paper. As can be seen, fixed effects practically do not change when using different prior specifications. The precision of random terms, as expected, shows more variability although changes are small. We decide to use the model with better DIC and WAIC across genders, that is, PC(1, 0.10).

Table S2: County Level Poisson Models, Prior Sensitivity, Women, CDC 2000-2014

	INLA Default	PC(1, .10)	PC(10, .10)	PC(10, 0.01)
Constant	−5.82	−5.82	−5.82	−5.82
	[−6.76; −4.89]	[−6.70; −4.94]	[−6.81; −4.83]	[−6.80; −4.84]
Income relative mobility	0.09	0.09	0.09	0.09
	[0.06; 0.11]	[0.06; 0.12]	[0.06; 0.12]	[0.06; 0.12]
Gini	0.01	0.01	0.01	0.01
	[−0.00; 0.03]	[−0.00; 0.03]	[−0.00; 0.03]	[−0.00; 0.03]
Random Effects				
SD observations	0.10	0.10	0.10	0.10
	[0.10; 0.10]	[0.10; 0.10]	[0.10; 0.10]	[0.10; 0.10]
SD age group	2.00	1.90	2.14	2.10
	[1.43; 2.73]	[1.43; 2.67]	[1.53; 3.10]	[1.51; 3.01]
SD counties	0.09	0.09	0.09	0.09
	[0.09; 0.09]	[0.09; 0.09]	[0.09; 0.09]	[0.08; 0.09]
SD states	0.06	0.06	0.06	0.06
	[0.05; 0.08]	[0.05; 0.08]	[0.05; 0.08]	[0.05; 0.08]
SD mobility by age	0.06	0.06	0.06	0.07
	[0.04; 0.08]	[0.04; 0.09]	[0.05; 0.09]	[0.05; 0.09]
SD gini by age	0.03	0.04	0.04	0.04
	[0.02; 0.05]	[0.03; 0.05]	[0.03; 0.05]	[0.03; 0.05]
DIC	364144	364144	364142	364134
WAIC	362517	362521	362514	362506

Note: Selected coefficients (mean of marginal posterior distribution). Poisson model with offset = `log(population)`. 95% credibility intervals.

Table S3: County Level Poisson Models, Prior Sensitivity, Men, CDC 2000-2014

	INLA Default	PC(1, .10)	PC(10, .10)	PC(10, 0.01)
Constant	−5.31	−5.31	−5.31	−5.31
	[−6.23; −4.40]	[−6.19; −4.44]	[−6.31; −4.32]	[−6.26; −4.37]
Income relative mobility	0.07	0.07	0.07	0.07
	[0.05; 0.09]	[0.05; 0.09]	[0.05; 0.09]	[0.05; 0.09]
Gini	0.02	0.02	0.02	0.02
	[0.01; 0.04]	[0.01; 0.04]	[0.01; 0.04]	[0.01; 0.04]
Random Effects				
SD observations	0.12	0.12	0.12	0.12
	[0.12; 0.13]	[0.12; 0.13]	[0.12; 0.13]	[0.12; 0.13]
SD age group	1.93	1.86	2.11	1.98
	[1.33; 2.64]	[1.32; 2.50]	[1.46; 2.90]	[1.40; 2.83]
SD counties	0.10	0.10	0.10	0.10
	[0.10; 0.11]	[0.10; 0.11]	[0.10; 0.11]	[0.10; 0.11]
SD states	0.06	0.06	0.06	0.06
	[0.05; 0.08]	[0.05; 0.08]	[0.05; 0.08]	[0.04; 0.08]
SD mobility by age	0.04	0.04	0.04	0.05
	[0.03; 0.05]	[0.03; 0.06]	[0.03; 0.06]	[0.03; 0.06]
SD gini by age	0.03	0.04	0.04	0.03
	[0.02; 0.05]	[0.02; 0.05]	[0.03; 0.05]	[0.02; 0.05]
DIC	391844	391854	391858	391850
WAIC	389682	389701	389714	389698

Note: Selected coefficients (mean of marginal posterior distribution). Poisson model with offset = `log(population)`. 95% credibility intervals.

1.4 Results Using Absolute Mobility

We run of the models of the paper using an absolute upward mobility score or "the mean rank (in the national income distribution) of children whose parents are at the 25^{th} percentile of the national parent income distribution" (Chetty et al. 2014, p. 7).

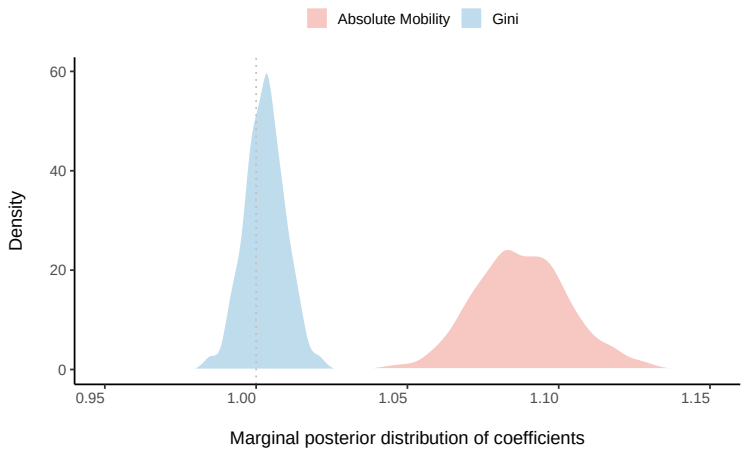
Although at the national level both the relative and absolute measure of mobility provide similar information, when studying small areas a child's rank in the national income distribution would be an absolute outcome because income in a given areas have little impact on the national distribution. We use a *permanent-resident* version of income mobility measures, that is, parents who stay in the same counties between 1996-2012. Note that children who grow up in a county may have moved out as adults.

Absolute upward income mobility ranges from 0 to 1, and higher values correspond to large income mobility. We multiply the absolute upward mobility score by -1 so that the interpretation and expected association of relative and absolute income mobility were the same (i.e., increases in income mobility and inequality is expected to rise mortality risk). The results are similar to the ones using relative income mobility, what is not surprising because the correlation between both measures is high (-0.70). Still, there are some differences is worth to mention.

IRM and GI ratios by age-group look more similar by gender than when using relative income mobility. The IRM male curve still look smoother than the female one, and the magnitude of the peak is greater for women (see Figure S6). Life expectancy differences are of the same order of magnitude (one year), but the decrease of differences at older ages is faster among males than females (see Figure S7). Relative differences are shown in Figure S8. Finally, there is also a much clear difference in the effect of IRM between Non-Hispanic White males and other groups (see Figure S9).

Figure S5: Posterior Distribution of $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ by Gender Model *Covariates* in Tables S4 and S5

(a) Women



(b) Men

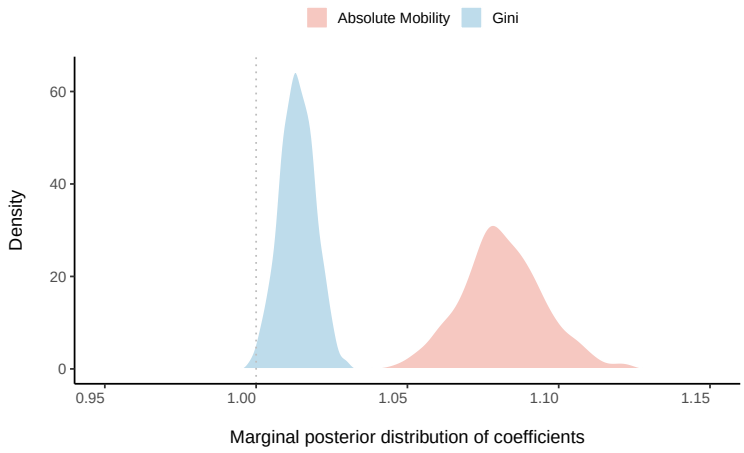


Table S4: County Level Poisson Models Absolute Mobility
PC prior $= Pr(\sigma > 1) < 0.10$, Women, CDC 2000-2014

