Income Mobility, Mortality and Health in the US

by

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Introduction

Just a test Epstein (2006)

Recent research on U.S. income mobility and health using community and individual data shows that higher mobility is associated with lower mortality risks (Venkataramani et al., 2016; Venkataramani et al., 2015). That relationship seems to be stronger and more consistent than the relationship between income inequality and health (a topic widely studied), although considerably smaller than the impact of income on mortality. This preliminary evidence suggests that income mobility might be a relevant determinant of health and mortality. Surprisingly, this potential pathway has received little attention in the literature.

This dissertation builds on that small literature and examines the robustness of the relationship between income mobility and health at the aggregate and individual level. By using different data sources and modeling approaches, I describe the magnitude and variability of this association in the U.S., and explore the plausibility and consistency of explanations offered in the literature. The central argument is that the effect of income mobility on health is stronger and larger than the impact of income inequality and that the mechanisms behind it, although related to income inequality, are theoretically distinct and independent of those of income and inequality, and can have powerful and lasting consequences.

To ground this argument, I use two strategies. First, I analyze aggregate and individual data to assess the magnitude, robustness, and variability of the *effects* of income mobility[1], and empirically examine whether some of the potential pathways and mechanisms proposed in the literature are *supported* by the data. Secondly, building on this evidence, I use a theoretical model to assess the conditions and plausibility of the potential mechanisms involved in the association between income mobility and health. By formulating a *generative model*, where I precisely define (represent) a set of mechanisms (causal relationships) likely to bring about the observed patterns, I am able to assess the internal consistency of the theory and evaluate its generative sufficiency (Billari et al., 2006; Epstein, 2006; Manzo, 2014; Miller & Page, 2007; Page, 2015). These *virtual* representations help me explore the implications of the theory and ask *what-if* questions. The goal is to go beyond statistical models (i.e., the detection of average differences that may reflect the aggregate statistical

[1] I use the term *effects* in a loose way and mean the size and sign of (path) coefficients measuring the strength of the relation between two variables. I do not presume the existence of a proven causal relation.

signature of *unspecified* underlying mechanisms), and to approximate plausible mechanism behind the observed patterns.[2]

In the following sections I outline the problem, formulate a theoretical framework and propose two analytic strategies to examine it. Each of these sections will correspond to a chapter in the dissertation. I will add an introduction and conclusion. Thus, the dissertation will have an introduction, four chapters, and a conclusion.

[2]Models are just learning tools that, even failing to create a model that generates the observed patterns through the computational representation of the mechanisms suggested by theory, help understand the system and phenomenon of interest.

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 & Phelan, 1995; Phelan et al., 2010, 1 suppl) 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.

2.1 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 & 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 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.

[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.

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:

$$h_i = f_i(y_i, I_c) \tag{2.1}$$

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:

$$h_c = f_c(y_c, I_c) \tag{2.2}$$

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 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,

[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).

[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

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 & 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, Kawachi et al. 1997; Wilkinson 1992). 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 & 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 (Marmot, 2004; Wilkinson, 1992). 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, 1 suppl).

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.

2.2 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

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.

(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 intra-generational class mobility is detrimental to health, specially in social and historical contexts that foster high social expectations for economic success (Newman, 1999). 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., 2016; Venkataramani et al., 2015). 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 (Chetty et al., 2014; Winship, 2015). However, the relationship between income inequality and opportunity, even when considering the largest estimates, is far from perfect, providing empirical grounds to estimate the independent effect of income mobility and inequality.

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

2.2.1 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, 2000). 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 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

[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 (Barlow et al., 2016).

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):

$$h_i = f_i(y_i, m_i, M_c, I_c) \tag{2.3}$$

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 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,

[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.

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 & 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.

2.2.2 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.

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

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

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.

Income Mobility, Income Inequality and Mortality in the United States

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. (Ezzati et al., 2008; Murray et al., 2006). 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 (Daly & Wilson, 2013; Kawachi et al., 1997; Pickett & Wilkinson, 2009; Pickett & Wilkinson, 2015; Subramanian & Kawachi, 2004; Wagstaff & van Doorslaer, 2000; Wilkinson, 1992; Wilkinson & Pickett, 2006, 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 (Piketty & Saez, 2003), 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 (Ezzati et al., 2008; Murray et al., 2006). 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 (Chetty et al., 2017; Hout, 1984, 1988; Hout et al., 1993). 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., 2016; Venkataramani et al., 2015).

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

3.1 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 (Blane et al., 1999; Blane et al., 1993; Chandola et al., 2003; Fox et al., 1982; Illsley, 1955; Solon, 1992)[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 more beneficial in communities with less inequality than in those with high inequality. The new

[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 3.10 in the *Methodological Supplement* displays the scatter plot between the relative income mobility and Gini coefficient. The correlation is moderate, and there are counties with high inequality and high mobility, and viceversa.

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 (Mackenbach, 2012; Meara et al., 2008). Further, there is evidence that early conditions and upbringing of individuals matter greatly for adult health and mortality disparities (Case et al., 2002; 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 (Marmot, 2004; Sapolsky, 2005) may be rooted in antecedent health conditions sculpted early in life (Case & Paxson, 2011). 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 (Cunha & Heckman, 2009; Heckman, 2007; Knudsen et al., 2006; Shonkoff et al., 2009). 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, 1972, 2000).

   We know from empirical research that negative affect, chronic stress, subordination, bleak future outlooks associated with poverty lead to increases in time discounting (Haushofer & Fehr, 2014). 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 (Eigsti et al., 2006; Schlam et al., 2013).

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.

3.1.1 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.

3.2 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 period2 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 1980 and 1982 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 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

[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 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.

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 3.9 in *Methodological Supplement*).[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).

3.3 Model Estimation

3.3.1 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:

[7] Results using absolute mobility are shown in the *Methodological Supplement*. 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_ε^2.

$$
\begin{aligned}
D_{ix} &\sim \text{Poisson}(\mu_i) \\
\log(\mu_i) &= \log(\tau_i) + \alpha + \beta_{\text{m}}\text{M}_i + \beta_{\text{g}}\text{G}_i + \beta X_i \\
\alpha &= \alpha_c + \alpha_{\text{state[i]}} + \alpha_{\text{county[i]}} + \alpha_{\text{obs[i]}} + \alpha_{\text{age[i]}} \\
\beta_{\text{m}} &= \beta_{\text{Mob}} + \beta_{\text{Mob}_{\text{age[i]}}} \\
\beta_{\text{g}} &= \beta_{\text{Gini}} + \beta_{\text{Gini}_{\text{age[i]}}}
\end{aligned} \tag{3.1}
$$

where D_{ix} 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.

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 (Gelman et al., 2012; Hill, 2013).

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 3.1 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)

[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 *Methodological Supplement*.

[14] `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 $\sigma = 31.62$. This diffuse prior is used for all fixed regression parameters, except for the intercept in which

priors which are designed to be weakly informative (for more details see Simpson et al., 2017).[15] Fortunately, estimates associated with income mobility and inequality are insensitive to the different prior specifications we examined.

3.3.2 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 3.3 in the Methodological Supplement 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 & Cameletti, 2015). We use the parameterization proposed by Riebler et al., 2016.

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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 *Methodological Supplement*.

3.3.3 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 3.1 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.

3.4 Results

3.4.1 Average Association

Tables 3.1 (female) and 3.2 (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 3.1) 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 3.1 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.

3.4.2 Effects by age

Average effects of income mobility and inequality in Table 3.1 and 3.2 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 3.1 and 3.2 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 3.2 show (exponentiated) estimates and CIs of β_m and β_g from Equation 3.1 by sampling posterior distributions of Model *Covariates* in Tables 3.1 and 3.2. 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 the Model *Covariates* (Tables 3.1 and 3.2) to predict counterfactual mortality rates by computing the following quantities:

$$\begin{aligned} \alpha &= \alpha_c + \alpha_{\text{age[i]}} \\ \beta_{\text{m}} &= \beta_{\text{Mob}} + \beta_{\text{Mob}_{\text{age[i]}}} \\ \beta_{\text{g}} &= \beta_{\text{Gini}} + \beta_{\text{Gini}_{\text{age[i]}}} \end{aligned} \tag{3.2}$$

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 3.3 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 3.4 and, as expected, they show an increasing trend by age, particularly at older adult ages, where the size of life expectancy declines rapidly.

3.4.3 Effects by Race/Ethnicity

Do the effects uncovered before vary by race and/or ethnic group? Figure 3.5 displays the posterior distribution of mortality ratios using the model *Covariates* of Table 3.2 and 3.1 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 3.6 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. There 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

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

considerably smaller (about 0.6 years of decrease in life expectancy at $E0$) than effects of IMR (between 1 and 1.5 years). Again, differences in impact are assessed using *relative* changes (see Figure 3.7). These quantities confirm differences between Whites and African Americans and reveal that the gaps between effects of GI and IRM increase with age.

3.4.4 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 3.1 and 3.2, but do so separately by four cause death as defined before. Figure 3.8 displays the posterior distribution of mortality ratios $\exp(\beta_m)$ and $\exp(\beta_g)$ (see Equation 3.1). 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 confirmed 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.

3.5 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

[18] See Table 3.8 in *Methodological Supplement* for details on the coding schema we use.

accounting for income mobility (and income inequality). However,our findings also show that the 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 (Heckman, 2007). 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.

3.6 Tables and Figures

Table 3.1: 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.

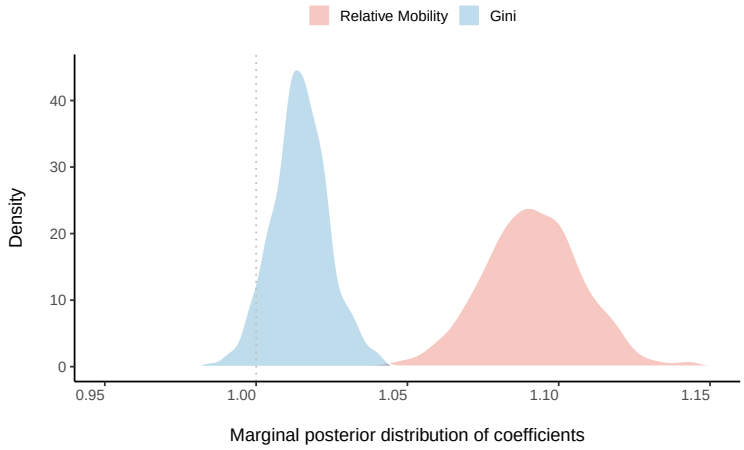
Table 3.2: County Level Poisson Models Relative Mobility
PC Prior = $Pr(\sigma > 1) < 0.10$, Men, CDC 2000-2014

	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 3.1: Posterior Distribution of $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ by Gender
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

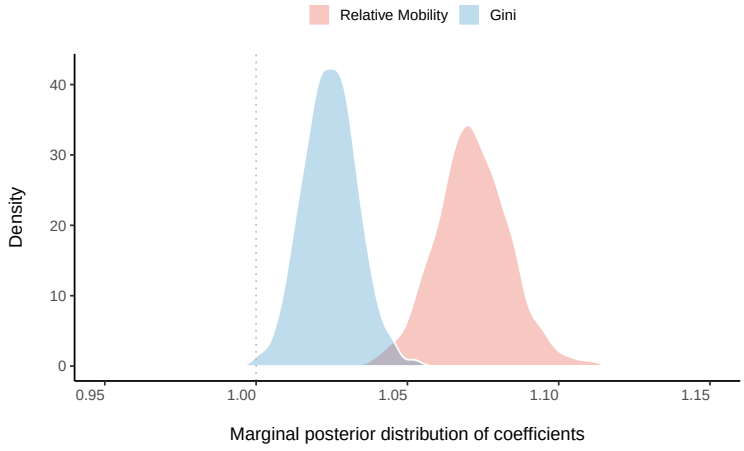
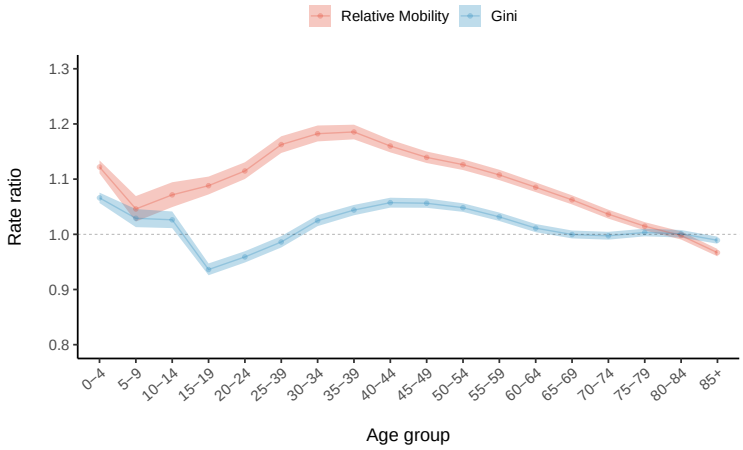


Figure 3.2: 95% Credibility Interval Posterior Distribution of $exp(\beta_m)$ and $exp(\beta_g)$ (Equation 3.1) by Age Group Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

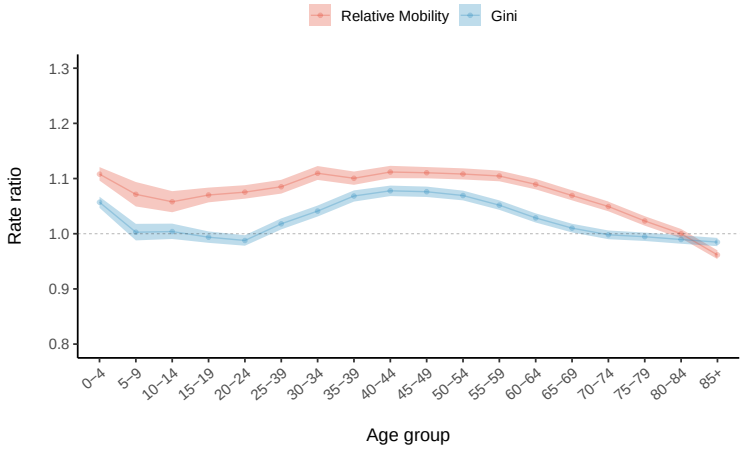
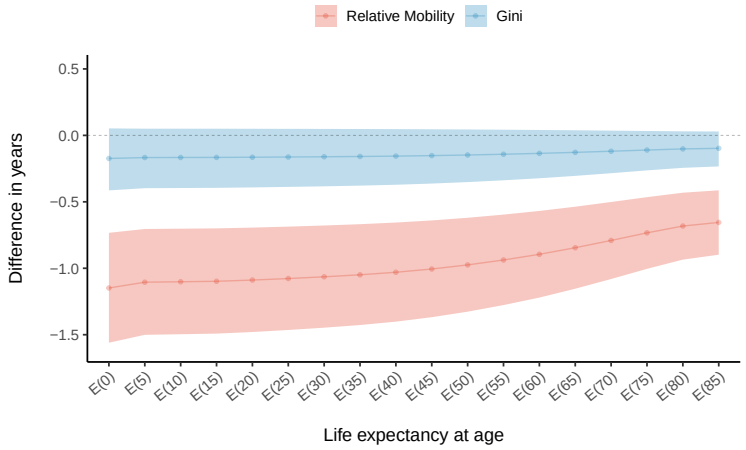


Figure 3.3: 95% Credibility Interval of Predicted LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

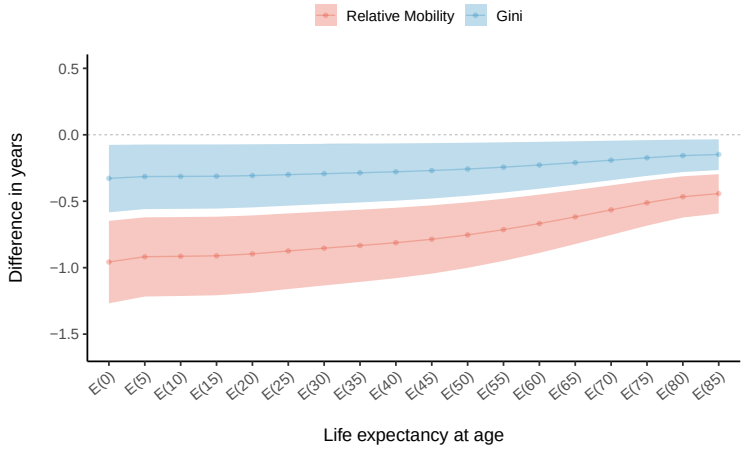
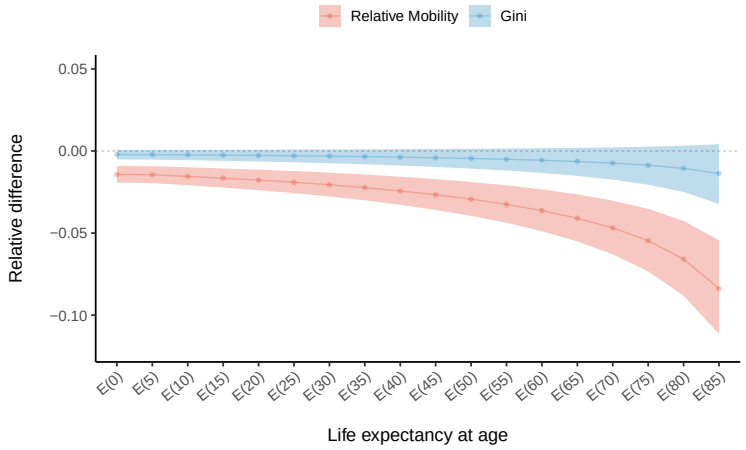


Figure 3.4: 95% Credibility Interval of Predicted Relative LE Differences by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

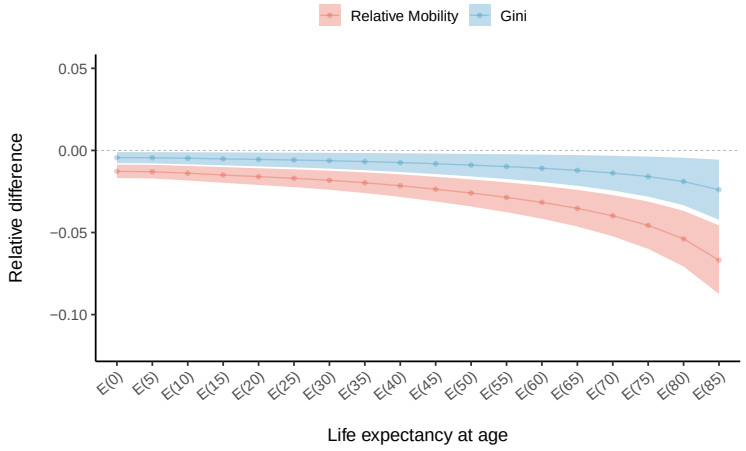
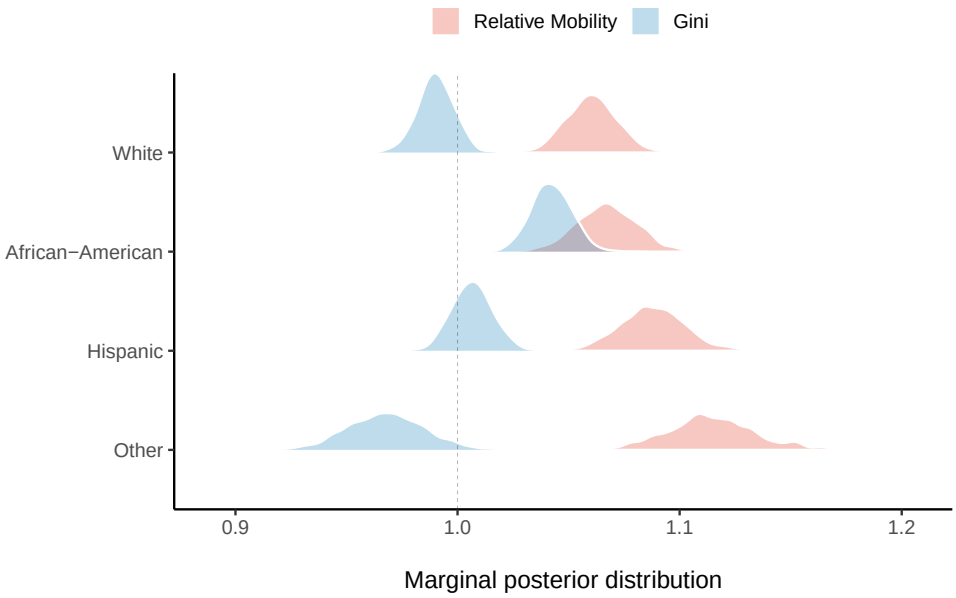


Figure 3.5: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ (Equation 3.1) by Race/Ethnicity and Gender

(a) Women



(b) Men

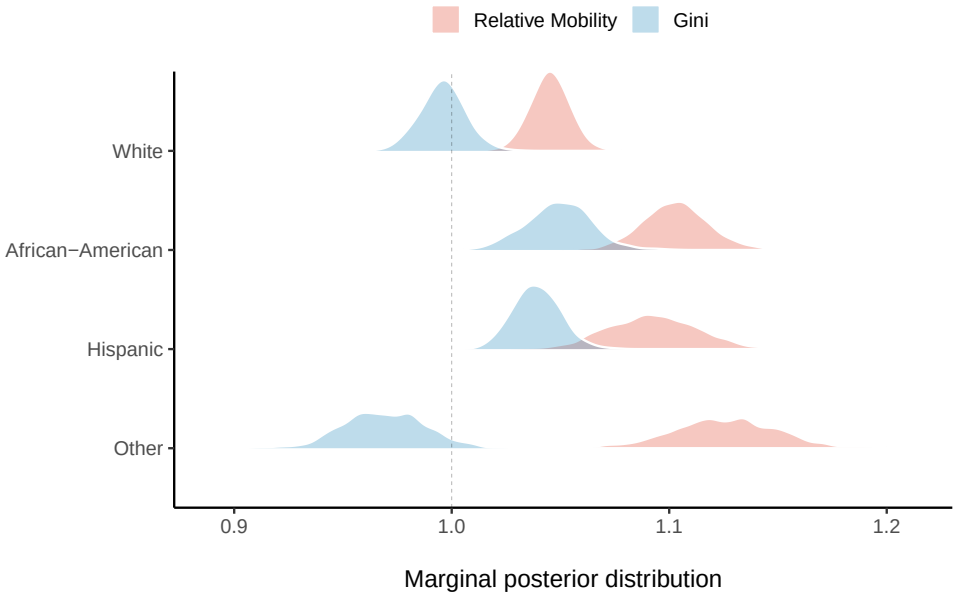
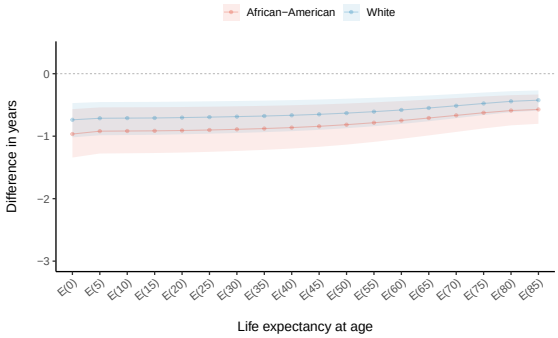
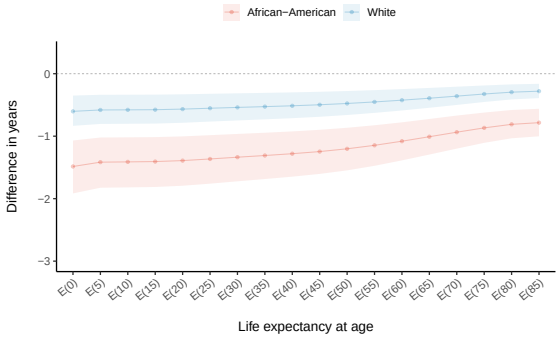


Figure 3.6: 95% Credibility Interval of Predicted LE Differences
by Age Group and Race/Ethnicity, Increase in One Standard Deviation

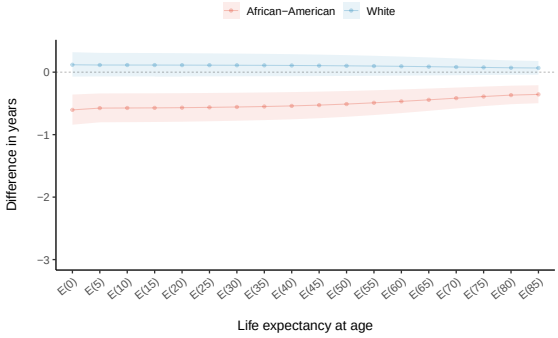
(a) Women - Relative Mobility



(b) Men - Relative Mobility



(c) Women - Gini



(d) Men - Gini

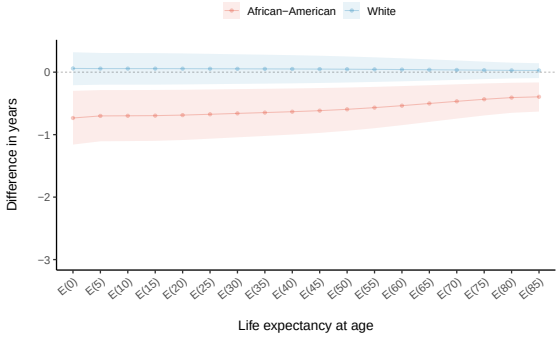
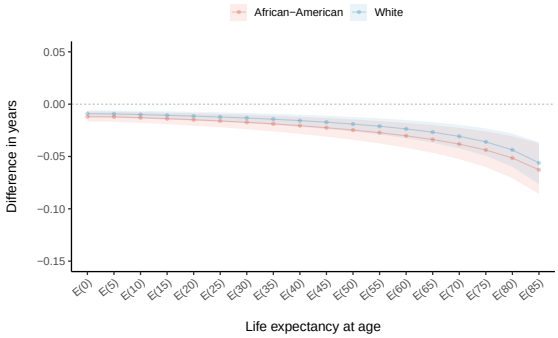
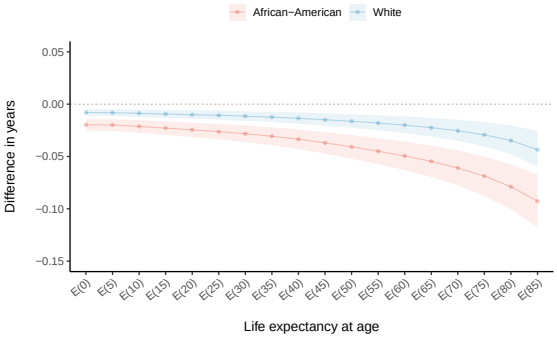


Figure 3.7: 95% Credibility Interval of Predicted LE Relative Differences by Age Group and Race/Ethnicity, Increase in One Standard Deviation

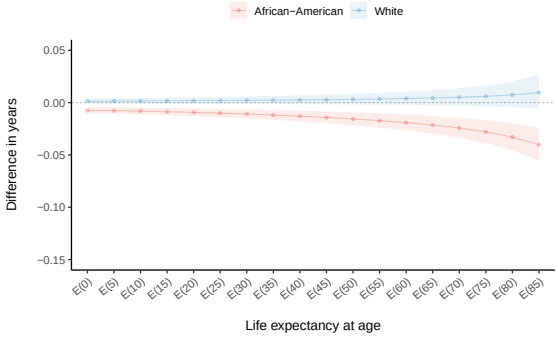
(a) Women - Relative Mobility



(b) Men - Relative Mobility



(c) Women - Gini



(d) Men - Gini

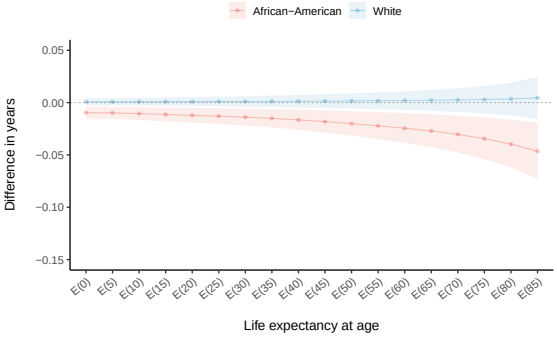
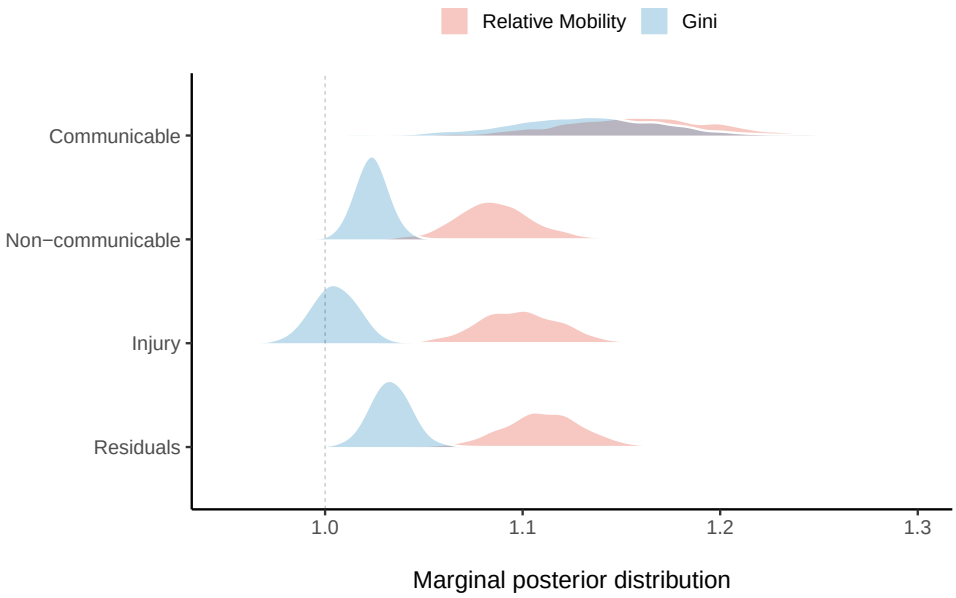
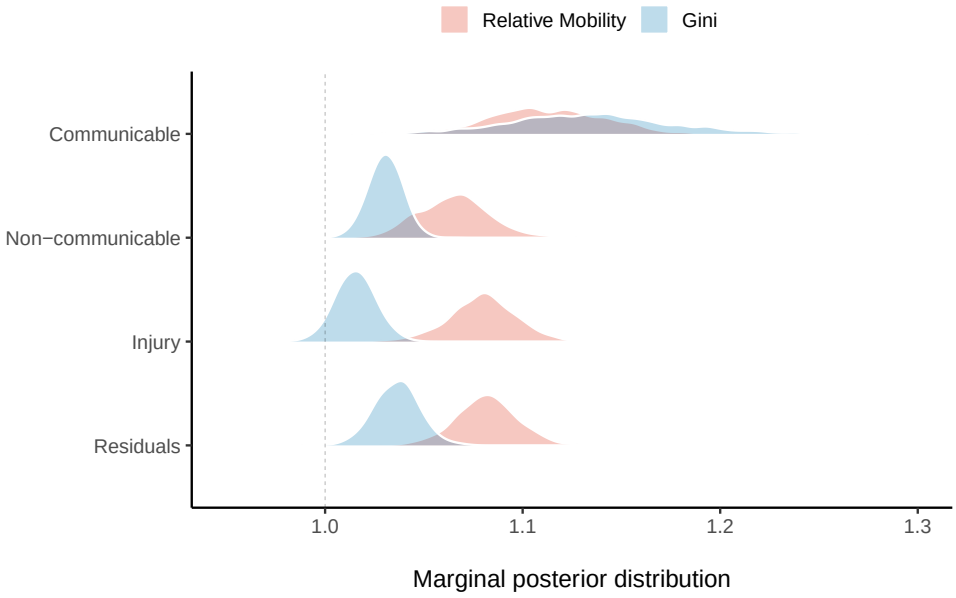


Figure 3.8: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta\text{Gini})$ by Cause of Death and Gender

(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.