	Baseline	Varying-Coefficient	Absolute mobility x Gini	Absolute mobility x Income	Covariates	Spatial
Constant	−5.81	−5.82	−5.82	−5.82	−5.82	−5.81
	[−6.70; −4.92]	[−6.70; −4.94]	[−6.73; −4.92]	[−6.69; −4.96]	[−6.70; −4.94]	[−6.71; −4.92]
Income absolute mobility	0.08	0.09	0.09	0.09	0.08	0.09
	[0.07; 0.08]	[0.06; 0.12]	[0.06; 0.12]	[0.06; 0.12]	[0.05; 0.11]	[0.06; 0.12]
Gini	0.00	0.00	0.00	−0.00	0.00	0.01
	[−0.00; 0.01]	[−0.01; 0.01]	[−0.01; 0.01]	[−0.01; 0.01]	[−0.01; 0.02]	[−0.01; 0.02]
Log income	−0.36	−0.36	−0.36	−0.36	−0.27	−0.25
	[−0.39; −0.34]	[−0.39; −0.33]	[−0.39; −0.33]	[−0.39; −0.33]	[−0.30; −0.24]	[−0.28; −0.22]
Absolute mobility x Gini			−0.00			
			[−0.00; 0.00]			
Absolute mobility x Log income				0.01		
				[−0.01; 0.03]		
Random Effects						
SD observations	0.13	0.11	0.11	0.11	0.11	0.11
	[0.12; 0.13]	[0.11; 0.11]	[0.11; 0.11]	[0.11; 0.11]	[0.11; 0.11]	[0.11; 0.11]
SD age group	1.91	1.92	1.91	1.92	1.90	1.89
	[1.46; 2.52]	[1.40; 2.69]	[1.40; 2.54]	[1.45; 2.62]	[1.41; 2.58]	[1.39; 2.61]
SD counties	0.10	0.10	0.10	0.10	0.09	0.12
	[0.10; 0.11]	[0.10; 0.10]	[0.10; 0.10]	[0.10; 0.10]	[0.09; 0.09]	[0.11; 0.12]
Phi counties						1.12
						[1.08; 1.18]
SD states	0.08	0.08	0.08	0.08	0.07	0.04
	[0.07; 0.10]	[0.07; 0.11]	[0.06; 0.10]	[0.06; 0.10]	[0.05; 0.08]	[0.03; 0.06]
SD mobility by age		0.06	0.06	0.06	0.06	0.06
		[0.04; 0.08]	[0.04; 0.09]	[0.04; 0.09]	[0.04; 0.09]	[0.04; 0.09]
SD gini by age		0.03	0.03	0.03	0.03	0.03
		[0.02; 0.04]	[0.02; 0.04]	[0.02; 0.04]	[0.02; 0.04]	[0.02; 0.04]
DIC	365596	364534	364527	364507	364447	364342
WAIC	363149	362588	362581	362473	362482	362406

Note: Selected coefficients (mean of marginal posterior distribution). Poisson model with offset = `log(population)`. 95% credibility intervals.

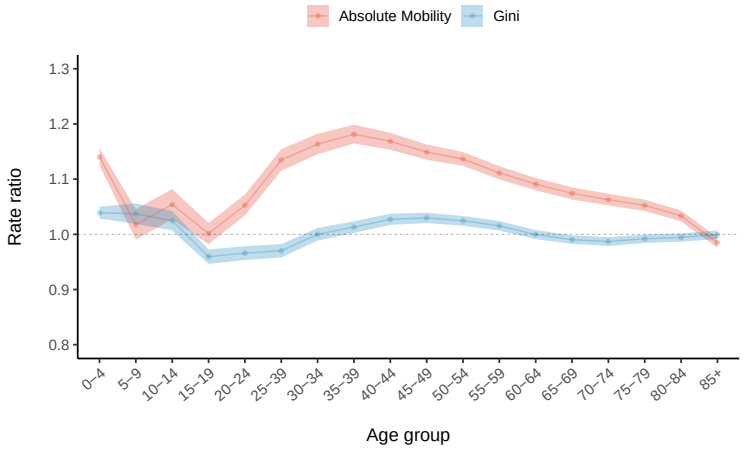
Table S5: County Level Poisson Models Absolute Mobility
PC Prior $= Pr(\sigma > 1) < 0.10$, Men, CDC 2000-2014

	Baseline	Varying-Coefficient	Absolute mobility x Gini	Absolute mobility x Income	Covariates	Spatial
Constant	−5.31	−5.32	−5.32	−5.32	−5.31	−5.31
	[−6.17; −4.46]	[−6.17; −4.47]	[−6.17; −4.47]	[−6.16; −4.47]	[−6.14; −4.49]	[−6.18; −4.43]
Income absolute mobility	0.08	0.09	0.09	0.09	0.08	0.09
	[0.07; 0.09]	[0.06; 0.11]	[0.06; 0.11]	[0.06; 0.11]	[0.05; 0.10]	[0.06; 0.11]
Gini	0.01	0.02	0.01	0.01	0.01	0.01
	[0.01; 0.02]	[0.00; 0.03]	[0.00; 0.02]	[0.00; 0.03]	[0.00; 0.03]	[0.00; 0.03]
Log income	−0.35	−0.35	−0.36	−0.36	−0.21	−0.16
	[−0.38; −0.32]	[−0.38; −0.32]	[−0.39; −0.33]	[−0.39; −0.33]	[−0.24; −0.18]	[−0.20; −0.13]
Absolute mobility x Gini			0.01			
			[0.00; 0.01]			
Absolute mobility x Log income				0.02		
				[−0.01; 0.04]		
Random Effects						
SD observations	0.14	0.13	0.13	0.13	0.13	0.13
	[0.14; 0.14]	[0.13; 0.13]	[0.13; 0.13]	[0.13; 0.13]	[0.13; 0.13]	[0.13; 0.13]
SD age group	1.82	1.83	1.82	1.82	1.93	1.84
	[1.37; 2.45]	[1.38; 2.41]	[1.36; 2.52]	[1.35; 2.54]	[1.37; 3.01]	[1.28; 2.49]
SD counties	0.12	0.11	0.11	0.11	0.10	0.16
	[0.11; 0.12]	[0.11; 0.12]	[0.11; 0.12]	[0.11; 0.12]	[0.10; 0.11]	[0.15; 0.17]
Phi counties						1.03
						[1.02; 1.07]
SD states	0.08	0.09	0.08	0.08	0.07	0.06
	[0.07; 0.11]	[0.07; 0.13]	[0.07; 0.10]	[0.07; 0.10]	[0.05; 0.09]	[0.05; 0.08]
SD mobility by age		0.05	0.05	0.05	0.05	0.05
		[0.03; 0.08]	[0.04; 0.08]	[0.04; 0.07]	[0.04; 0.07]	[0.04; 0.08]
SD gini by age		0.02	0.02	0.02	0.02	0.02
		[0.02; 0.03]	[0.01; 0.03]	[0.02; 0.03]	[0.02; 0.03]	[0.02; 0.03]
DIC	392500	391546	391536	391534	391460	391387
WAIC	389443	389107	389078	389076	388991	388923

Note: Selected coefficients (mean of marginal posterior distribution). Poisson model with offset = `log(population)`. 95% credibility intervals.

Figure S6: 95% Credibility Interval Posterior Distribution of $exp(\beta_m)$ and $exp(\beta_g)$ (see Equation 2.1) by Age Group Model *Covariates* in Tables S4 and S5

(a) Women



(b) Men

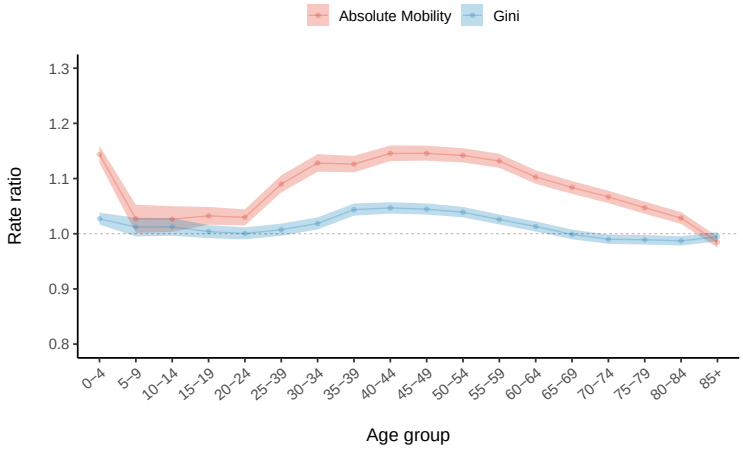
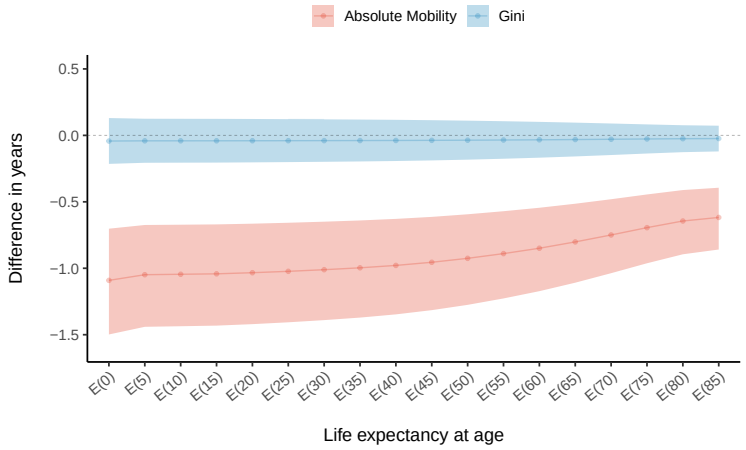


Figure S7: 95% Credibility Interval of Predicted LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables S4 and S5

(a) Women



(b) Men

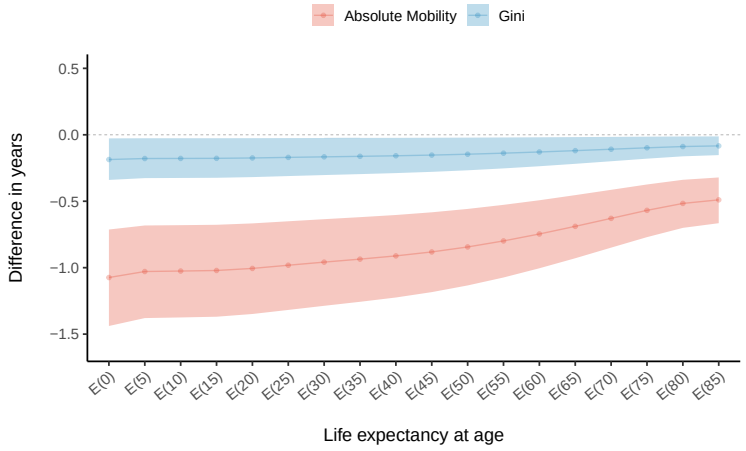
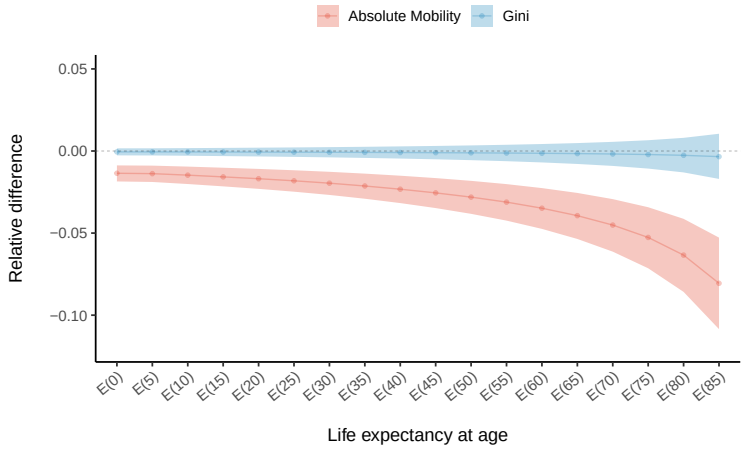


Figure S8: 95% Credibility Interval of Predicted Relative LE Differences by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables S4 and 2.2

(a) Women



(b) Men

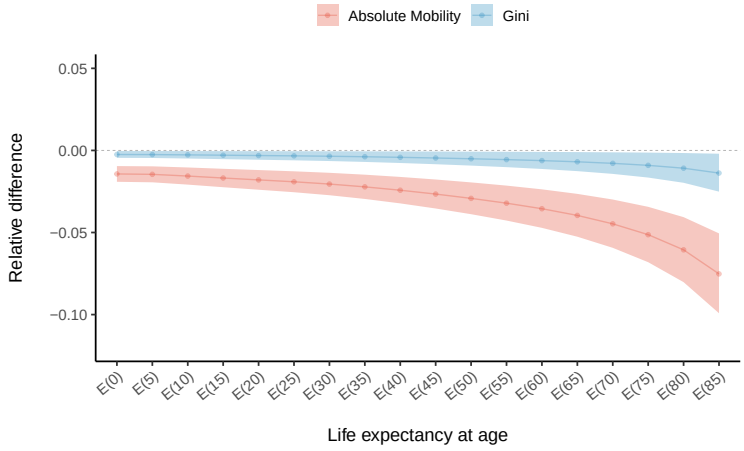
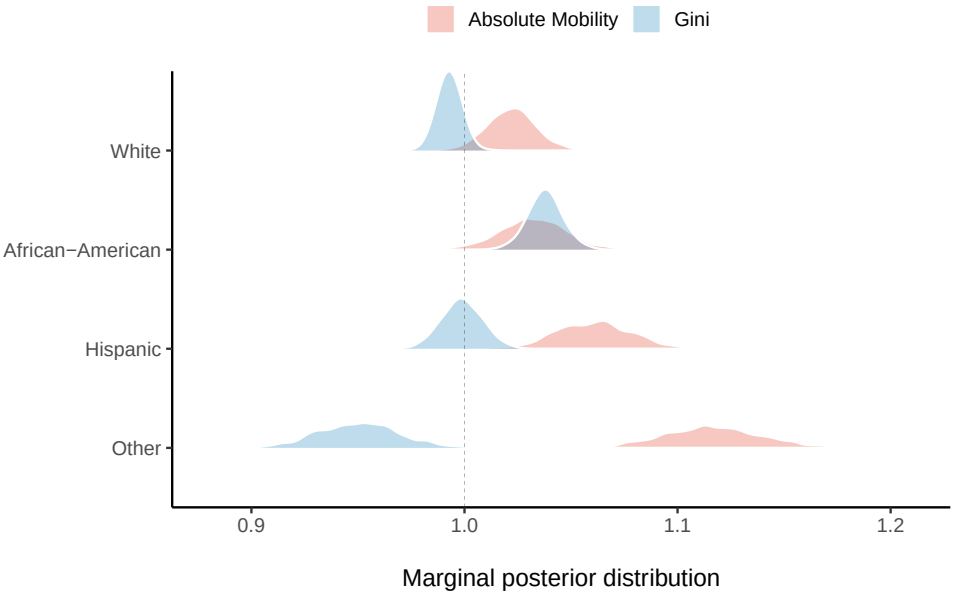


Figure S9: Posterior Distribution $exp(\beta_m)$ and $exp(\beta_g)$
Model *Covariates*

(a) Women



(b) Men

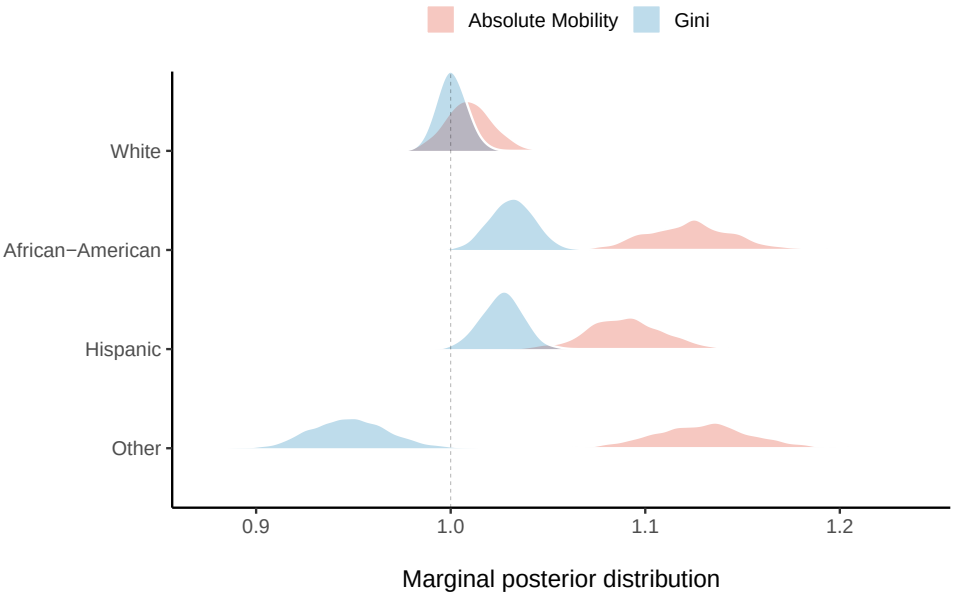
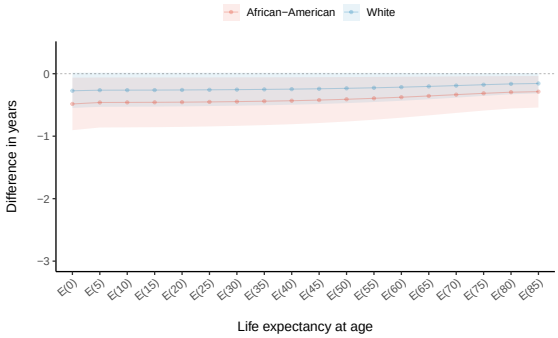
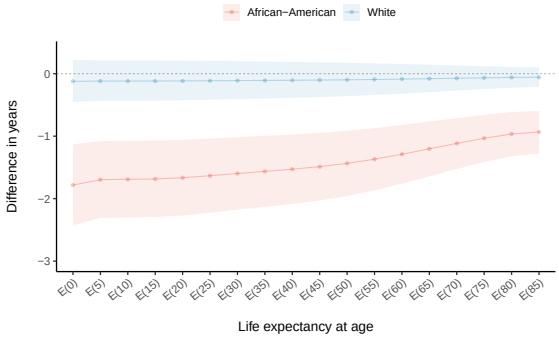


Figure S10: 95% Credibility Interval of Predicted LE Differences
by Age Group and Race/Ethnicity, Increase in One Standard Deviation