3.7.1 Descriptive Statistics County Level

Figure 3.9: County Coverage Income Mobility Measures (Colored)
2867 counties (91%)

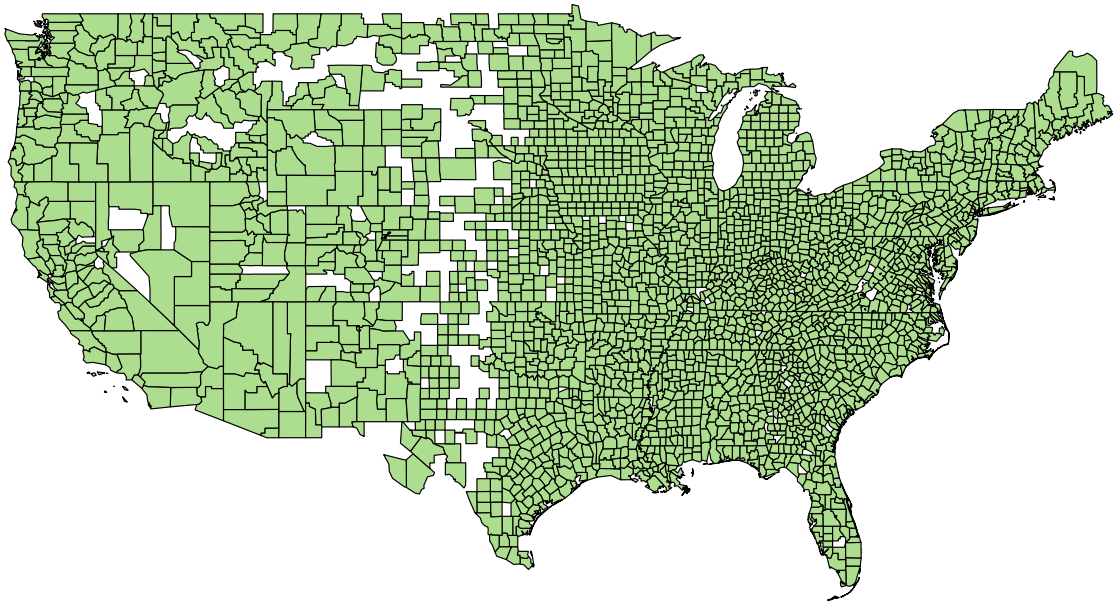
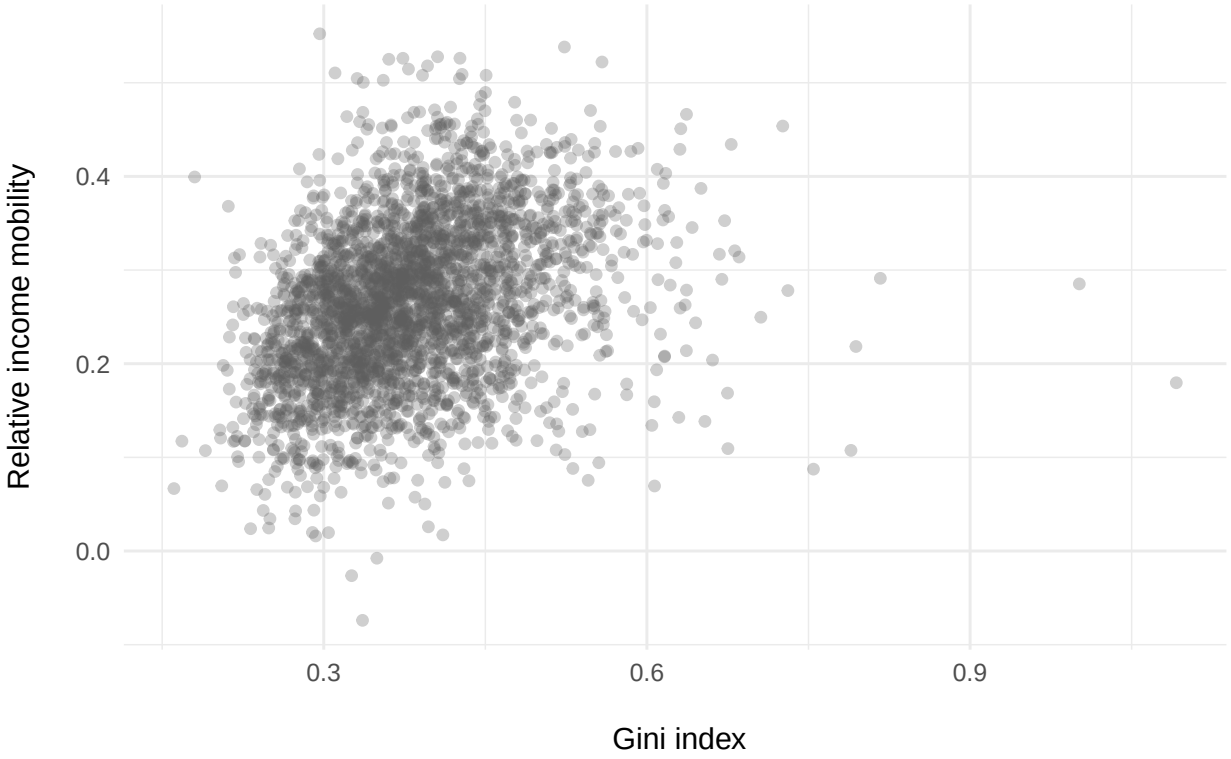


Table 3.3: Descriptive Statistics

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 3.10: Income Mobility and Inequality, 2867 counties



3.7.2 Goodness of Fit

We examine the goodness of fit (GOF) of the model *Covariates* in Table 3.1 and 3.2 using the *leave-one-out* predictive measure *probability integral transform* (PIT) (Wang et al., 2018).

The probability integral transform (PIT) is defined as:

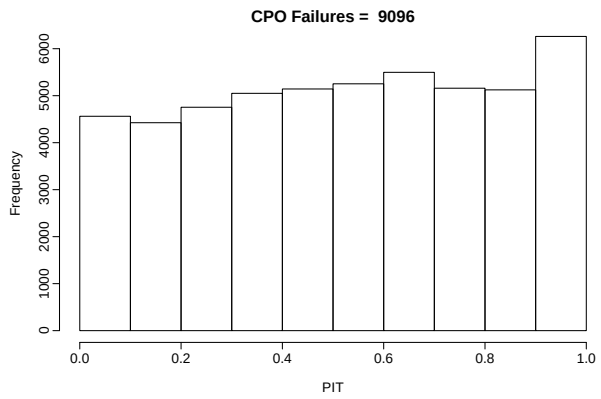
$$PIT_i = p(y_i^{new} \leq y_i | y_{-i})$$

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.

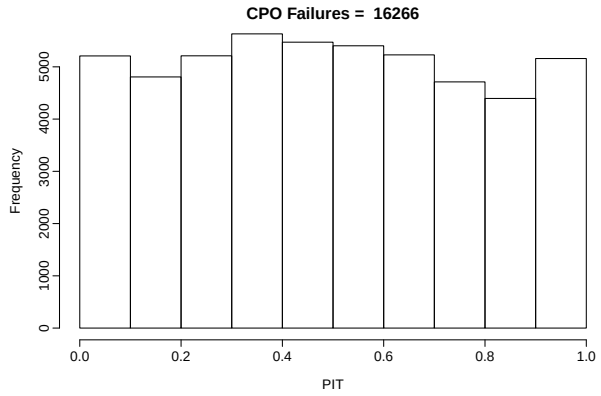
Figures 3.11 and 3.12 show the histogram and the uniform Q-Q plot of PITs for females and males. As can be seen, the distribution of the PITs is close to a uniform distribution, suggesting that the model reasonably fits the data.

Figure 3.11: PIT Distribution
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

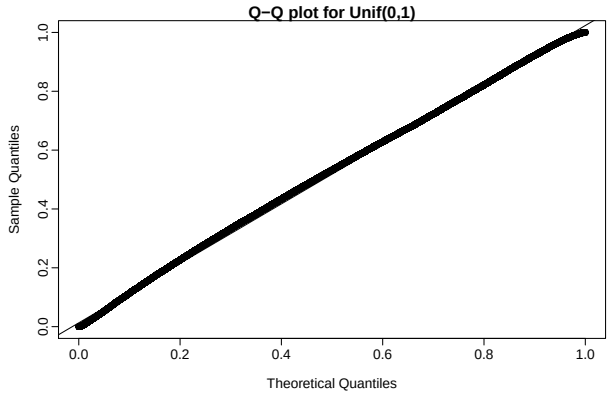


3.7.3 Prior sensitivity analysis

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 Simpson et al., 2017). 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 3.12: Q-Q Plot PIT
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

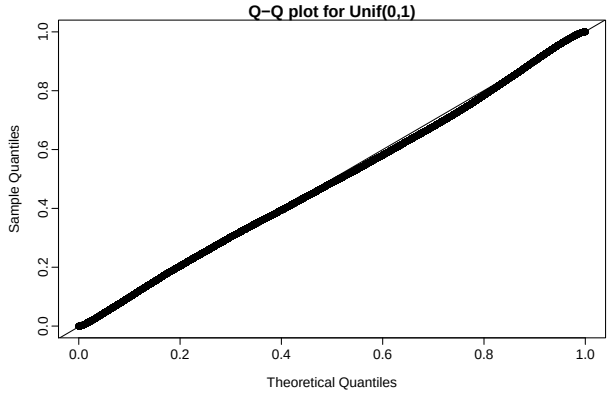


Table 3.4 and 3.5 show the results using different priors with a model equivalent to the *Covariates* model in Table 3.1 and 3.2 of the main paper. As can be seen, fixed effects practically do not change when using different prior specifications. The precision of random terms, as expected, shows more 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 3.4: 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 3.5: 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.

3.7.4 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.

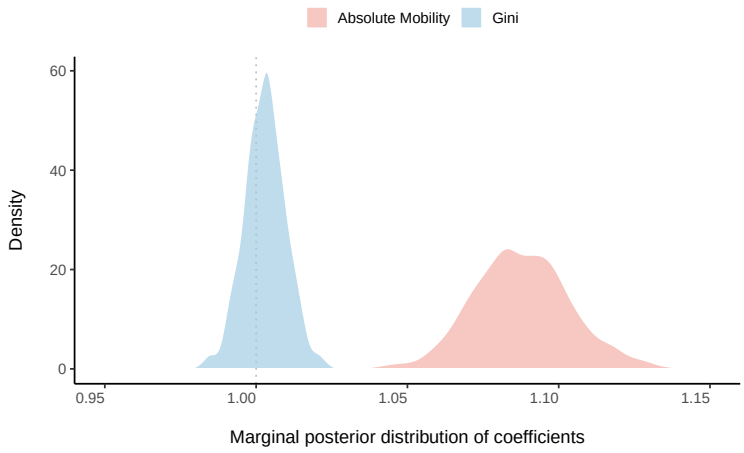
Absolute upward income mobility ranges from 0 to 1, and higher values correspond to large income mobility. We multiply the absolute upward mobility score by -1 so that the interpretation and expected association of relative and absolute income mobility were the same (i.e., increases in income mobility 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 3.14). 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 3.15). Relative differences are shown in Figure 3.16. Finally, there is also a much clear difference in the effect of IRM between Non-Hispanic White males and other groups (see Figure 3.17).

3.7.5 Tables and Figures

Figure 3.13: Posterior Distribution of $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ by Gender
Model *Covariates* in Tables 3.6 and 3.7

(a) Women



(b) Men

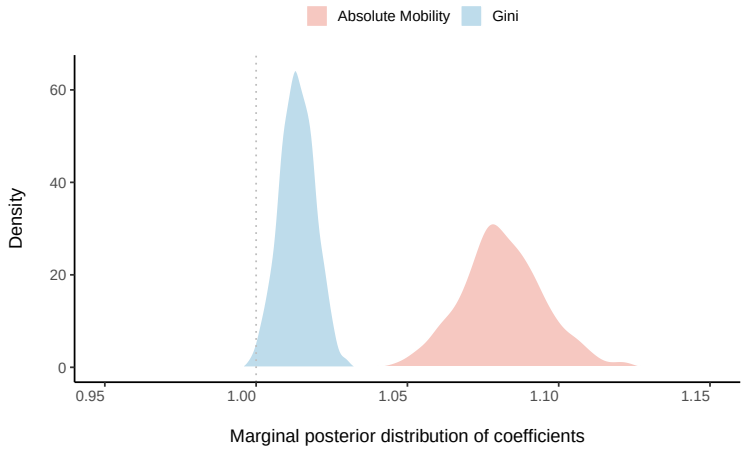


Table 3.6: County Level Poisson Models Absolute Mobility
PC prior $= Pr(\sigma > 1) < 0.10$, Women, CDC 2000-2014

	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.