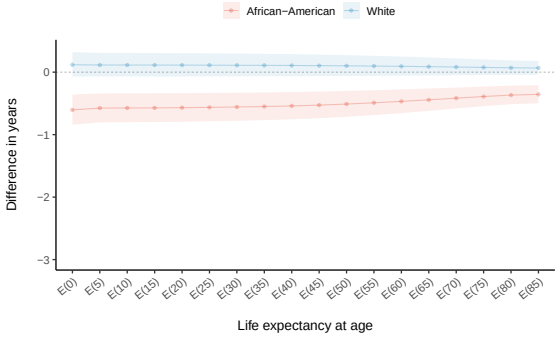
(a) Women - Absolute Mobility



(b) Men - Absolute Mobility



(c) Women - Gini



(d) Men - Gini

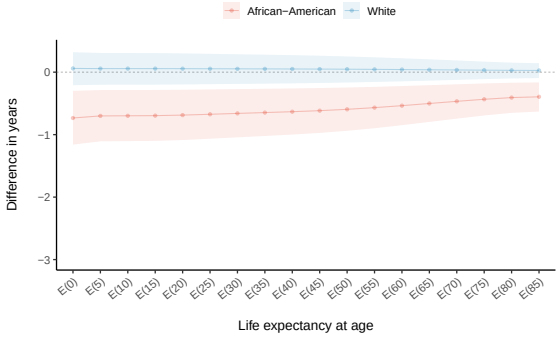
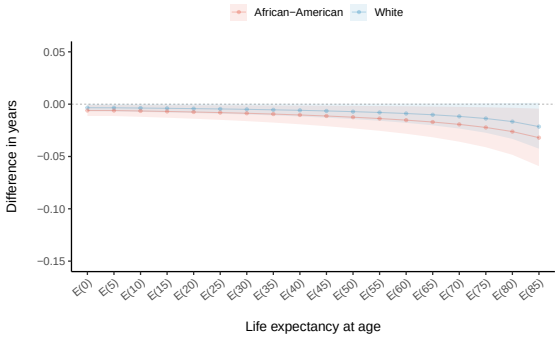
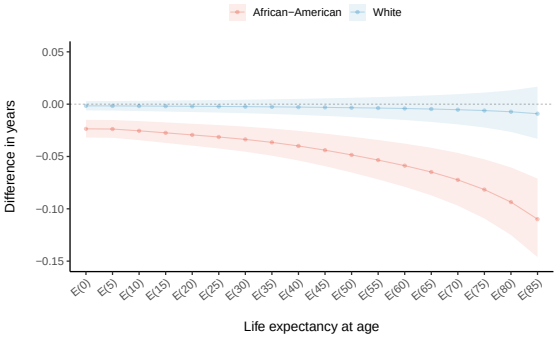


Figure S11: 95% Credibility Interval of Predicted LE Relative Differences by Age Group and Race/Ethnicity, Increase in One Standard Deviation

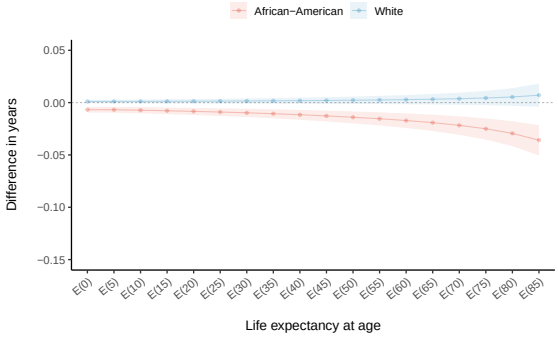
(a) Women - Absolute Mobility



(b) Men - Absolute Mobility



(c) Women - Gini



(d) Men - Gini

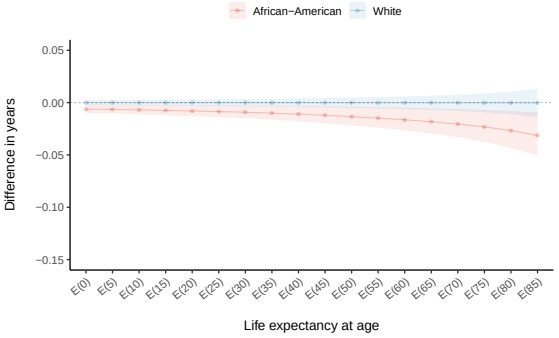
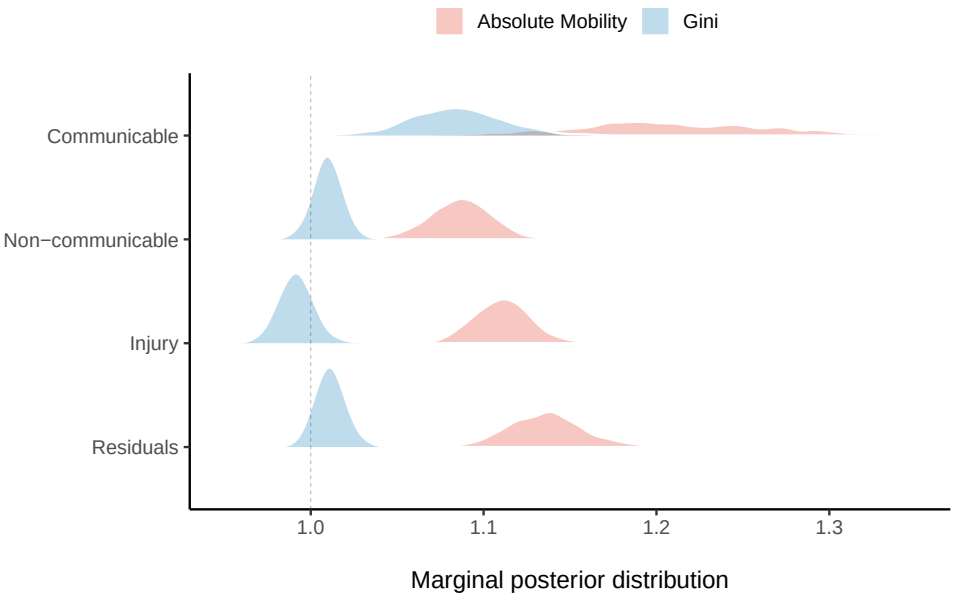
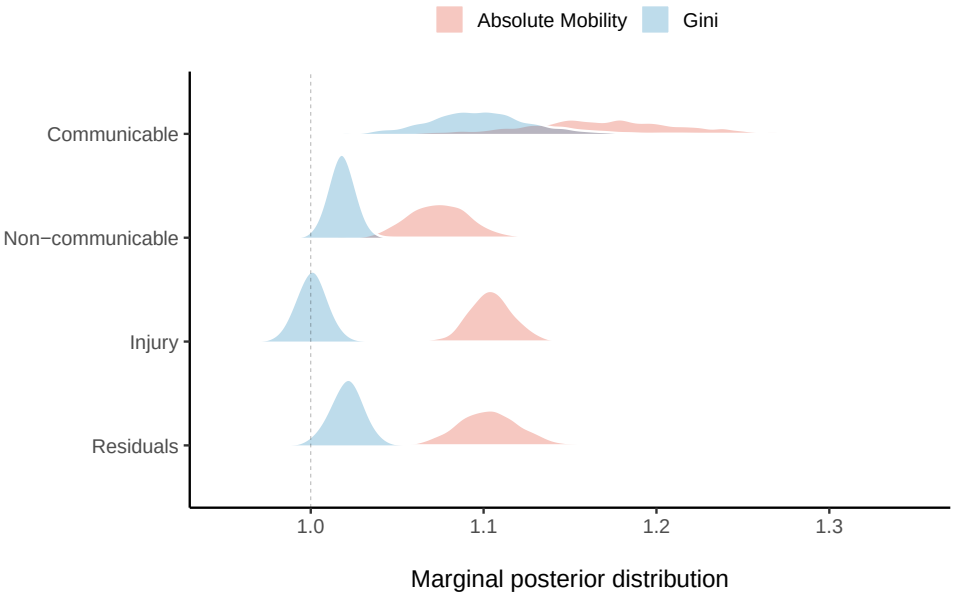


Figure S12: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta \text{Gini})$ by Cause of Death and Gender

(a) Women



(b) Men



1.5 Cause of Death Coding

Table S6: Code for Causes of Death, CDC

Code CDC Database	Cause Title and ICD-10 Codes
Group 1	
001	Tuberculosis (A16A19)
002	Syphilis (A50A53)
003	Human immunodeficiency virus (HIV) disease (B20B24)
027	Influenza and pneumonia (J10J18)
Group 2	
004	Malignant neoplasms (C00C97)
005	Malignant neoplasm of stomach (C16)
006	Malignant neoplasms of colon, rectum and anus (C18C21)
007	Malignant neoplasm of pancreas (C25)
008	Malignant neoplasms of trachea, bronchus and lung (C33C34)
009	Malignant neoplasm of breast (C50)
010	Malignant neoplasms of cervix uteri, corpus uteri and ovary (C53C56)
011	Malignant neoplasm of prostate (C61)
012	Malignant neoplasms of urinary tract (C64C68)
013	NonHodgkin's lymphoma (C82C85)
014	Leukemia (C91C95)
015	Other malignant neoplasms (C00C15, C17, C22C24, C26C32, C37C49, C51C52, C57C60, C62C63, C69C81, C88, C90, C96C97)
016	Diabetes mellitus (E10E14)
017	Alzheimer's disease (G30)
028	Chronic lower respiratory diseases (J40J47)
035	Sudden infant death syndrome (R95)
029	Peptic ulcer (K25K28)
030	Chronic liver disease and cirrhosis (K70,K73K74)
031	Nephritis, nephrotic syndrome, and nephrosis (N00N07,N17N19,N25N27)
032	Pregnancy, childbirth and the puerperium (O00O99)
033	Certain conditions originating in the perinatal period (P00P96)
034	Congenital malformations, deformations and chromosomal abnormalities (Q00Q99)
036	Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified (excluding sudden infant death syndrome) (R00R94,R96R99)
018	Major cardiovascular diseases (I00I78)
019	Diseases of heart (I00I09,I11,I13,I20I51)
020	Hypertensive heart disease with or without renal disease (I11,I13)
021	Ischemic heart diseases (I20I25)
022	Other diseases of heart (I00I09,I26I51)
023	Essential (primary) hypertension and hypertensive renal disease (I10,I12)
024	Cerebrovascular diseases (I60I69)
025	Atherosclerosis (I70)
026	Other diseases of circulatory system (I71I78)
Group 3	
038	Motor vehicle accidents (V02V04, V09.0, V12V14, V19.0V19.2, V19.4V19.6, V20V79, V80.3V80.5, V81.0V81.1, V82.0V82.1, V83V86,V87.0V87.8, V88.0V88.8, V89.0,V89.2)
039	All other and unspecified accidents and adverse effects (V01, V05V06, V09.1, V09.3V09.9, V10V11,V15V18, V19.3,V19.8V19.9, V80.0V80.2, V80.6V80.9, V81.2V81.9, V82.2V82.9, V87.9, V88.9,V89.1, V89.3,V89.9, V90X59, Y40Y86, Y88)
042	All other external causes (Y10Y36, Y87.2, Y89)
040	Intentional self-harm (suicide) (*U03, X60X84, Y87.0)
041	Assault (homicide) (*U01-*U02, X85Y09, Y87.1)
Group 4	
037	All other diseases (Residual) (A00A09, A20A49, A54B19, B25B99, D00E07, E15G25, G31H93, I80J06, J20J39, J60K22, K29K66, K71K72, K75M99, N10N15, N20N23, N28N98)

Chapter 3

County Income Mobility and Individual Health Behaviors, Health Status and Mortality

The growing life expectancy gap by geography and income represents a fundamental challenge for health policy in the United States. The best performing counties in the U.S. have life expectancies that are now 20 years greater than the poorest performers. Recent work by Chetty and colleagues in the United States (Chetty et al. 2014) demonstrates that differences in life expectancy (at age 40) between the richest and poorest quartiles of the income distribution grew from 9 years to about 11 among men and from 5.2 years to 6.6 years among women. These gaps are substantial – representing 25 percent of remaining life expectancy among men and 13% among women.

Wodtke et al. (2011)

Designing interventions to ameliorate these gaps requires understanding their origin. However, results from multiple recent analyses suggest that neither access to medical care or socioeconomic factors fully explain observed geographic or income disparities in longevity. Consequently, it is important to identify other drivers.

Social (or economic) mobility – defined as the ability of individuals to exceed the income of their parents – may play an important role in explaining these disparities. Low mobility may harm health by raising despair and diminishing the motivation to engage in health behaviors. Economic mobility is distinct from income inequality – individuals living in areas characterized by similarly high degrees of income inequality may experience different probabilities of social mobility - and therefore may have different implications health outcomes. While the association between income inequality and health has been studied as part of a 20-year literature, recent work suggests that its contribution disparities in longevity may be small. In contrast, the health consequences of economic mobility have been understudied. This gap in the literature is particularly salient given

emerging evidence of falling social mobility in the U.S. - among the same birth cohorts currently experiencing divergence in their life expectancies.

We used newly available, local-area level data on income-specific life expectancy and social mobility to assess the relationship between economic mobility and longevity by gender and income quintile. We also assessed the extent to which differences in economic mobility may contribute to income gaps in longevity.

3.0.1 Goal and Questions

The goal of this chapter is to empirically assess the plausibility of the contextual effect of income mobility on health behaviors and mortality at the individual level, and to examine some of the mechanisms proposed by **?** and Venkataramani et al. (2015, 2016). Thus, I extend the analyses from my previous chapter by using individual data.

The key original questions I examine in this chapter were:

1. What is the direction and size of the association of income mobility at the community level (i.e,) with individual mortality, subjective and mental health, after adjusting for income, income inequality and other confounders at the individual and community level?Are the effects of income mobility sizeable and in the expected direction? How big/small is the magnitude of the mobility effect relative to individual income and income inequality?
2. What is the relationship between income mobility at the community level and individual health behaviors and conditions such as smoking, physical activity, obesity, after adjusting for income, income inequality and other confounders at the individual and community level?
3. How do these effects vary at the state and commuting zone level? How do these effects vary by income, income inequality, age, race, and gender?
4. Is there any link between income mobility and some of the mediators proposed by the theory, namely, cognitive and non-cognitive skills, outlooks and expectations?
5. After adjusting for early upbringing (type of family, parental background), early cognitive and non-cognitive traits, health behaviors and status, what is the magnitude and direction of the remaining income mobility effect on current risky behaviors, subjective health, and mortality?

I restricting these questions to something more specific.

3.1 NLSY 97 setup

The sample of NLSY 97 corresponds to a cohort of about 8,000 people born between 1980 and 1984. That is the cohort that Chetty uses to estimate income mobility.

My idea here is to create a model to estimate the effect of being exposure to a given income mobility regime at the county level on health and health behaviors. I am focusing more on the getting an effect of exposure than examining mechanisms.

More specifically the dependent variables will be:

1. Self-report heath status
2. Substance use (alcohol, smoking, marijuana)
3. Health conditions / BMI
4. Mental health

These outcomes will be measures at the end of the study (2013-2015), that is around when respondents are about 30-35 years old. The **key dependent** variable will be the average exposure to a give mobility regime between ages 12 and 20.

$$\frac{\sum_{i=12}^{20} \text{income mobility}_i}{8}$$

Twelve years old is the earliest geographic information I have, and I set 20 years as an upper limit, because I am interested in assessing somewhat early exposure. The average number of moves is 1.7, and around 60% of respondents move at least one.

To deal with selection, I would use a marginal structural model so that I adjust (at least partially) for selection into counties and dropout.

I would create a model to predict the mobility regime a respondent is at time t using covariates such as:

1. Household income previous year
2. Outcome variable from age 12 to 20
3. Gender
4. Race
5. Parent education
6. Personality traits at baseline

I have to think a bit more about other covariates I can add, but this would be a baseline model. This process will be modeled assuming autocorrelation (e.g., AR1). I will created stabilized weights to then run a model that estimates the effect of income mobility (average exposure) in the outcome variable.

Once I get this, I can iterate, adjust missing values and create alternative models.

Table 3.1: Income mobility and inequality exposure models (NLSY 97)

	Self-reported healh	BMI	Depression	Smoking	Day smoking last month
Income relative mobility average exposure	0.08	0.39	0.01	0.01	0.08
	(0.07)	(0.22)	(0.02)	(0.07)	(0.06)
Gini average exposure	−0.17	−0.49	−0.04	−0.14	−0.07
	(0.10)	(0.25)	(0.04)	(0.12)	(0.07)
Observations	8984	8984	8984	8984	8984

Note: Each row represents a model.

3.1.1 Variables

Variable	Waves	Type
General health (self-reported)	1997-2015	Outcome
Mental health (5 items)	2000-2015	Outcome
BMI	1997-2015	Outcome
Smoking	1997-2015	Outcome
Gender	1997	Covariate
Birth date	1997	Covariate
Parents highest completed grade	1997	Covariate
Age biological mother when R was born	1997	Covariate
Living with both parents	1997	Covariate
Household income	1997-2015	Covariate
R Marital status	1997-2017	Covariate
Lived hard times	1997	Covariate
R Education	1997-2015	Covariate
Optimism scale	1997	Covariate
Math - Verbal Test	1999	Covariate
R Job status	1997-2017	Covariate

3.2 Tables and Figures

Methodological Supplement

The code used to create the database and run the models and plots is available at: `https://github.com/sdaza/dissertation/tree/master/ch03`.

Chapter 4

A Generative Model for Income Mobility and Mortality

The relationship between inequality, income mobility, and health is not simple. As discussed in previous research, the processes generating a link between health, income mobility and inequality involve individual, contextual, spatial, and reciprocal effects, feedback loops, and cumulative processes. As a first condition, the assessment of the consequences of income mobility for health requires a clear definition of exposure to the mobility regimes in a context where residential mobility is observed, and neighborhood characteristics might independently affect health (**?**). For instance, a person's socioeconomic position could contribute to the type of neighborhood in which she/he can afford to live. At the same time, the individual socioeconomic position is a product of the types of income-generating opportunities afforded by the neighborhood socioeconomic achievement, and neighborhood characteristics might impact the access to facilities that contribute to better health. Early exposure to poor socioeconomic environments might also impact health later in life.