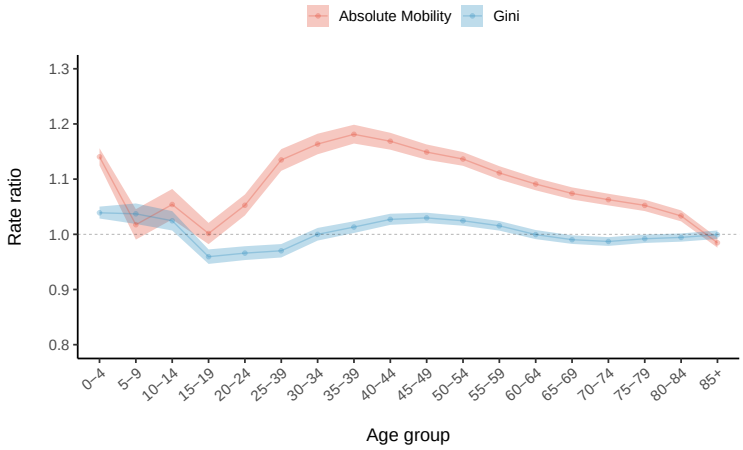
Table 3.7: County Level Poisson Models Absolute Mobility
PC Prior $= Pr(\sigma > 1) < 0.10$, Men, CDC 2000-2014

	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 3.14: 95% Credibility Interval Posterior Distribution of $exp(\beta_m)$ and $exp(\beta_g)$ (see Equation 3.1) by Age Group Model *Covariates* in Tables 3.6 and 3.7

(a) Women



(b) Men

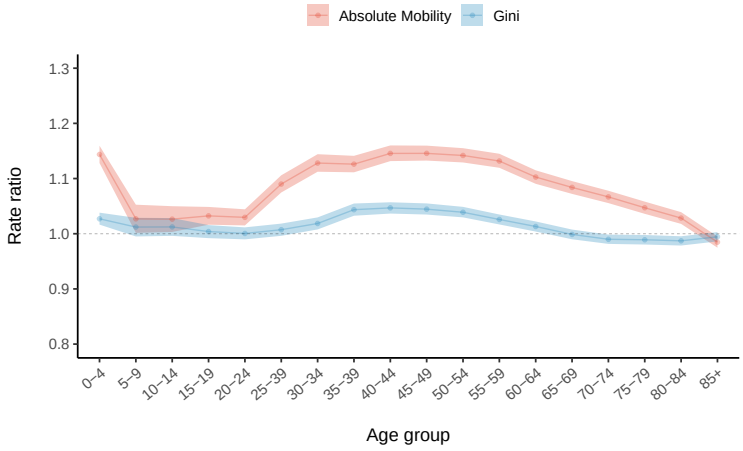
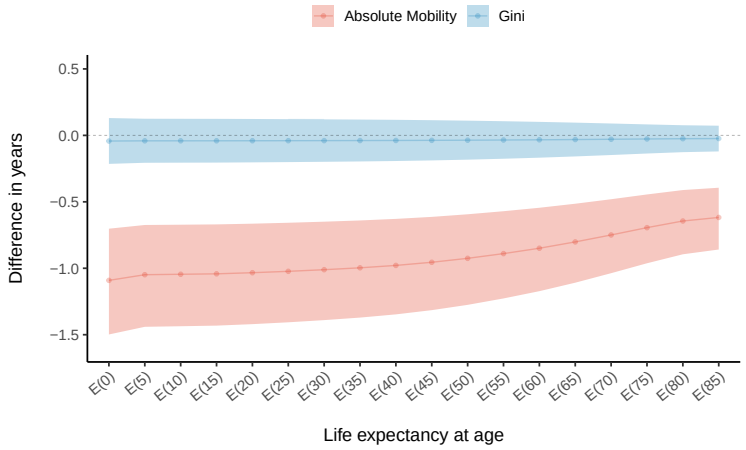


Figure 3.15: 95% Credibility Interval of Predicted LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.6 and 3.7

(a) Women



(b) Men

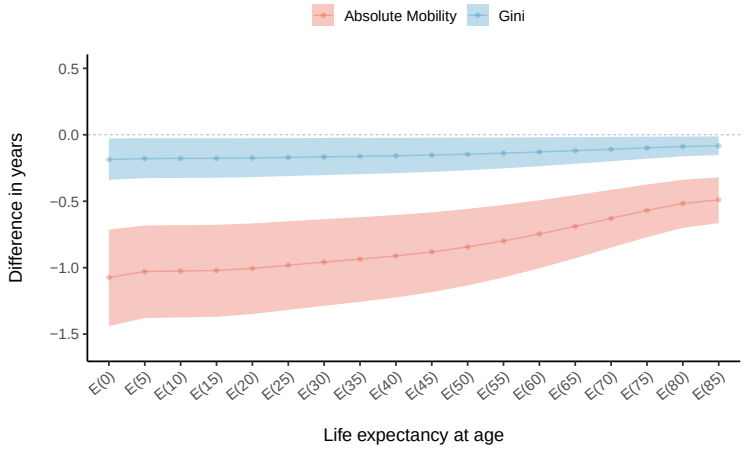
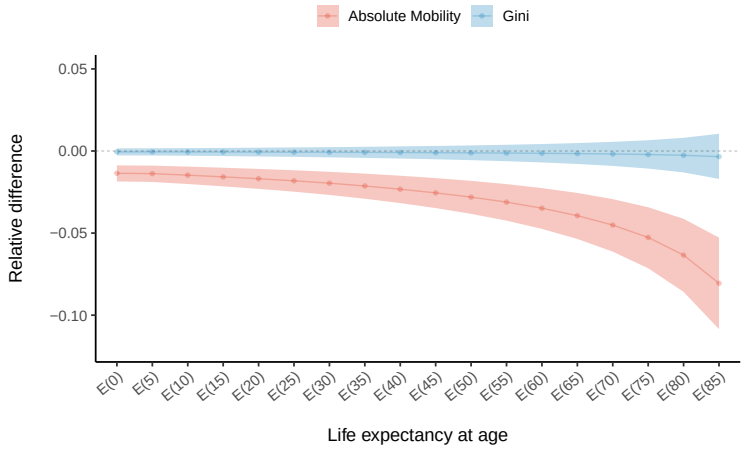


Figure 3.16: 95% Credibility Interval of Predicted Relative LE Differences by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.6 and 3.2

(a) Women



(b) Men

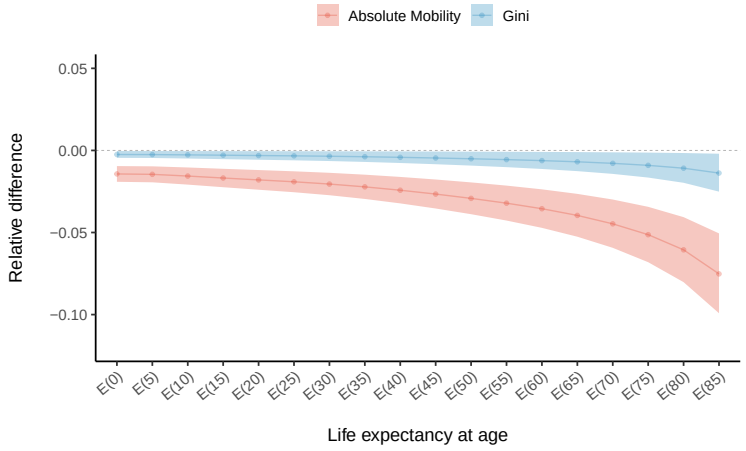
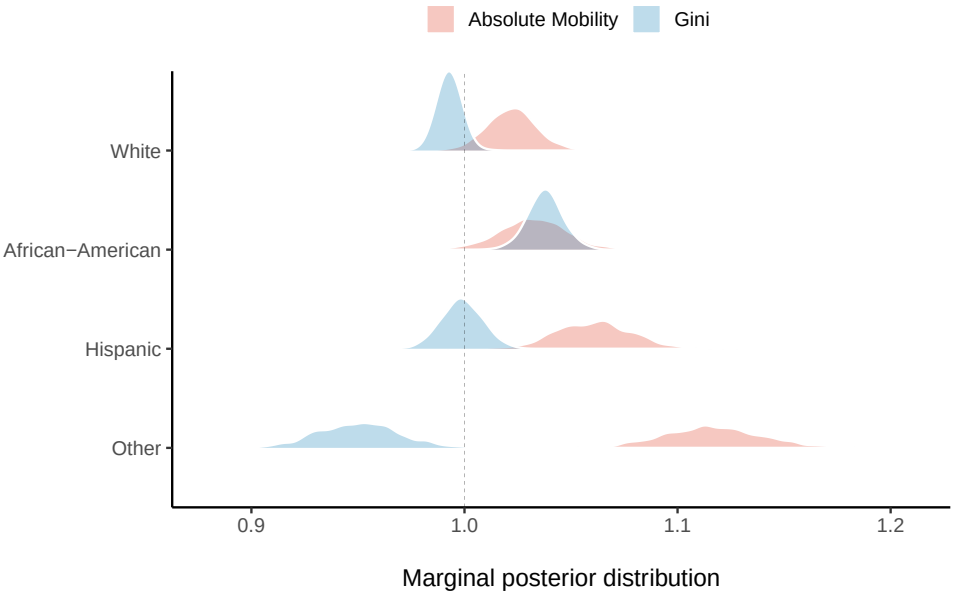


Figure 3.17: Posterior Distribution $exp(\beta_m)$ and $exp(\beta_g)$
Model *Covariates*

(a) Women



(b) Men

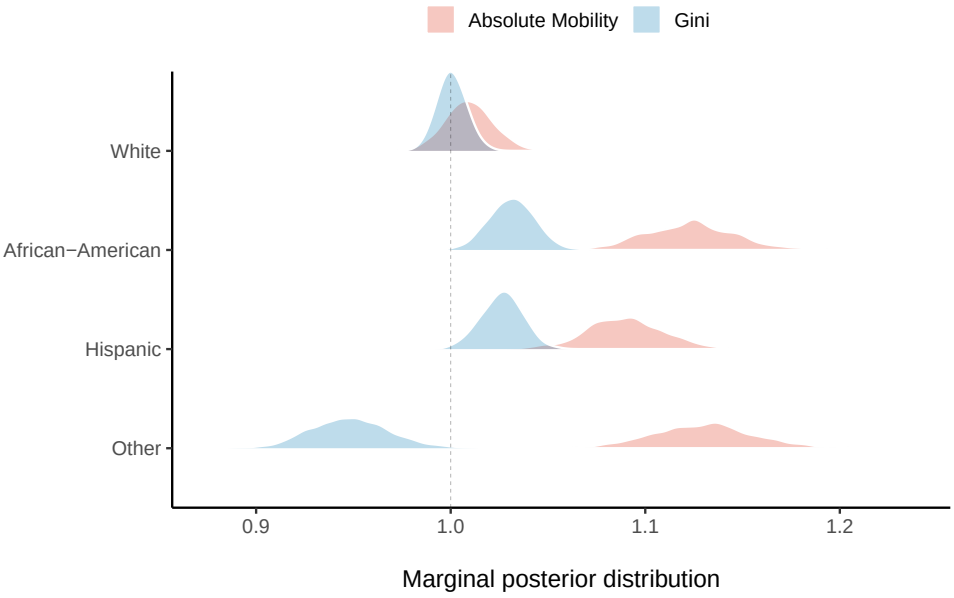
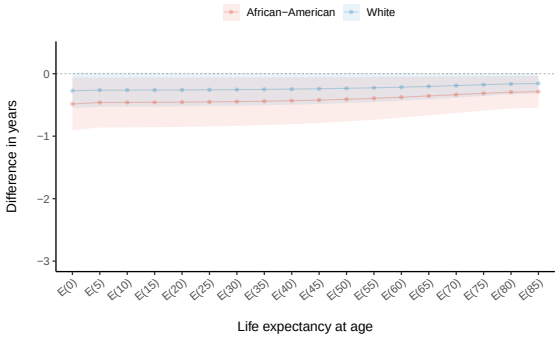
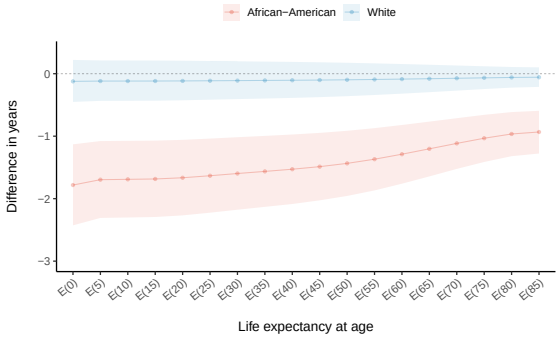


Figure 3.18: 95% Credibility Interval of Predicted LE Differences
by Age Group and Race/Ethnicity, Increase in One Standard Deviation

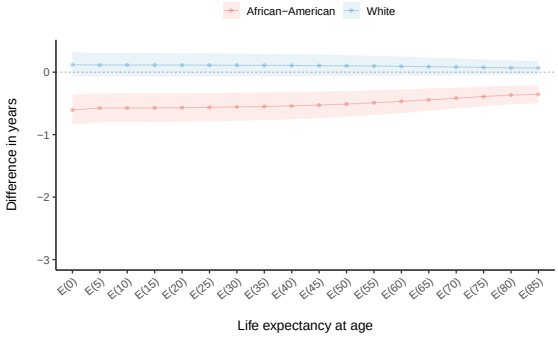
(a) Women - Absolute Mobility



(b) Men - Absolute Mobility



(c) Women - Gini



(d) Men - Gini

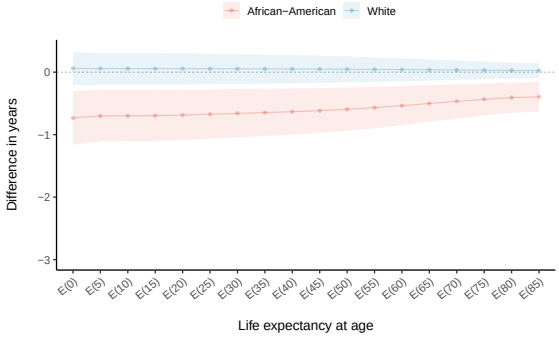
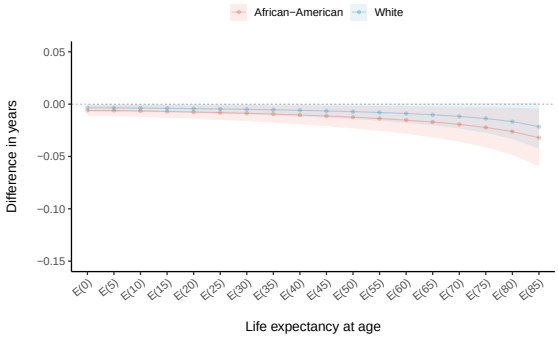
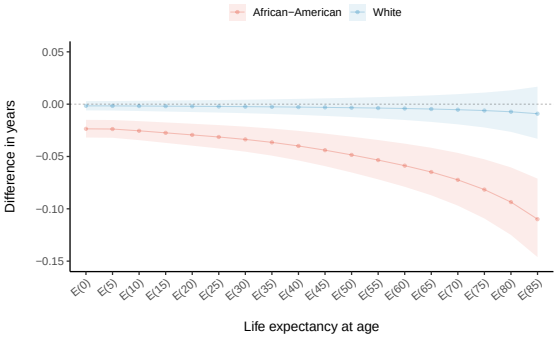


Figure 3.19: 95% Credibility Interval of Predicted LE Relative Differences by Age Group and Race/Ethnicity, Increase in One Standard Deviation