These processes are usually difficult to grasp and understand using statistical models as they tend to produce the same summaries from data generated by different mechanisms.[1] This is the so-called the *inverse problem* and consist of the challenge of figuring out causes from observations when many different causes can produce the same evidence (**?**). In this paper, we follow a different approach from statistical modeling and develop a generative model of the intergenerational connection between income mobility and health. While statistical models run in reverse of generative models - they assume observed behavior (the data) and *simulate* strategies (parameters) - generative models assume strategies (causal connections and mechanisms) and simulate observed behavior, forcing us to express our ideas and theories formally. In the case of being wrong, gener-

[1] A linear regression model, for instance, is just an attempt to learn about the mean and variance of some measurement, using an additive combination of other measurements. Different mechanisms and processes can generate similar mean and variance summaries.

ative models will be wrong in a precise way, which is useful because that gives us hints about what cause and effect relationships are problematic or at fault.

The goal of this paper is to formalize some of the ideas and mechanisms proposed in previous research and examine their behavior under different conditions. Simulation models have the *potential* of being useful tools to understand and learn from complex systems by offering simplified representations of the mechanisms that generate and preserve health inequalities (**?**). Following that tradition, we propose a computer simulation model to specify the mechanisms discussed in the literature and assess the consequences of the socioeconomic mobility regime for health, and examining the behavior of the mechanisms proposed by exploring *what-if* questions and scenarios.

4.1 Income mobility, inequality and health

We start by briefly discussing potential causal mechanisms that might generate an association between place's income mobility and health. We are particularly modeling the relation between place's income mobility and health/mortality, that is, the linkage between a contextual characteristic (place's income mobility) and an individual trait such as health and mortality. When discussing income mobility we are referring to prospects for upward mobility afforded by, which follows the interpretation in Chetty's work that economic opportunity is a characteristics of places (**?**). Our point of view is that those prospects — for instance, measured as the either the correlation between the parents' position on the income distribution (not income itself, but the rank of income) -– independently affect health and mortality.

Thus, we do not focus on the connection between individuals' lifetime income mobility experiences and their adult mortality (intra-generational mobility) – a problem studied in a large and distinguished body of research (Chandola et al. 2003; Illsley 1955; Fox et al. 1982; Blane et al. 1999)[2] – or individual inter-generational changes of income. Instead, we concentrate in the link between an *aggregate* property of the stratification system, on one hand, and individual experiences, on the other. It is, of course, possible that individuals' experiences of occupation or SES mobility are also influenced by the prevailing aggregate regime of income mobility. Indeed, these experiences may be one of many other pathways through which aggregate income mobility and individual mortality are related. Thus, we argue that an association between places' income mobility and mortality could exist if communities with higher income mobility host social and economic environments that reduce mortality risks relative to communities with lower income mobility, *independently of the income level and income inequality*. Individuals and groups who occupy the most vulnerable and exposed social positions within unequal communities may be comparatively better off when they face advantageous income mobility prospects than when they do not. Just as individuals who command lower incomes in communities with more equitable income distributions may experience better health than individuals with similar incomes in societies with higher

[2]The bulk of this literature is concerned with the long run impact of early occupational (career) shifts or the short run effects of late occupational (career) shifts. This work is based on empirical research that focuses on patterns of relationships between individuals occupation (or SES status broadly conceived) at an early point in their adult life and subsequent older adult health and mortality.

income inequality, so too could individuals and groups that occupy lower ranked positions in societies with higher income mobility enjoy better health than counterparts in societies with more rigid stratification systems.

In theory, communities could be classified in different combinations of income mobility and inequality, where each combination is characterized by a health and mortality profile. The standard conjecture is that indicators of mortality and health will be more beneficial in communities with less inequality than in those with high inequality. The new conjecture is that at a given level of income inequality, better health and mortality conditions will be experienced by members of communities with higher income mobility.

We propose four pathways that might produce a link between income mobility and mortality:

1. *Residential mobility, adult health and mortality*: As stated above, an association between aggregate income mobility and individual health and mortality may be the outcome of a composition effect, namely, places with higher income mobility contain a population composition biased toward individuals who experience mobility (and their health and mortality consequences). In this case, the association between the aggregate property of the stratification system and individual experiences of health and mortality reflects the influence of individual residential mobility patterns (and associated selection processes).

2. *Individual early experiences*: A large body of literature on health and mortality disparities demonstrates that SES (income, education) health and mortality gradients are pervasive, persistent and, as of recent, increasing everywhere in high-income countries (**??**). Further, there is evidence that early conditions and upbringing of individuals matter greatly for adult health and mortality disparities (Palloni et al. 2009; **?**). Thus, some of the health differentials between men in low and high ranking positions initially attributable to chronic stress among those in subordinate positions (**??**) may be rooted in antecedent health conditions sculpted early in life (**?**). If early conditions are influential for SES health and mortality disparities, they may also be influential as vehicles that establish relations between income inequality, income mobility, and adult health and mortality.

   During early stages of socialization individuals experience sensitive and critical windows for the acquisition of cognitive and non-cognitive abilities that are the foundation of skills acquired later in life (**????**). Some of these traits involve the development of outlooks and attitudes that influence investments in skill acquisition and health, including propensities to adhere health-related behaviors. Thus, behaviors critically associated with modern chronic illnesses, such as smoking uptake and desistance, alcohol consumption, substance abuse, choices of diet and physical activity, are in part determined by capabilities sculpted early in life. Early avoidance of unhealthy behaviors has large payoffs in adulthood because these behaviors are closely related and reinforce each other, the physiological and psychological damage they produce are accumulated over time, and they all are strongly non-reversible. Early adoption of healthy behaviors is facilitated by socialization that emphasizes robust future outlooks, self-confidence, and self-reliance, beliefs in the neutrality and fairness of social reward allocation systems, hopefulness and optimism, and incentives to succeed. These are all traits that reduce time discounting so that the addition of one year of healthy life in

the future of an individual is endowed with significant rewards and returns (Grossman 2000, 1972).

We know from empirical research that negative affect, chronic stress, subordination, bleak future outlooks associated with poverty lead to increases in time discounting (**?**). Higher time preferences favors resistance to the adoption of behaviors that may yield immediate rewards but are health-damaging and discourage those that have a more distant and elusive pay-off but are health-preserving (**??**).

This mechanism alludes to influences that shape environments during critical stages of individuals' upbringing. The conjecture is that a places' income mobility regime is powerful enough to shape those environments. But so is an individual's ancestral income mobility experiences, particularly parental and possibly grand parental mobility. Strictly speaking these are two very different mechanisms that can be properly identified only if we simultaneously observe both the influences of a place's aggregate income mobility and individuals familial income mobility experiences.

3. *Community endowments*: Communities with low-income mobility may distort opportunities and incentives, reinforce unequal allocation of favorable traits, undervalue public institutions that contribute to the formation of skills with a high wage premium and, many of them, support non-meritocratic reward allocation strategies. These community properties directly influence the suite of opportunities available to individuals and shapes the way parents socialize children and favor (discourage) the adoption of positive outlooks and the value of skill acquisition. Rigid or weak income mobility fosters individual hopelessness, despair, mistrust, disbelief in a level playing field for all, weaken aspirations and, more generally, diminish the value of adoption of attitudes and behaviors that promote good health.

4.2 MIA: Mortality and Income Mobility Agent-Based Model

We develop an agent-based model (ABM), called the *Mortality and Income Mobility Agent-Based Model* (MIA) that generates intergenerational data to study the connection between income mobility, inequality, residential segregation and mortality. The purpose of the model is, through simulation, to formalize some initial ideas about how income mobility might affect health outcomes, and under which conditions we could recover estimates of those effects (**?**). This is just a first effort to formalize a set of ideas about the implications of income mobility on health, and consequently, a first step in developing further theoretical exposition of the link between aggregate income mobility and health [3] It is important that any ABM used to test SIENA not follow its same actor behavior assumptions, as we already know the real processes do not follow SIENA's algorithm. This is what we mean by misspecification: There is no direct translation between the generative processes of ISA and SIENA, so any SIENA specification would be just an approximation to the *actual* mechanisms, as occurs when analyzing real data. The question is whether SIENA is able to make accurate statistical inferences given this known difference.

[3] As (**?**) define it: *Characterising (or assessing) hypotheses about the general behaviour of a set of mechanisms using a simulation.*

While agent-based modeling cannot by itself solve the empirical estimation and identification problems of the selection and influence effects, it offers a unique opportunity to rigorously implement theoretical and empirical assumptions on these processes. By construction, all group and individual level variables defining agent attributes are perfectly known (i.e., there is no unobserved heterogeneity). Also, the way in which agents are linked and the composition of their local neighborhood are also completely transparent.