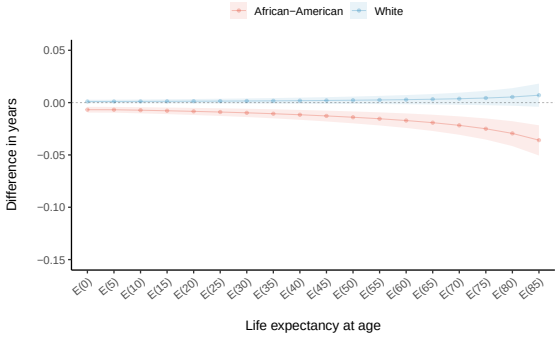
(a) Women - Absolute Mobility



(b) Men - Absolute Mobility



(c) Women - Gini



(d) Men - Gini

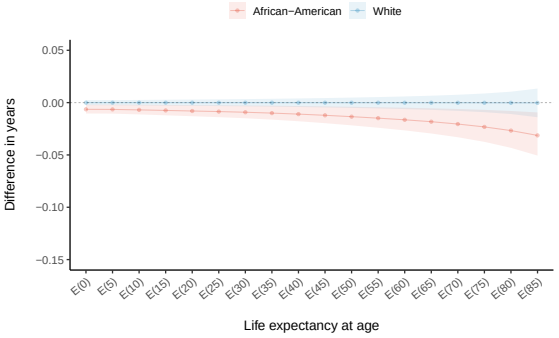
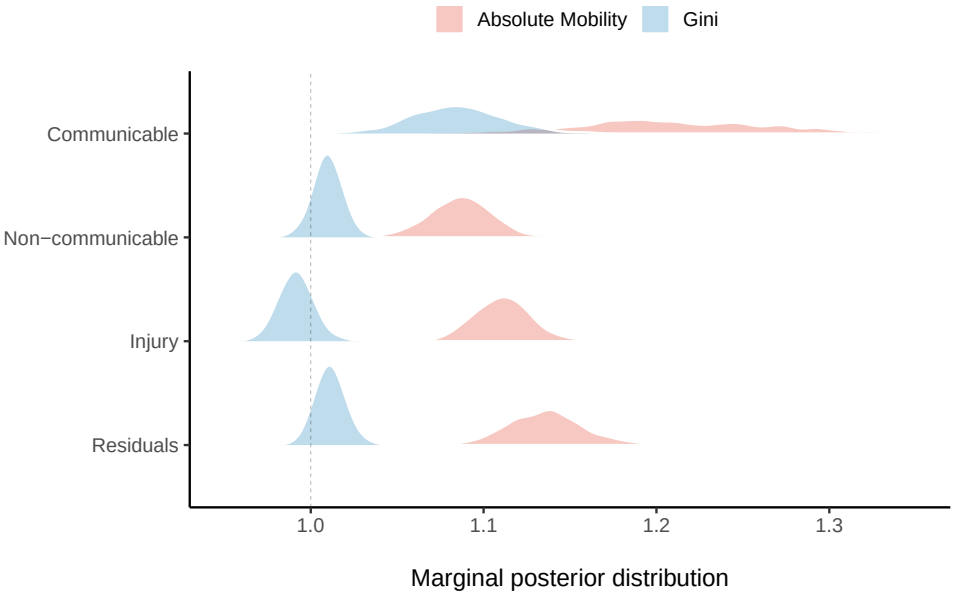
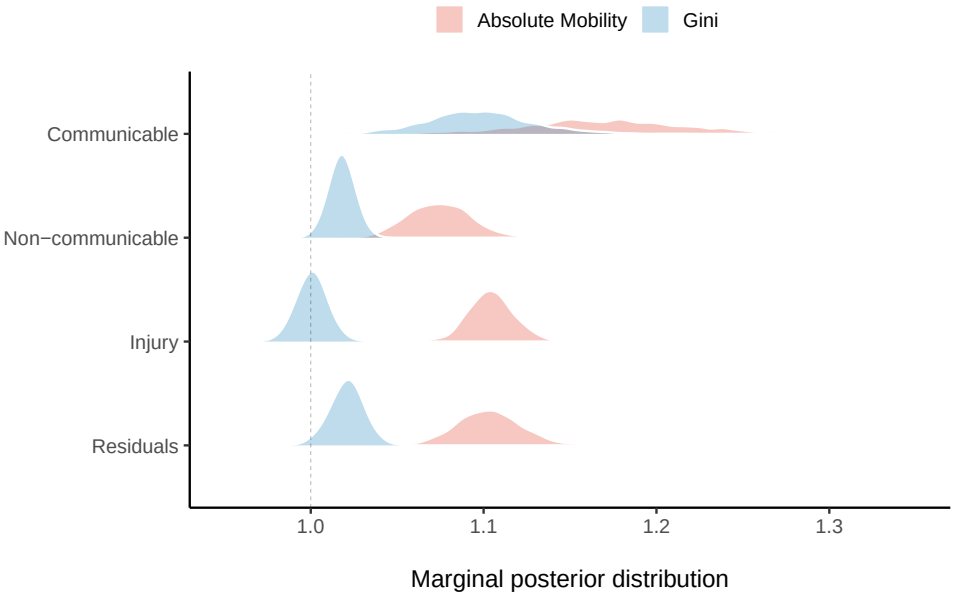


Figure 3.20: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta\text{Gini})$
by Cause of Death and Gender

(a) Women



(b) Men



3.7.6 Cause of Death Coding

Table 3.8: 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)

Early Exposure to County Income Mobility and Adult Individual Health in the United States

The growing life expectancy gap by income represents a fundamental challenge for health policy in the U.S. The best performing U.S. counties have life expectancies that are 20 years greater than the poorest performers. Recent work by Chetty and colleagues (Chetty et al., 2016) shows that the difference in life expectancy (at age 40) between the richest 1% and poorest 1% in the United States is 14.6 years for men and 10.1 years for women. Also, between 2001 and 2014, life expectancy has increased 2.34 years for men in the top 5% of the income distribution and 2.91 years for women, but only 0.32 years for men in the bottom 5% of the distribution and 0.04 years for women. These gaps are substantial – representing about 35% of remaining life expectancy at age 40 among men and 25% for women.

A large body of recent research demonstrates that neither access to medical care nor socioeconomic factors fully explain observed geographic or income disparities in longevity. The search for drivers of the longevity gap has led scholars to suggest that contextual income mobility — defined as the ability of individuals to exceed their parents' income – may play an essential role in explaining health disparities (Daza & Palloni, 2018; Venkataramani et al., 2016; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020). For instance, low-income mobility may harm health by raising despair and diminishing the motivation to engage in healthy behaviors. These effects would be distinct to the consequences of income inequality for health. Individuals living in areas characterized by similar high degrees of income inequality may experience different probabilities of income mobility – and therefore may have different impacts on health outcomes. While the association between income inequality and health has been studied over the last 20 years, recent work states that its contribution to disparities in longevity may be small (Chetty et al., 2016). In contrast, the health consequences of economic mobility remain understudied. This gap in the literature is particularly salient given emerging evidence of falling income mobility in the U.S., especially among the same birth cohorts currently experiencing divergence in their life expectancy (Chetty et al., 2017).

Previous evidence on the link between income mobility and health comes mostly from the analysis of aggregate data (Daza & Palloni, 2018; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020), and individual cross-sectional surveys (Venkataramani et al., 2016). We identify at least three main limitations of previous research. First, associations observed at the

aggregate (e.g., county-level data) might not be kept at the individual level when most of the mechanisms proposed in the literature consist of individual processes (i.e., ecology fallacy), not aggregate ones (Daza & Palloni, 2018). Consequently, a direct way to assess the hypothesis on the link between contextual income mobility and health would require individual-level data. Second, as the neighborhood effects literature has pointed out (Sampson et al., 2002; Wodtke et al., 2016; Wodtke et al., 2011), residential mobility might produce spurious associations between contextual variables and individual outcomes. To obtain unbiased estimates of the effect of contextual income mobility on health outcomes, we need either experimental or longitudinal data that allow us to adjust for selection associated with residential mobility. Lastly, previous research has not defined clearly when exposure to a place's income mobility during the life course would have significant consequences for health. Are the consequences the same whether the exposure was in childhood or at age 40? The theory suggests that this would not be the case. Thus, it is necessary to define clearly what exposure is and how it is measured to correctly interpret the associations found in the data and avoid over-interpreting spurious relationships.

Our study extends previous research by estimating the effect of average exposure during childhood and adolescence on health outcomes and behaviors measured during young adulthood (the early thirties and forties) using longitudinal data. We employ both the NLSY97 and PSID with geocode data to assess the link between county-level income mobility (Chetty et al. 2014's estimates) and health outcomes and behaviors such as self-report health, BMI, depression, and smoking. Also, we use data that match better the cohorts used by Chetty to estimate of income mobility in the U.S. at the county level (i.e., children born between 1980 and 1982), account for selection associated with residential mobility over time, and adjust for time-varying confounders using marginal structural models (MSM). Thus, we provide a more precise assessment of the hypothesis that exposure to income mobility may determine health later in life and explain the longevity gap.

4.1 Individual mechanisms

We briefly discuss potential individual mechanisms that would generate an association between the place's income mobility and health. First, we notice we are examining the relationship between a *place's income mobility* (i.e., an aggregate property of the stratification system) and health outcome (i.e., an individual trait), not the relationship between individuals' lifetime income mobility and adult health – a problem studied in a large and distinguished body of research (Blane et al., 1999; Blane et al., 1993; Chandola et al., 2003; Fox et al., 1982; Illsley, 1955; Solon, 1992).[1] 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 a place's income mobility and individual health are linked. However, in this paper, we focus on the *total* effects of place's income mobility on individual health, not in the precise empirical identification of mediating pathways.

[1]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 adult life and subsequent older adult health and mortality.

We argue that a link 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 who occupy the most vulnerable and exposed social positions within unequal communities may be comparatively better off when facing 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, persons that occupy lower-ranked positions in societies with higher income mobility may enjoy better health than counterparts in societies with more rigid stratification systems.

Theoretically and empirically, communities can be classified in different combinations of income mobility and inequality, where each combination is characterized by a health and mortality profile.[2] The standard conjecture is that indicators of mortality and health will be more beneficial in communities with less inequality than those with high inequality. We extend that conjecture by stating that, given a level of income inequality, better health and mortality conditions will be experienced by community members with higher income mobility. Following a neo-material reinterpretation of the effect of income inequality on health (Lynch et al., 2004), we can argue that the place's income mobility by itself is not the root of health disparities, but only a manifestation of a cluster of conditions that affect population health, so that the association between income inequality and health would be contingent on people's resources at the individual and community levels. Accordingly, to estimate the independent effect of inequality or mobility on health, we would need to account for material factors at the community level that might confound the association between health and stratification characteristics, taking the precaution of not adjusting for mediators of the causal process between income mobility and health.

We identify at least four pathways that might produce a link between income mobility and health. First, the association between aggregate income mobility and individual health may be the outcome of a composition effect. Namely, places with higher income mobility contain a population composition biased toward individuals who experience socioeconomic mobility. In this case, the association between a stratification trait and individual experiences of health and mortality would reflect the influence of individual residential mobility patterns and selection.

Second, exposure to income mobility during childhood and adolescence may be influential as vehicles that establish relations between income inequality, income mobility, and adult health. There is evidence that individuals' early conditions and upbringing matter greatly for adult health and mortality disparities (Case et al., 2002; 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 (Marmot, 2004; Sapolsky, 2005) may be rooted in antecedent health conditions sculpted early in life (Case & Paxson, 2011). For instance, individuals can experience sensitive and critical windows for the acquisition of cognitive and non-cognitive abilities during early stages of socialization, which, in turn, are the foundation of skills acquired later in life

[2]Our data, for instance, show communities (counties) with both an unequal income distribution and flexible mobility regimes (high income mobility), but also communities with a generous income distribution and high social rigidity.

(Cunha & Heckman, 2009; Heckman, 2007; Knudsen et al., 2006; Shonkoff et al., 2009). Some of these traits involve the development of outlooks and attitudes that influence investments in skill acquisition and health, including propensities to adhere to 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, would be determined, in part, by capabilities sculpted early in life. Early avoidance of unhealthy behaviors has large pay-offs 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, 1972, 2000).

We also know from empirical research that negative affect, chronic stress, subordination, bleak future outlooks associated with poverty lead to increases in time discounting (Haushofer & Fehr, 2014). Higher time preferences favor 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 (Eigsti et al., 2006; Schlam et al., 2013). 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. Nevertheless, so are individual's ancestral income mobility experiences, particularly parental and, possibly, grand-parental socioeconomic mobility. Strictly speaking, these are mechanisms that can be properly identified only if we simultaneously observe the influences of a place's aggregate income mobility and individuals' familial income mobility experiences.

Lastly, 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 shape how 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, diminishes the value of adopting attitudes and behaviors that promote good health. This is in line with the hypothesis proposed by Case and Deaton (2020), who reported the fastest-rising death rates of causes such as suicides, drug overdoses, and alcoholic liver disease in the U.S., especially among those without a bachelor's degree. These self-inflicted deaths have been designated as *deaths of despair* as they affect those who face economic, social, or psychological adversities, hopelessness, and lack of well-being. We suggest that these population's adversities, in addition to a rigid or weak income mobility regime, would decrease the adoption of healthy behaviors, affecting not only the current generation but also those to come.

Based on these mechanisms, especially the hypothesis that early exposure to a given income mobility regime has substantial consequences for adult health, we examine whether exposure to a given mobility regime during childhood and adolescence impacts health indicators later in life,

after adjusting for residential mobility selection and time-variant confounders. Thus, we extend previous research by carrying out a more precise assessment of the link between contextual income mobility and health.

4.2 Data

Our data result from combining different sources. The first is the Health Inequality Project Data (HIPD) created by Chetty and colleagues (Chetty et al., 2016). Those data – the result of linking 1.4 billion tax records to Social Security Administration records – contain information on income for the period 1999-2014 by U.S. counties and commuting zones.[3] 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 1980 and 1982 and their parents' income.[4] First, we use the index of relative mobility (IRM or *rank-rank slope*) at the county level that is the correlation between children's income rank – within a birth cohort – and their parents' income rank.[5] The relative income mobility indicator ranges between -1 and 1, and larger values correspond to lower income mobility (i.e., higher rank-rank correlation between parents' and child's income). We also use an absolute upward mobility score or "the mean rank (in the national income distribution) of children whose parents are at the 25th percentile of the national parent income distribution" (Chetty et al., 2014, p. 7).[6] Absolute upward income mobility ranges from 0 to 1, and higher values correspond to larger 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 health are the same. Finally, we use the Gini coefficient as an indicator of income inequality.

The second database is the National Longitudinal Survey of Youth 1997 (NLSY97), a nationally representative sample of 8,984 American youth born between 1980 and 1984. Surveys were conducted annually, beginning in 1997 when the youth were between 12 and 18 years of age. In the first round, both the eligible youth and one of their parents were administrated personal interviews. The restricted NLSY97 geocoded data file contains information on the geographic residence of each respondent since age 12, allowing us to merge it with Chetty's county level income mobility measures. Importantly, the NLSY 97 sample matches the cohorts of the core sample used by Chetty et al. (2014) (1980-1982), so we can align better the timing of early exposure to the place's

[3] Chetty et al. (2014)'s core sample data include children who (1) have a valid Social Security number or individual taxpayer identification number, (2) were born between 1980 and 1982, and (3) are U.S. citizens as of 2013. There are approximately 10 million children in the core sample.

[4] 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 out-migrated as adults.