Our ABM is not intended to perfectly represent the actual processes at play, but provides a simplified analogy of potential selection and social influence mechanisms. Although these simplified mechanisms might seem plausible, they are still artificial. If SIENA performs well using a different but simplified version of homophilous selection and social influence, we can argue SIENA's estimates are robust to particular levels of misspecification (those given by our ABM). This does not confirm SIENA's ability to produce valid selection and influence estimates in the *real world*. It does, however, improve our confidence in SIENA estimates, especially if our ABM is empirically appealing. If SIENA performs poorly, we can argue its ability to estimate selection and influence is limited and sensitive to the misspecifications given by our ABM. The next question would be which assumptions seem to be the most critical, and how likely they are met in reality.

With these assumptions in mind, we design ISA following four key design concepts. First, we follow the *social circles* model proposed by **??** to define the social network dynamic. That model, inspired by Georg Simmel's theory (**?**), adapts the idea that people are embedded in *social circles*. It generates a social network by giving each agent a social reach (a radius r defining its social circle) of alter agents, and produces personal (ego-centric) networks that are limited in size (low network density), vary in size between individuals, display right-skewed distributions, high clustering (i.e., communities), and are positively assortative by the degree of connectivity (i.e., well-connected agents tend to be connected to other well-connected agents).[4] Second, homophilous selection occurs because agents prefer alters that are more similar in behavior: when creating a tie, they choose the agent with the most similar behavior value. Third, we model social influence using a very simple mechanism: agents choose an *influencer* among their connections and adjust their behavior to be incrementally more similar.[5] Finally, to explore the consequences of heterogeneity for SIENA's estimates, we include two important sources of heterogeneity in our ABM: social reach (size of the radius to select peers) and behavioral change independent from social influence.

4.2.1 Model Description

The purpose of our model is to simulate a simplified data-generating process to examine the interaction between income mobility and mortality.

In order to allow agents to experience influence and selection, agents need to track their be-

[4] We do not use network models such as small world and preferential attachment where connections are established or broken exogenously, mostly because they do not display important features observed in real social networks (**?**). Our focus is on modeling social influence through social networks generated from (endogenous) micro-behavior of agents that reproduces empirical patterns.

[5] Following **?**, we also implemented an ABM using opinion dynamics, but that lead to too much opinion convergence, and we finally discarded that approach.

havior and their social connections over time. Therefore, the primary characteristics of agents are their *behavior* (measured using a continuous scale from 0 to 1), and social contacts (connections that form their out-degree). Secondary characteristics are position in space, and social circle radius (to capture new potential alters or destroy existing ties).

- **Segregation**: This is a relatively well-studied mechanism that generate inequality at the aggregate level. The famous Schelling's model (**?**) shows how a simple rule of satisfaction generates residential segregation even when agents are tolerant to live in neighborhood with people of different *color*. The same mechanism can be extended to characteristics such as income, education and health. Although Schelling's model is dynamic (agents move to different neighborhood until equilibrium is reached), the population remains constant (nobody is born or die), and *color* (a key characteristics of agents) does not change. Assuming that we are study income inequality, this mechanism alone will generate inequality but not mobility: people would not change their income, nor next generations would be observed. This mechanism would generate income mobility differences as soon as agents change their income, for instance, based on the characteristics of their neighborhood.
- **Neighborhood constrains income opportunities**: Income mobility can be implemented simply by conditioning the success of an agent on where she/he lives. If the opportunities of an agent are strongly defined by his environment (for instance, early years), we would say that society is rigid. If agents earn income independently of their initial residential conditions, we would say that society is flexible. This mechanism does not consider family transmission of skills, cultural capital, or habits, but only neighborhood's constrains. Mobility and inequality would be, I guess, strongly related due to reinforcing loops: those who are at the bottom of the income distribution would also have less opportunities in the social ladder what will increase inequality and reduce mobility over generations. An alternative to this approach would be to specify opportunity constrains based on family income.

This will be the baseline model where I can switch on/off these mechanisms or change their importance and see how aggregate measures of inequality and income mobility behave. The next step is to connect income, inequality, and income mobility to health behaviors (let's say for now, smoking), and mortality.

I have to write a bit more here. Just to have an idea of what to do.

Agent actions include movement, creation/destruction of ties, and interaction. Regarding movement, each agent has the same step size d_{move} so that at a rate r_{move}, they choose a new random location d_{move} away from their current position, moving to it at a constant speed of 100 units per model-second. This movement procedure is important to control for network saturation in our model.

In parallel, at a rate defined by $r_{network}$, agents perform a network decision. Figure **??** shows the action chart of network decisions (creation or destruction of ties). For each such decision, agents either create a new tie (with probability p_{create}), or break a current tie. When agents choose to create a new tie, they choose either the most similar agent by behavior (with probability p_{sel}), or a

random agent within their social circle at their current position (d_{circle}). When choosing the most similar agents, they can select either from within their social circle (with probability p_{inside}) or from the whole agent population. This latter rule operationalizes two types of homophily distinguished by **?**: baseline and inbreeding. While *baseline homophily* poses constraints to the local social world of individuals who incidentally will establish connections with people who do not match their behavior (shortage of alter candidates), *inbreeding homophily* describes the tendency to choose similar peers above the opportunity set (e.g., social circle), recruiting alters from wider surrounding of ego. When breaking a tie, in turn, agents define, with probability $p_{outside}$, if the tie to break will be randomly selected from alters exclusively outside the social reach d_{circle} or from any of their connections.

At a rate defined by r_{int}, agents become influenced by another agent. This is done by moving their behavior value closer to that of a random agent from their social connections. To determine their new behavior value, b_e^*, agents use the simple influence update rule:

$$b_e^* \leftarrow b_e + \alpha(b_a - b_e)$$

where b is behavior of the (e)go or the (a)lter, and α is the behavior change rate.

Agents are also allowed to make behavior and network changes independently from influence/selection so that dynamic behavior and networks can still observed when influence or selection are disabled during the experiments. Independent behavior change is controlled by a separate rate, r_{ib}. At each independent behavior change choice, agents use a *behavior tendency*, β, to decide how to internally change behavior. This tendency is a binary value, selecting between two different internal patterns (triangular distributions): one that increases behavior on average, and one that decreases on average. Independent network change, on the other hand, is controlled by p_{sel}, the probability that an agent will choose selection versus random choice within the social circle when updating their network. At the end, the resulting social network from this implementation is a dynamic and directional graph.

The initial population is of 200 agents, randomly placed in a 500x500 unit toroidal space. Model time is continuous, meaning there is no fundamental clock "tick"[6].

The initial social network is created by connecting each agent to two other randomly chosen agents. Both the social circle and initial behavior are defined using uniform distributions. For each agent, the chance of an increasing versus decreasing behavior tendency is 50%. Table **??** describes all the model parameters with their initialization values.

To generate the output data, the model simulates a measurement, outputting location, social network, and agent behavior data for each agent every $T_m = 30$ days. The model runs for $T = 300$

[6]Rather than specifying a smallest time increment (a "tick"), AnyLogic uses an event-driven scheduler. For example, an event scheduled for 10 model-seconds in the future will always occur before one scheduled for 11 model-seconds, and after one scheduled for 9 model-seconds. All relevant model parameters are updated at each event. This approach improves model efficiency and accuracy (since only changes are simulated).

days. We implement ISA using AnyLogic Personal Learning Edition 8.5 [7]

4.2.2 ISA Scenarios, Calibration and Verification

We generate data for six ABM scenarios. Table **??** displays their differences. While ISA calculates a strength value for each influence and selection, we have not defined a mechanism to either scale the strength of influence or selection, nor have we defined a method to calculate a strength comparable to SIENA (i.e., there is no a direct translation of ISA's parameters and SIENA coefficients.)[8] As a result, when comparing ground-truth with SIENA predictions, we measure simply presence or absence. Therefore, we exclude scenarios where selection and social influence are at play simultaneously. Table **??** displays descriptive statistics of the outcomes of 100 replications of each scenario (details of the experimental design are discussed in 4.3).

The first scenario (B) corresponds to our baseline where both (homophilous) selection and influence are disabled, and there is heterogeneity in agents' *social circles*. This was done to ensure the model does not artificially generate non-zero selection or influence (measured by behavioral-network autocorrelation). Table **??** provides (network) statistics that characterize each scenario (averaged over four waves). Scenario B shows a network density of 0.03, average degree of 10.28[9], and high levels of reciprocity and transitivity.

To measure for any unintended selection or influence pressures, we use one of the most popular standardized measures for network autocorrelation, Moran's I:

$$I_M = \frac{n \sum_{ij} x_{ij}(z_i - \bar{z})(z_j - \bar{z})}{\left(\sum_{ij} x_{ij}\right)\left(\sum_{i}(z_i - \bar{z})^2\right)}$$

where n is the number of agents, z is the variable of interest, and x is an element of a matrix of network weights. This coefficient is based on cross-products of behavioral scores of relational partners. Values close to zero indicate that relational partners are not more similar than one would expect under random pairing, while values close to one indicate a very strong network autocorrelation. As expected, for scenario B, the Moran's I coefficient is zero both for behavior and the size of social circles (radius). That is, there is no emergent property in our ABM that generates behavior or radius autocorrelation other than the selection and influence mechanism.