[5] Rank-rank slopes (or Spearman's correlation) 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 intergenerational income elasticity (of child income relative to parents' income) tend to be 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 in a given area has little impact on the national distribution.

income mobility. This under the assumption that the income mobility of this cohort measures the *socioeconomic mobility regime* to which this generation was exposed early in life and that may affect their health later. After merging the two databases, we kept 8,810 NLSY97 respondents. Only 174 respondents (2%) were removed from the analytic sample because income mobility information did not match the NLSY97 data. The total number of counties matched was 1607. Figure 4.1 in the *Methodological Supplement* shows counties included in the NLSY97 sample by income mobility, inequality, and the log of county's population. Figure 4.1 shows that counties included in the sample have a larger population, less variability and extreme values in the income mobility measures than the counties excluded. The distribution of income inequality is much more symmetric by population size. Although our analyses adjust for sampling weights and county's population size, we note our sample differs from the county composition of previous aggregate and individual level studies that have a higher county coverage. The potential consequences of this coverage difference for our results are discussed later in the *Conclusion and discussion* section.

The third database is the Panel Study of Income Dynamics (PSID), a nationally representative sample of U.S. men, women, children, and their families followed for more than 40 years. The PSID began interviewing a sample of about 5,000 families in 1968 and were re-interviewed each year through 1997 when the data collection became biennial. Similarly to the NLSY, restricted geographic data allow us to merge individual records with county income mobility measures. Unlike the NLSY 97, the PSID data permit us to estimate the effect of exposure to contextual mobility from birth to age 20. However, we lose statistical power because the number of respondents who match the Chetty et al. (2014)'s cohort is smaller. For instance, between 1975 and 1985, the PSID panel had 4,771 newborns.[7] Of these, 2,358 were the *reference person* or *spouse/partner* of the household at any time during their participation in the panel.[8] Although that cohort does not match exactly the cohort used by Chetty et al. (2014), it offers a reasonable approximation to the mobility regime exposure of that generation, provided income mobility does not change dramatically before 1980-82. After merging PSID and HIPD databases, we obtained 2,273 respondents.[9]. Only 85 respondents (4%) were removed because income mobility information did not match the PSID data. The total of counties matched was 1120, and the distribution of counties by income mobility, inequality, and population size looks similar to the NLSY97.[10] Even though the PSID analytical sample is considerably smaller, we think it is worth to estimate the effects of exposure from birth to age 20 years, and compare those results with the NLSY97.

Using different longitudinal data sources provides a broader picture to examine our research questions. It also offers a more precise definition of exposure to contextual income mobility as

[7]These newborns are PSID *gene* respondents. All 1968 sample members have the PSID *gene*, and they are followed in all subsequent waves across their entire lives, regardless of where they live. All individuals born to or adopted by somebody with the PSID *gene* acquires the gene themselves, and therefore are followed. Respondents who also were the household head or spouse/partner were asked most of our health outcomes overtime and had less missing data.

[8]The outcome variables included in our analysis were mostly asked to *reference persons* and their spouses or partners. That is why we only consider respondents who were a reference person or partner at least once during the observation period.

[9]We also used the PSID *Well being and Daily Life Supplement* 2016 complete missing data of variables such as depression symptoms in the PSID core database.

[10]Due to disclosure rules for restricted PSID data, we cannot show the scatter plot of individuals by county.

both of studies track respondents' county of residence during early life and over a relatively long period of time.

4.3 Analytical Strategy

This paper aims to estimate the effect of average exposure to county income mobility during childhood and adolescence on health outcomes such as smoking, BMI, self-reported health, and mental health during young adulthood. The key independent variable is the average income mobility exposure between ages 12 and 20 in the case of the NLSY97, and ages 1 to 20 for the PSID. Outcomes, in contrast, were measured during the last NLSY97 and PSID waves when respondents were in their thirties or forties. As a benchmark, we used both relative and absolute income mobility and estimated the effect of average county income inequality exposure (i.e., Gini coefficient), to compare the magnitude and direction of the associations. We used residualized income mobility and inequality scores from a county-level regression model that adjusted for characteristics such as population size, proportion of African-Americans, average household income, and income inequality (or income mobility). For completeness, we show the results with non-residualized exposure treatments in the *Methodological Supplement*.

We modeled the health outcomes as a function of duration-weighted exposures to different levels of county mobility regimes. By using inverse probability of treatment weighting (IPT), we emulated a counterfactual scenario in which we compared children with the same combination of *observed* covariate values during the exposure time, who did not select systematically into different county mobility regimes. Thus, we adjusted for confounding by time-varying covariates that might be affected by past treatment (Hernán et al., 2002; Hernán & Robins, 2006; Hernán et al., 2000; Wodtke et al., 2011), and generated a pseudo-population in which treatment was no longer confounded with measured covariates. Weights balance treatment assignment across prior confounders and give more or less weight to children with covariates histories that are underrepresented (or over-represented) in their current treatment group. To reduce the variability of weights, we used stabilized IPT weights (Hernán et al., 2000; van der Wal & Geskus, 2011). As the estimation of stabilized weights includes time-invariant covariates in the numerator and denominator, final outcomes models need to condition on time-invariant covariates in order to obtain unbiased estimates of the treatment.

As a sensitivity analysis, and because IPT weights using a continuous treatment are more sensitive to misspecification and outliers (Naimi et al., 2014; Thoemmes & Ong, 2016), we estimated weights for both continuous and categorical scores of income mobility and inequality. While we used linear regression in the first case, we ran ordinal logistic regressions to estimate the probability of exposure to county income mobility quintiles. Finally, following the strategy suggested by Dugoff et al. (2014), we included the sampling weights when computing IPT weights, multiplied them, and considered survey design variables (i.e., strata, clusters) and compound weights when estimating exposure models.

4.3.1 NLSY97

The NLSY97 has information of respondents' location (county) since age 12, so the exposure to county income mobility between ages 12 and 20 can be defined as:

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

We employed several covariates to adjust for potential confounding of county income mobility effects on health outcomes. Time-invariant covariates include race, gender, parents' education (years), age by the end of the study (categorical variable), the number of residential moves by age 12, the Armed Services Vocational Aptitude Battery (CAT-ASVAB) score, and mother's age at birth. Time-variants covariates, in turn, are inflation-adjusted family income (log), family size, the cumulative number of county changes, whether parents are employed and married, self-report health status, the number of days smoked in the last month, and BMI.[11] Table 4.5 in the *Methodological Supplement* shows descriptive statistics of the variables included in our models.

To estimate stabilized IPT weights in Time 1 (Age 12), we employed only time-invariant covariates. From Time 2 to 8, we used both time-invariant, baseline, and lagged time-variant covariates so that weights for later time points included all previous variables.[12] Table 4.7 in the *Methodological Supplement* shows descriptive statistics for the stabilized IPT weights for both continuous and categorical exposure treatments.

We used multiple imputation with multilevel models to address both item-specific non-response and attrition.[13] By design, respondents interviewed for the first time after their 12$^{\text{th}}$ birthday do not have information between age 12 and the age of the first interview. About 32% of the NLSY97 respondents had full exposure information (i.e., eight interviews from age 12 to 20). On average, respondents reported 6.5 years (out of 8), and only 7% of the sample participated in four or fewer years (e.g., older interviewees). When the county of residence was missing over the follow-up period, we imputed lost counties using most recent or earliest county of residence based on the evidence that most people do not change their county of residence often.[14] Matched NLSY97 counties with the HIPD data cover, on average, 5.9 out of 8 years of exposure, and only 4% of the

[11] The number of cigarettes smoked during the last month was asked only until 2011, that is why, we decided to use the number of days smoked during the last month. Moreover, rounds 4, 6, 8, 10, 12, 14 and 17 of the NLSY97 include a five-item short version of the Mental Health Inventory (MHI-5) to screen for depressive symptoms. Respondents reported the frequency of being nervous, feeling calm and peaceful, feeling downhearted and blue, being happy, and feeling so down in the dumps that nothing could cheer them up using a four-point scale to rate the frequency of their feelings. Because the MHI-5 was only measured in later rounds of the survey, we do not include that scale as a time-variant covariate.

[12] Details on model specification are available in https://github.com/sdaza/dissertation/tree/master/ch03.

[13] See **vanbuuren2018a** for an example of selective drop-out correction through multiple imputation.

[14] According to the U.S. Census Bureau migration estimates (Current Population Survey and Annual Social and Economic Supplement 1948-2019), 16% of the U.S. population changed their residence between 1999 and 2000. Of those, 56% remain in the same county (see https://www.census.gov/data/tables/time-series/demo/geographic-mobility/historic.html). In practice, we implemented the Last Observation Carried Forward (LOCF) and Next Observation Carried Backward (NOCB) methods.

sample have less than three years. At the end, we imputed missing records by creating 20 multiple imputed data-sets.[15]

We implemented different outcome models depending on the nature of the dependent variable. We estimated ordinal logistic regression for the effect of income mobility and inequality on *self-reported health status* (poor, fair, good, very good, excellent), Generalized linear models (GLM) for *BMI* and *depression symptoms*, logistic regression in the case *current smoking status*, and quasi-Poisson models (also called over-dispersion with quasi-likelihood) for the *number of days smoking in the last month*. Outcome models adjusted only for baseline and time-invariant covariates and took into account sampling design variables (strata, clusters) and weights.

4.3.2 PSID

The PSID sample includes newborns. Thus, we could define the average exposure to county income mobility from age 1 to 20 as:

$$\frac{\sum_{i=1}^{20} \text{county income mobility}_i}{20}$$

We included a relatively similar set of covariates as the NLSY97 sample. Time-invariant covariates involved race, gender, age by the end of the study, mother's age and marital status at birth, and weighed less than 55 pounds at birth. In turn, time-variants covariates were inflation-adjusted family income (log), family size, the cumulative number of county changes, head of household education, whether the head was employed, married, and owns the house where that family was living. Outcomes included self-report health status, BMI, depression, current smoking, and number of cigarettes smoked during the last month.[16] Unlike the NLSY97, PSID outcome variables were not measured systematically during the exposure period. Thus, we decided not to use outcomes as time-variant predictors when estimating IPT weights. Table 4.6 in the Methodological Supplement shows descriptive statistics of the variables used.

We followed the same procedure described in the previous section to estimate IPW weights. First, we computed stabilized IPT weights for Time 1 by including only time-invariant covariates. Then, we created weights from Time 2 to 20 using time-invariant and lagged time-variant covariates so that weights for subsequent time points include all previous variables. Table 4.8 in the *Methodological Supplement* shows descriptive statistics of the PSID stabilized IPT weights

Again, we used multiple imputation with multilevel models (20 multiple imputed data-sets). When the county of residence was missing during the exposure period, we employed LOCF and NOCB methods. About 44% of the PSID sample moved to a different county during the exposure

[15]For a discussion and assessment of our multiple imputation models, see the *Methodological Supplement*.

[16]The PSID screens mood or anxiety disorder using the Kessler Psychological Distress Scale (K6) in 2001-2003, 2007-2017. The scale includes six items: *During the past 30 days, about how often did you feel nervous, hopeless, restless or fidgety, so depressed that nothing could cheer you up, that everything was an effort, worthless.*

time. On average, matched counties with the HIPD data cover 17.3 out of 20 years of exposure, and only 1.4% of the whole sample report less than seven years of exposure.

Lastly, we used different outcome models depending on the nature of the dependent variable: ordinal logistic regression when estimating the effect of income mobility and inequality on *self-reported health status*, Generalized linear models (GLM) for *BMI* and *depression symptoms*, logistic regression in the case *current smoking status*, and quasi-Poisson models for the *number of cigarettes smoked during the last month*. The outcome models adjusted only for baseline and time-invariant covariates, and considered sampling design variables (strata, clusters) and weights.

4.4 Results

4.4.1 Sample characteristics

Tables 4.5 and 4.6 in the *Methodological Supplement* show descriptive statistics of our analytical samples and the proportion of missing data by variable. These tables provide insights about differences regarding design, composition, and length of exposure in NLSY97 and PSID samples. For instance, among NLSY97 respondents, the first interview was, on average, at age 14 (min 12, max 18), while the last interview was at age 33. This contrasts with the PSID sample whose respondents entered the study since they were born, and had their last interview at age 37 on average (min 30, max 47). Due to these differences in measurement, the number of residential changes is also different between samples. Whereas the proportion of NLSY97 respondents who moved to another county during the observation period was 27%, 44% of PSID interviewees have changed their residential county in 20 years.

In addition to these differences, Tables 4.5 and 4.6 show the PSID sample has slightly more White respondents than the NLSY97 (59% versus 52%). However, on average, the PSID respondents seem to have lived in counties with a higher proportion of African-Americans (19% versus 15% among NLSY97 respondents). The nature of some covariates also differ. For example, while parents' education is time-invariant in NLSY97 (only measured at the baseline), the PSID recorded that variable over time (i.e., time-variant).

The outcome variables – measured at the end of the follow-up period – show relatively similar values in both samples, except for current smoking. The self-reported health scale (1-5) is around 3.5 points in both samples, while the BMI ranges between 28.7 and 28.9, and the proportion of respondents currently smoking is higher in the NLSY97 (30%) than in the PSID (20%). The remaining outcome variables (depressive symptoms and smoking intensity) are not strictly comparable. The proportion of missing data in the outcome variables ranges between 20% and 31%, and reaches its maximum in BMI (31% in the PSID, and 24% in the NLSY97).

For the rest of the covariates, missing data are considerably lower in the PSID than in the NLSY97. While the PSID's highest proportion of missing cases is observed in the variable *weight of the respondent when was born* (12%), the NLSY97 has considerably higher levels of missing

data, especially in time-variant variables such as household income (69%), family size (35%), and parents' working status (32%). This pattern is due, in part, to the design of the NLSY97 study. As the observation window did not always start at age 12, when the first interview was after age 12, no information was collected on several covariates between age 12 and the age of the first interview. In those cases, we had to use retrospective parents' reports to determine where respondents lived when they were 12 years old and imputed missing covariates during that period.

We note that, although we compare the results from these two datasets, the descriptive tables 4.5 and 4.6 show relevant differences in design, composition, and exposure in the NLSY97 and PSID analytical samples that need be considered when interpreting our findings.

4.4.2 IPT Weights

Tables 4.7, 4.8, 4.11 and 4.14 in the *Methodological Supplement* show descriptive statistics of the stabilized IPT weights separated by sample and type of exposure variable: income mobility or income inequality, continuous or categorical, residualized or non-residualized. These descriptives come from IPT weights estimated using 20 different datasets with imputed data, and linear or ordinal logistic regression depending on the nature of the exposure variables (continuous or categorical). Because we adjusted attrition through imputation, we did not compute attrition weights.

IPT weights exhibit desirable properties when observed means are close to one, and they have small variance. Tables 4.7, 4.8, 4.11 and 4.14 show that all estimated weights are well-behaved and centered around one (ranging from 0.98 to 1.08). We found, however, substantial differences regarding the variability of the IPT weights. First, as expected, the variability of weights was much higher when using a continuous exposure treatment than a categorical one (quintile). Second, very high standard deviations of weights were mostly due to outliers. For instance, Tables 4.11 and 4.14 show that the standard deviation of the continuous version of the Gini coefficient and upward mobility are considerably large. However, once weights are truncated at the 1^{th} and 99^{th} percentiles, weights become stable, and standard deviations decrease considerably. Thus, we decided to use truncated weights in order to improve the efficiency of estimates and avoid the disproportionate influence of extreme observations (Hernán & Robins, 2006; Thoemmes & Ong, 2016; van der Wal & Geskus, 2011).