In order for SIENA estimates to be valid, SIENA' authors indicate that the network must have a minimum amount of network stability over time. SIENA's authors measure this using Jaccard index, that is, Jaccard distances between time-successive networks:

[7] All the files and code to reproduce our analysis are available at https://github.com/sdaza/dissertation/tree/master/ch04.

[8] It is possible, in principle, to do so, but this is left for future work.

[9] The degree distribution is also positively skew (not shown).

$$J = \frac{N_{11}}{N_{01} + N_{10} + N_{11}}$$

where N_{hk} is the number of tie variables with value h in one wave and value k in the next wave (**?**). All ISA scenarios in Table **??** satisfy the levels of network stability recommended by SIENA's authors ($J \geq 0.30$). Furthermore, our behavior stability measures look similar to those we find in empirical applications of SIENA (**??**).

The ISA scenarios I and S enable influence or selection, respectively. The influence mechanism does not directly affect network dynamics, so network statistics between scenarios B and I are almost identical, as expected. This is not the case for scenario S, where ties are being defined through a selection process while behavior remains unaffected. As expected, network statistics are different from scenario B, slightly increasing the degree and reducing reciprocity, transitivity and the Jaccard index. In scenario I, Moran's coefficient for behavior is 0.22, and behavior stability is lower than scenario B because behavior is changing in a systematic way and not only through a random process (behavior tendency). For scenario S, Moran's coefficient is 0.22, the same as scenario I but less noisy (i.e., smaller standard deviation). Importantly, both influence and selection mechanisms independently increase network autocorrelation.

In order to examine the consequences of agent heterogeneity, scenarios, B+CR, I+CR and S+CR (CR stands for *constant radius*) set all agents' social circles to the same value (50). This changes the network structure, increasing degree, reciprocity, and transitivity. In other words, agents are *more connected*, increasing Moran's coefficient for scenario I+CR (there are more chances of being influenced), and decreasing that coefficient in scenario S+CR (relatively less clustering of behavior).

4.3 Experimental Design

Table **??** shows all relevant combinations of the six ISA scenarios and the seven SIENA specifications, resulting in 18 experimental scenarios. As mentioned above, each of the six ISA scenarios is run 100 times to allow the Moran's I network autocorrelation to stabilize.[10] Each ISA run outputs the social and behavioral landscape at four measurement times, which are then fed into the respective SIENA specification. This results in fitting SIENA 1,800 times, 100 for each of the 18 experimental scenarios. Those models that reach overall convergence are kept and included in Table **??**.[11]

To evaluate the SIENA's performance we use two measures: standardized bias and coverage rate. Standardized bias is the raw bias (average parameter estimates across replications minus the true parameter value) divided by the standard deviation of the estimates across all replicates. For instance, a value of -0.5 means that the estimate on average falls one-half of a standard

[10] See supplementary section **??** for details on how the number of replicates was determined.

[11] We follow the SIENA authors' recommendation of an overall maximum convergence ratio smaller than 0.25 (**?**). All our models reached overall convergence.

deviation below the parameter. **?** provides the rule of thumb that absolute values of 0.4 or higher are practically significant. Coverage rates are the proportion of replications whose 95% confidence interval includes the true parameter estimate. The actual coverage should be approximately equal to or greater than the *nominal* coverage rate (95%). According to (**?**), problematic coverage rates are below 90%. We also estimate aggregate coefficients for selection and influence effects by using meta-analysis (**??**), so that we can compare the magnitude of effects across SIENA specifications and estimate the probability of observing a positive effect $Pr(\beta > 0)$ when it is present in our ABM. This probability might be interpreted as a measure of statistical power of selection/influence estimates, but we should note that it does not tell us anything about the bias of coefficients.

To assess the goodness-of-fit (GOF) of our models, we employ distributions of auxiliary statistics such as out-degree, in-degree, geodesic distance, triad census, and behavior and explore the differences (Mahalonobis distance) between the observed distributions (summed across the four waves of data) and the simulated distributions from SIENA (summed across 1000 random networks). Most of our specifications reproduce well the degree and behavior distributions. GOF for geodesic distances and triad census, in contrast, is weak. The connection between bias and GOF, though, is not straightforward. While some of our best SIENA estimates (e.g., S+I+R+D) have poor GOF with statistics such as geodesic distance and triad census, specifications with a similar (or even better) GOF appear to have more biased estimates (e.g., N+S+I). Section *Goodness of Fit* in the **??** provides details on the GOF of our models, although more research would be needed to explore the connection between bias and GOF in SIENA.

4.4 Results

1. First generation
   (a) Income
   (b) Age
   (c) Place of residence (income, income mobility)
2. Second generation
   (a) Cognitive skills (uniform variable)
   (b) Place of residence characteristics

Education of offsprings will be define as:

$$\text{education}_c = \alpha + \beta_1 \times \text{cognitive\_skills} + \beta_2 \times \text{parent\_income} + \beta_3 \times \text{city\_income} + \beta 4 \times \text{income\_mobility} + \sigma_c$$

$$\text{income}_c = \alpha + \beta_1 \times \text{age} + \beta_2 \times \text{education}_c$$

$$\text{smoking}_c = \alpha + \beta_1 \times \text{age} + \beta_2 \times \text{income}_c$$

$$\text{mortality}_c = \alpha + \beta_1 \times \text{age} + \beta_2 \times \text{smoking}_c$$

4.5 Research questions

According to Alberto this are good research questions. I still don't get them completely.

Alberto: When I think of the ABM exercise I think of the possibility of using the simulation to generate data that you can then use as if it where Chetty's data. Among other things you could investigate the relations between a measure of IM in each place (whatever the place is in the ABM) and the mortality risks of individuals who reside in the place.

Reproduction: This should NOT be a priority. For the time being just think of everybody have 1 child at age 30.

Some of my ideas:

1. To use it to make a more clear argument on public health policy
2. To specify well-grounded (or theoretical) dynamic mechanism and point out its potential importance, and ways to falsify it.
3. Identification (and formalization) of specific dynamic mechanisms of the effect of income mobility on health remains an important gap
4. Goal: to develop a better theoretical understanding of how one particular mechanism (income mobility) can independently produce and reproduce health disparities across place. The model might offer guidance for future empirical work in this area, as well as the potential to facilitate policy and intervention design.
5.

I think all of these are legitimate goals. One of the goals I had in mind is the following: use the results of the ABM to assess the degree to which the relation place income mobility and individual mortality changes as you change conditions generating the population being simulated. One could discover for example that no matter how tightly related IM and individual mortality are it is impossible to retrieve key parameters from county (or city) based data due to say residential mobility.

4.5.1 Original questions

These are the original research questions, but I think I should use this to define a more clear theory on how income mobility might (or might not) affect health/mortality, and their consequences at the aggregate level.

I am still unclear about which questions are the more relevant in this case:

1. What are the consequences of feedback mechanisms whereby the distribution of individuals' by health status influences income mobility on health disparities over several generations?[12]
2. How would health behave under different income inequality and mobility scenarios (e.g., inequality observed mainly between the top 1% and the rest of population, income mobility at different positions of the income distribution)?
3. How the system behaves when allowing selective migration (i.e., segregation) of agents across neighborhoods/counties?
4. What regression coefficients are actually telling us? Does a net effect of income mobility on health make sense or it is just an statistical figure with not practical translation to the real world?
5. Is it possible to really distinguish between income mobility and inequality effects on health with aggregate data?

4.5.2 Model Construction

Residential Segregation

Agents live in neighborhoods. At rate *t*, agents decide whether to move or stay in their neighborhood based on the proportion of people within the same quintile of income. Following Schelling segregation model, agents have a tolerance threshold (e.g., 20%) for people in the same quintile of income. If the proportion of people of that quintile is lower than the tolerance threshold, agents move to another neighborhood chosen randomly from a pool of neighborhood that has not reach its population limit (e.g., more than 30% its original size).

This mechanism generates income segregation.

[12] It is well-known that an important determinant of individuals' income/status attainment depends on labor market performance (Palloni et al. 2009). To the extent to which the latter is influenced by individual health, there is the potential of feedback effects whereby a regime of income mobility may be maintained steady, made more flexibly or rigid as a function of the distribution of children and adults by health status.

4.6 Design notes

4.6.1 Main ideas

Suppose we want to assess the effects of place income mobility on adult mortality. The idea is to start with a parental generation (a family or G1) characterized by well defined properties (see below). G1 then generates a child generation (G2) and endow this generation with traits that determine G2's properties.

1. Agents and properties
    (a) Generation G1 (parental) at age 30-40 is characterized by three properties
        i. Education
        ii. Income
        iii. Place of residence (city)
    (b) Place of residence (city)
        i. Income mobility
        ii. Income inequality
        iii. Income distribution
        iv. Segregation
    (c) Generation G2(child) at age 5
        i. Smoking status
        ii. Cognitive skills
        iii. Aspirations, time preferences future outlooks
        iv. Access to schools of a given quality

Methodological Supplement

The code used to create the database and run the models and plots is available at: `https://github.com/sdaza/dissertation/tree/master/ch04`.

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