4.4.3 NLYS97 estimates

We estimated four sets of models based on the following categories: unadjusted or adjusted, continuous or categorical exposure. Unadjusted models provide *naive* estimates by regressing exposure on outcomes without adjustments and IPT weighting, except for sampling weighting. Within each set of models, we ran independent models for each exposure variable: relative income mobility, absolute income mobility, and income inequality.

Table 4.1 shows the coefficients of average residualized exposure on health outcomes for

NLSY97. The first three rows in Table 4.1 show the *naive* association of average county exposure from age 12 to 20 with five health outcomes. All the exposure treatments represent a negative trait, so we expect adverse consequences for health. To keep consistency with that interpretation, we multiplied *upward mobility* by -1, so that any increase in exposure would consist of a negative condition (i.e., less income mobility, more rigidity of the stratification system, more inequality).

The rank-rank score coefficient (relative income mobility) for self-reported health status is 0.02 (SE = 0.04). Because that coefficient comes from an ordinal logistic regression, an increase in one standard deviation on the average exposure to a rigid stratification enviroment implies an increment of 2% ($exp(0.02) = 1.02$) in the odds of reporting excellent health (versus bad health)[17]. Similar coefficients are observed regarding upward mobility (i.e., absolute income mobility) and income inequality (Gini coefficient). However, those estimates are very imprecise and noisy. Coefficients regarding BMI and depression symptoms in Table 4.1 are easier to interpret. An increase of one standard deviation in the rank-rank score rises BMI by 0.04 points, and the depression scale by 0.02 points. Again, these estimates are very imprecise and switch their sign in a non-systematic fashion.

Smoking models show more systematic associations. Both increases in the rank-rank correlation or lack of upward mobility raise the odds ratio of smoking later in life by 25% and 23%, respectively ($\beta_{\text{rank}} = 0.22$, $\text{SE}_{\text{rank}} = 0.04$, $\beta_{\text{upward}} = 0.21$, $\text{SE}_{\text{upward}} = 0.05$). Surprisingly, the naive association between the Gini coefficient and current smoking is negative. Similar associations are observed when modeling the number of days smoking during the last day. The quasi-Poisson coefficients suggest increasing the rank-rank correlation or reducing upward mobility raise the incidence rate ratio by 23% and 22%. Again, and contrary to our expectations, the naive Gini coefficient suggests a negative relationship between income inequality and days smoked during the last month.

In sum, unadjusted point estimates for income mobility were relatively small and not systematic across health outcomes. Only smoking behavior seems to have a systematic association with exposure to income mobility in the expected direction (the higher the income mobility, the better the health outcome). Depression, self-report health status, and BMI estimates, instead, are small and uncertain. As a sensitivity analysis, we estimated unadjusted models using non-residualized exposure variables. In that case, the association between exposure and health outcomes can be spurious as income mobility and inequality might relate to counties' characteristics that also impact health. Table 4.9 in the *Methodological Supplement* shows unadjusted models with non-residualized exposure variables. The patterns are relatively similar to the residualized exposure variable models. Most of the systematic associations are observed between income mobility and smoking, but also BMI.

The estimates discussed above might be biased because of non-random selection into counties (residential mobility) and confounding. The next set of results comes from models using a weighted pseudo-population in which county exposure at each wave is independent of prior time-varying covariates. The second section of Table 4.1 (*adjusted models*) shows IPT-weighted esti-

[17]The proportional odds assumption in original logistic models is not simply that the odds are the same, but that odds ratios are the same across categories.

mates of the effect of standardized income mobility and inequality exposure on health outcomes. Under assumptions of no unmeasured confounders, no model misspecification, and positivity – there is a non-zero probability of treatment for every level and combination of confounders (Hernán & Robins, 2006) – stabilized IPT weighting provides unbiased estimates of average causal effects.

Most of the estimates are small and very imprecise to claim they are systematically positive or negative. Relative income mobility estimates on smoking and BMI seem slightly more precise, even after IPT weighting reduces them in about half with respect to the *unadjusted estimates*. We should note, though, that the standard errors in Table 4.1 are underestimated as we are using several outcomes and making multiple comparisons. Consequently, there is a higher chance of finding false positives. Moreover, given the small and noisy BMI estimate in the *unadjusted* model, the positive coefficient ($\beta_{\text{BMI}} = 0.33$, SE = 0.16) in the *adjusted* model, still imprecise, must be interpreted with caution.

Finally, IPT weights using continuous exposure are sensitive and unstable due to parametric misspecification and outliers. Thus, we also estimated the effect of income mobility and inequality using a categorical version of exposure (quintile). Table 4.2 shows both unadjusted and adjusted models by health outcome. Although the pattern of the coefficients is similar to Table 4.1, estimates tend to be – as expected – more precise and smaller due to the change in the scale of exposure (1 to 5). In some cases, coefficients even switch their sign. For instance, the unadjusted smoking coefficients for upward mobility and depression symptoms were, as expected, positive, but they become negative when using the categorical version of income upward mobility. Similar to Table 4.1, smoking estimates and BMI reveal a systematic relationship with *relative income mobility*. For instance, exposure to the most rigid stratification level (5) compared to counties in the 3$^{\text{rd}}$ quintile (average), increases the odds of smoking by about 17% ($exp((5-3) \times 0.08) = 1.17$). Again, although BMI has a positive and relatively precise coefficient, it only appears in the adjusted models what suggests it is not systematic.[18]

4.4.4 PSID estimates

The same set of models was estimated with the PSID sample. Although the period of exposure in this case is longer (from birth to age 20), the sample size of the 1975-1980 cohort is substantially smaller (2,273 respondents versus 8,810 in the NLSY97). Table 4.3 shows unadjusted and adjusted models for residualized income mobility and inequality on health outcomes. In contrast to the NLSY97 results, most of the non-adjusted coefficients are very imprecise and noisy. The only stable coefficients are those related to current smoking and number of cigarettes. The adjusted results in the bottom section of Table 4.3, in turn, show effects that, although most of the time in the expected direction, are so imprecise to suggest either positive or negative consequences for health.[19] Using a categorical version of exposure do not change results or improve estimates (see Table 4.4). Similar to the NLSY97 analysis, we estimated the model using non-residualized

[18]Table 4.10 in the *Methodological Supplement* shows unadjusted models with non-residualized and categorical exposure variables. They show a similar pattern to the models already discussed.

[19]Similar results were obtained when redefining the cohort of respondents (e.g., those born between 1970 and 1985) in order to increase the sample size, at the cost of adding imprecision to the exposure measures.

exposure variables to examine how sensitive our results were to aggregate adjustments of income inequality and mobility indexes. Tables 4.12 and 4.13 in the *Methodological Supplement* show the effects of exposure to a rigid or unequal stratification environment are non-systematic and noisy, even in the non-adjusted models.

4.5 Conclusion and discussion

This paper aims to estimate the effect of average exposure during childhood and adolescence to a rigid and unequal stratification environment on health outcomes and behaviors during adulthood. Thus, we extend previous research on the association between income mobility, health, and mortality, and assess the hypothesis that early-life exposure to a given income mobility regime may determine health later and explain, at least in part, the longevity gaps observed across places in the U.S.

Our analysis suggests the connection between income mobility and health is not as systematic as previous research shows. Our most robust effects are related to smoking behavior (i.e., currently smoking, number of days smoked in the last month), although only for NLSY97 and *relative income mobility* (rank-rank correlation). This particular finding implies income mobility might directly affect behaviors critically associated with modern chronic illnesses, such as smoking uptake and desistance, alcohol consumption, substance abuse, choices of diet and physical activity, as theory indicates. However, we were not able to replicate these findings using the PSID sample that comprehensively measures exposure from birth to age 20, and where selection bias could be better reduced by using IPT weights. Surprisingly, not even our naive coefficients – without adjustments – were strong or systematic enough. The association between income inequality and health outcomes did not hold, either.

The relative consistency between unadjusted and adjust models suggests selection is not the only reason there might be inconsistencies between previous aggregate and cross-sectional results and our findings. Although adjustment does reduce the NLSY97 smoking coefficients, unadjusted models do not seem reveal a systematic association between average exposure to economic opportunities and health outcomes as previous research shows (Venkataramani et al., 2016; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020). There might be two explanations for these findings.

First, our results might be affected by measurement error. As recently Mogstad et al. (2020) have pointed out, income mobility measures and rankings computed by Chetty et al. (2014)'s and colleagues are estimates rather than *true values*, so they might carry considerable uncertainty as population size varies considerably across counties. Figure 4.1 in the *Methodological Supplement* provides some evidence on how measurement error might affect our results. Figure 4.1 shows most of the counties in the NLSY97 sample have a larger population than those counties excluded. This is expected given the usual sampling design of nationally representative samples. Given the population size of counties, we would expect higher uncertainty of estimates in the small counties. Figure 4.1 shows smaller counties do actually have the most extreme income mobility values,

likely due to higher uncertainty. This seems to be especially the case of absolute income mobility (the correlation between population and upward mobility is around -0.37), although relative income mobility has also the most extreme values among smaller counties except that they are evenly distributed across positive and negative values. Thus, by mostly including bigger counties, the NLSY97 and PSID sampling scheme is excluding uncertain income mobility estimates from smaller counties. We agree that this is far from an optimal strategy to account for measurement error when estimating the effect of the county economic opportunity environment on health, but unfortunately, we do not have access to the standard errors of the income mobility estimates at the county level.

In comparison, Venkataramani et al. (2016)'s pool several cross-sectional samples from the Behavioral Risk Factor Surveillance Survey (BRFSS), reaching nearly 147,000 individuals of ages 25 to 35. That sample covers 2242 counties, which represents about 78% of all the counties used by Chetty et al. (2014) to estimate income mobility. The same is true concerning aggregate analyses by Venkataramani et al. (2015) and Daza and Palloni (2018), where almost all the counties used by Chetty et al. (2014) were included in the analysis. In contrast, the NLSY97 and PSID samples cover only 55% and 39% of the counties, respectively. Although we do not know what would happen to previous research results if only the counties of NLSY97 and PSID samples were considered, or better, if the measurement error were considered in the analysis, our findings and the work by Mogstad et al. (2020) suggest that measurement error might exaggerate estimates. Future research will be needed to assess the consequences of measurement error thoroughly, provided the standard errors of income mobility estimates are available.

A second way to interpret our findings stresses the complexity of the effects being estimated. The mechanisms we outlined when discussing the reasons why we should expect a causal relationship between the stratification system and health are not simple. The size of these effects is probably small, and they might be relevant to specific groups of the population and not others (i.e., heterogeneity). For instance, an individual's family mobility experience might be equally influential and may offset harmful effects stemming from a place's income mobility. In this context, estimating the population's average effect would not necessarily provide an unbiased and robust estimate. We are also assuming – due to data limitations – that the aggregate level that matters is counties, but it perfectly possible that the adequate level when estimating these exposure effects is neighborhoods. These factors, in addition to measurement error, make it difficult to estimate long-term implications of society's opportunity system for health. Our results do not necessarily indicate the causal link between income mobility and health does not exist, but that our data and analytical strategies are not strong enough to show they are systematic and in the expected direction.

Some additional limitations of our specific analysis should be noted. First, although IPT-weighted estimates avoid some problems associated with conditioning on observed time-varying confounders, selection bias may still occur if unobserved factors simultaneously affect decisions about where to live and health behavior. Unfortunately, the assumption of no unobserved confounding is not testable with observed data, despite adjusting for an extensive set of observed covariates. Second, although we use different specifications and results were relatively stable (not shown), treatment models may still be misspecified. We also need to assume a positive probability

of treatment for every level and combination of prior confounders. Theoretically, there is no reason to expect zero treatment probabilities across a set of covariates over time subgroups, except for the inherent limitations of sampling. Third, we assume that measuring income mobility in a cohort – a measure that necessarily realizes in the future – accounts for the latent socioeconomic rigidity to which people were exposed early in life. It is possible that what really matters is the income mobility of the previous generation, as those experiences would determine the socialization and investments of the next generation. Finally, we imputed missing values and adjusted attrition using multiple imputation. Even though we obtained reasonable values and distributions, it is still possible that our imputation models are misspecified, and assumptions such as *missing at random* (MAR) do not hold.

Overall, our paper is the first to provide individual estimates of the effect of income mobility on health using a precise definition of exposure and accounting for selection and time-varying confounders. Thus, by focusing on individual outcomes during adulthood and influences during early formative years, we assess more directly the hypothesis that growing up in a community with a rigid stratification system may discourage adoption of behaviors that provide immediate rewards but are highly noxious, difficult to abandon, and bearers of large effects on health that take a long time to manifest. Our results suggest a weak connection between income mobility and health, and a non-systematic link between income inequality and health outcomes, even using two different datasets. Selection and measurement error might exaggerate estimates from previous research, but it is also possible that income mobility effects are relatively *small* and heterogeneous, making it difficult to estimate them precisely. Future research should focus on finding new indicators of socioeconomic mobility, both at the individual and appropriate aggregate level, to assess the magnitude of the consequences of the society system of opportunities for health.

4.6 Tables and Figures

Table 4.1: Estimates of average residualized continuous exposure on health indicators, NLSY97

	Health status	BMI	Depression	Smoking	Days smoking last month
Unadjusted models					
Rank-rank	0.02	0.04	0.02^{*}	0.22^{***}	0.21^{***}
	(0.04)	(0.13)	(0.01)	(0.04)	(0.04)
Upward mobility × -1	0.04	-0.04	0.02^{*}	0.21^{***}	0.20^{***}
	(0.04)	(0.16)	(0.01)	(0.05)	(0.05)
Gini	0.01	-0.08	-0.00	-0.15^{***}	-0.16^{***}
	(0.03)	(0.11)	(0.01)	(0.03)	(0.03)
Adjusted models					
Rank-rank	-0.03	0.33^{*}	0.01	0.12^{**}	0.11^{**}
	(0.04)	(0.16)	(0.01)	(0.05)	(0.04)
Upward mobility × -1	0.02	0.11	0.01	0.07	0.05
	(0.05)	(0.15)	(0.01)	(0.05)	(0.04)
Gini	0.02	-0.07	-0.01	-0.05	-0.07^{*}
	(0.04)	(0.11)	(0.01)	(0.04)	(0.03)
Individuals	8810	8810	8810	8810	8810

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 12 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (days smoking last month). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.2: Estimates of average residualized categorical (quintile) exposure on health indicators, NLSY97

	Health status	BMI	Depression	Smoking	Days smoking last month
Unadjusted models					
Rank-rank	0.01	0.07	0.01	0.11^{***}	0.11^{***}
	(0.02)	(0.07)	(0.01)	(0.02)	(0.02)
Upward mobility × -1	−0.01	0.00	-0.01^{*}	-0.08^{***}	-0.08^{***}
	(0.02)	(0.08)	(0.01)	(0.02)	(0.02)
Gini	0.02	−0.04	−0.00	-0.10^{***}	-0.10^{***}
	(0.02)	(0.07)	(0.00)	(0.02)	(0.02)
Adjusted models					
Rank-rank	−0.01	0.18^{*}	0.00	0.08^{***}	0.07^{***}
	(0.02)	(0.07)	(0.01)	(0.02)	(0.02)
Upward mobility × -1	−0.01	−0.05	−0.01	−0.03	−0.02
	(0.02)	(0.07)	(0.01)	(0.02)	(0.02)
Gini	0.03	−0.04	−0.00	−0.04	-0.05^{**}
	(0.03)	(0.08)	(0.00)	(0.03)	(0.02)
Individuals	8810	8810	8810	8810	8810

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 12 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (days smoking last month). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.3: Estimates of average residualized continuous exposure on health indicators, PSID

	Health status	BMI	Depression	Smoking	Cigarettes smoked
Unadjusted models					
Rank-rank	−0.03	−0.10	0.03	0.26*	0.26*
	(0.08)	(0.32)	(0.03)	(0.13)	(0.13)
Upward mobility × -1	−0.10	0.40	0.04	0.31	0.37*
	(0.10)	(0.36)	(0.04)	(0.20)	(0.18)
Gini	0.11	−0.34	−0.01	−0.15	−0.16*
	(0.08)	(0.29)	(0.02)	(0.09)	(0.08)
Adjusted models					
Rank-rank	−0.01	−0.15	0.03	0.10	0.18
	(0.09)	(0.26)	(0.04)	(0.15)	(0.14)
Upward mobility × -1	0.01	0.09	0.03	0.05	0.14
	(0.10)	(0.32)	(0.04)	(0.19)	(0.17)
Gini	0.13	−0.12	−0.00	−0.04	−0.08
	(0.09)	(0.28)	(0.02)	(0.11)	(0.08)
Individuals	2273	2273	2273	2273	2273

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 1 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (cigarettes smoked). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.4: Estimates of average residualized categorical (quintile) exposure on health indicators, PSID

	Health status	BMI	Depression	Smoking	Cigarettes smoked
Unadjusted models					
Rank-rank	−0.00	−0.01	0.01	0.09	0.09
	(0.05)	(0.17)	(0.02)	(0.07)	(0.07)
Upward mobility × -1	0.04	−0.34	−0.01	−0.16*	−0.20**
	(0.05)	(0.18)	(0.02)	(0.08)	(0.07)
Gini	0.08	−0.32*	−0.00	−0.08	−0.06
	(0.05)	(0.15)	(0.01)	(0.06)	(0.05)
Adjusted models					
Rank-rank	0.03	−0.09	0.01	0.03	0.03
	(0.05)	(0.13)	(0.02)	(0.08)	(0.07)
Upward mobility × -1	−0.01	−0.13	−0.01	−0.06	−0.10
	(0.05)	(0.18)	(0.02)	(0.08)	(0.07)
Gini	0.09	−0.23	−0.00	0.00	0.00
	(0.05)	(0.14)	(0.01)	(0.06)	(0.05)
Individuals	2273	2273	2273	2273	2273

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 1 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (cigarettes smoked). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Methodological Supplement

The code to create analytic data sets, models and plots is available at: https://github.com/sdaza/dissertation/tree/master/ch03. Some of the variables in the paper are restricted and obtained under special contractual arrangements to protect the anonymity of respondents. These data are not available from the authors. Those interested in obtaining PSID restricted data should contact PSIDHelp@isr.umich.edu. Those interested in the NLSY restricted data, visit www.bls.gov/nls/geocodeapp.htm.

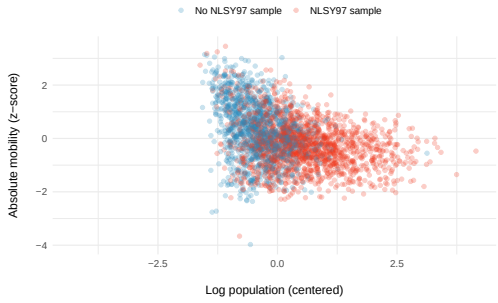
County coverage

Figure 4.1 displays counties by the log of population and measures of income mobility and inequality, highlighting in red the counties included in the NLSY sample.

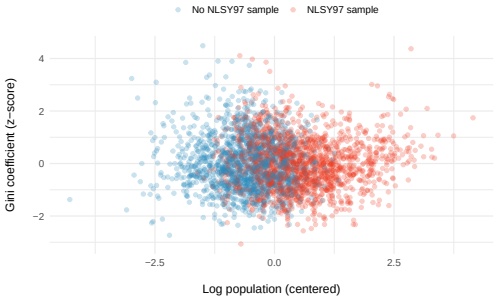
Figure 4.1: County income mobility and inequality over population by NLSY97 sample coverage

(a) NLSY97 Relative mobility

(b) NLSY97 Absolute mobility



(c) NLSY97 Inequality



This figure provides insights on county coverage of the NLSY97 individual sample, and the relationship between the size of counties and the values of income mobility and inequality. Due to disclousure rules for restricted data, we cannot publish the same plots for the PSID sample. However, the patterns displayed in Figure 4.1 are similar to what we observed in the PSID sample.

Descriptive statistics

Tables 4.5 and 4.6 show descriptive statistics of the variables in our analysis. The PSID table cannot show min and max values due to disclosure rules for restricted data.

Table 4.5: NLSY97 descriptive statistics of covariates and outcomes

	Mean	SD	Min	Max	% Missing	Valid observations
Time-invariant covariates						
Male	0.51	0.50	0.00	1.00	0.00	8810
Age first interview	14.35	1.49	12.00	18.00	0.00	8810
Age last interview	32.88	1.45	30.00	36.00	0.00	8810
Race-Ethnicity						
White	0.52	0.50	0.00	1.00	0.00	8810
Black	0.26	0.44	0.00	1.00	0.00	8810
Hispanic	0.21	0.41	0.00	1.00	0.00	8810
Mixed	0.01	0.10	0.00	1.00	0.00	8810
ASVAB Test Score	45.38	29.17	0.00	100.00	0.21	8810
Parent's Education (years)	13.15	3.06	1.00	20.00	0.07	8810
Mother's age at birth of respondent	25.48	5.39	12.00	54.00	0.07	8810
Number of residential moves by age 12	3.17	2.75	1.00	40.00	0.13	8810
Proportion moved to a different county	0.27	0.44	0.00	1.00	0.00	8810
Time-variant covariates						
Family size	4.26	1.65	1.00	17.00	0.35	70480
Respondent living with any parent	0.82	0.39	0.00	1.00	0.29	70480
Parent is working	0.89	0.31	0.00	1.00	0.32	70480
Parent is married	0.65	0.48	0.00	1.00	0.31	70480
Log household income	-0.11	2.42	-10.40	2.98	0.69	70480
County log income	0.74	1.08	-3.07	4.32	0.00	70480
County log population	1.76	1.15	-1.62	4.15	0.00	70480
County proportion Black	0.15	0.16	0.00	0.80	0.00	70480
Cumulative number of county moves	0.15	0.47	0.00	6.00	0.00	70480
Exposure variables						
County rank-rank correlation (original)	0.26	0.07	0.04	0.53	0.00	70480
Quintile county rank-rank correlation (original)	2.88	1.37	1.00	5.00	0.00	70480
Residualized county rank-rank correlation	-0.29	0.76	-3.21	2.50	0.00	70480
Quintile residualized county rank-rank correlation	2.52	1.30	1.00	5.00	0.00	70480
County upward mobility (original)	0.44	0.05	0.33	0.67	0.00	70480
Quintile county upward mobility (original)	2.18	1.16	1.00	5.00	0.00	70480
Residualized county upward mobility	0.30	0.64	-2.66	2.34	0.00	70480
Quintile residualized county upward mobility	3.59	1.33	1.00	5.00	0.00	70480
County Gini coefficient (original)	0.45	0.04	0.34	0.60	0.00	70480
Quintile county Gini coefficient (original)	3.45	1.38	1.00	5.00	0.00	70480
Residualized county Gini coefficient	0.21	0.92	-2.84	4.99	0.00	70480
Quintile Residualized county Gini coefficient	3.39	1.45	1.00	5.00	0.00	70480
Outcomes						
Self-reported health	3.63	1.00	1.00	5.00	0.21	8810
BMI	28.70	5.96	15.00	40.00	0.24	8810
Depressive symptoms	1.83	0.50	1.00	4.00	0.21	8810
Current smoking	0.31	0.46	0.00	1.00	0.22	8810
Days smoked	6.21	11.60	0.00	30.00	0.22	8810

Note: Statistics based on non-imputed data. SD = Standard deviation. Observations correspond to respondents in the case of time-invariant and outcome variables, and person-years (N times exposure) for time-variant variables. Outcomes were measured in 2015.

Table 4.6: PSID descriptive statistics of covariates and outcomes

	Mean	SD	% Missing	Observations
Time-invariant covariates				
Male	0.46	0.50	0.00	2273
Age last interview	37.03	3.25	0.00	2273
Birth year	1980.23	3.18	0.00	2273
Race-Ethnicity				
White	0.59	0.49	0.00	2273
Black	0.39	0.49	0.00	2273
Other	0.02	0.14	0.00	2273
Weighted less than 55 oz	0.07	0.26	0.12	2273
Mother marital status at birth	0.76	0.43	0.04	2273
Mother's age at birth of respondent	25.18	4.95	0.00	2273
Proportion moved to a different county	0.44	0.50	0.00	2273
Time-variant covariates				
Family size	4.30	1.38	0.06	45460
Respondent living with any parent	0.73	0.45	0.06	45460
Parent's years of education	12.87	2.44	0.07	45460
Parent is working	0.61	0.49	0.06	45460
Parent is married	0.83	0.38	0.06	45460
Log household income	0.09	1.13	0.06	45460
County log income	0.64	1.03	0.00	45460
County log population	1.57	1.14	0.00	45460
County proportion Black	0.19	0.19	0.00	45460
Cumulative number of county moves	0.55	1.07	0.00	45460
Exposure variables				
County rank-rank correlation (original)	0.28	0.07	0.00	45460
Quintile county rank-rank correlation (original)	3.27	1.34	0.00	45460
Residualized county rank-rank correlation	-0.20	0.73	0.00	45460
Quintile residualized county rank-rank correlation	2.63	1.36	0.00	45460
County upward mobility (original)	0.43	0.05	0.00	45460
Quintile county upward mobility (original)	2.12	1.21	0.00	45460
Residualized county upward mobility	0.25	0.59	0.00	45460
Quintile residualized county upward mobility	3.56	1.28	0.00	45460
County Gini coefficient (original)	0.45	0.04	0.00	45460
Quintile county Gini coefficient (original)	3.46	1.39	0.00	45460
Residualized county Gini coefficient	0.11	0.83	0.00	45460
Quintile Residualized county Gini coefficient	3.23	1.40	0.00	45460
Outcomes				
Self-reported health	3.53	0.99	0.23	2273
BMI	28.91	5.90	0.31	2273
Depressive symptoms	1.63	0.67	0.23	2273
Current smoking	0.20	0.40	0.23	2273
Number of cigarettes	2.15	5.35	0.23	2273

Note: Statistics based on non-imputed data. SD = Standard deviation. Observations correspond to respondents in the case of time-invariant and outcome variables, and person-years (N times exposure) for time-variant variables. Outcomes were measured in 2017.

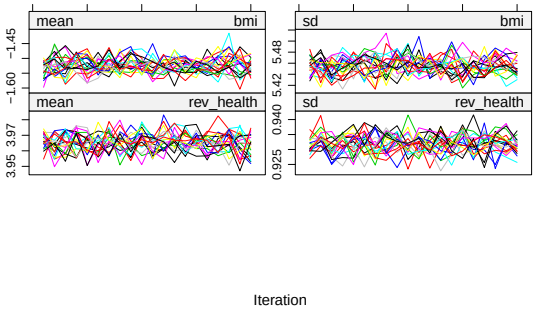
Imputation

We employed multiple imputation for item non-response and attrition. For each exposure variable (e.g., relative and absolute income mobility), we ran multilevel models to impute values for both time-variant and invariant covariates. We produced 20 complete data-sets and pooled the results using Rubin's Rules (**vanbuuren2018a**).

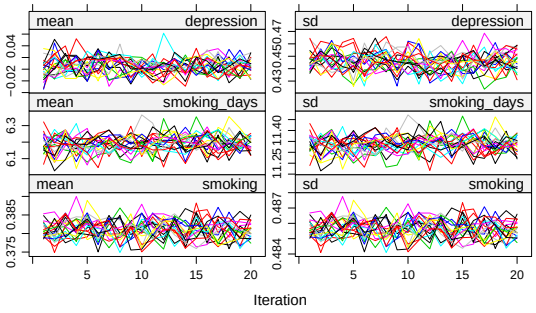
Multiple imputation model specifications are available in our code repository. For each exposure variable, we produced 20 complete datasets per data source (PSID and NLSY9). Different imputed data-sets were created for residualized and non-residualized, continuous and categori-

Figure 4.2: NLSY97 Imputation plots with relative mobility as exposure, 20 iterations

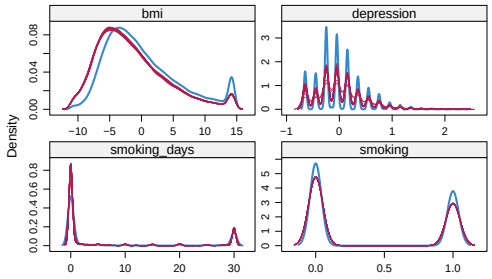
(a) Iterations



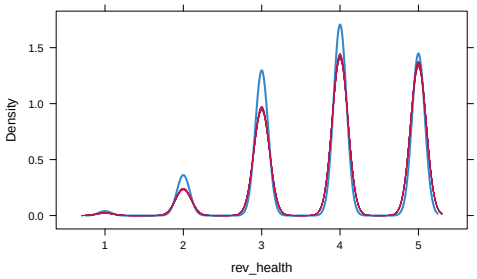
(b) Iterations



(c) Imputations vs Observed Values



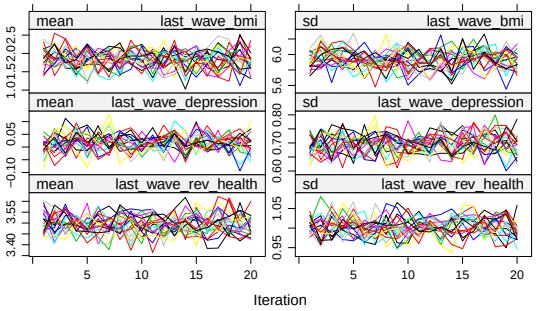
(d) Imputations vs Observed Values



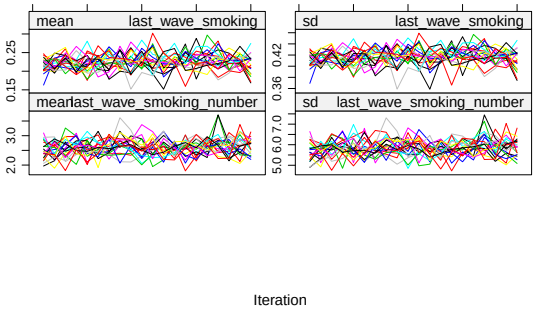
cal exposure (360 datasets in total). We assessed convergence and feasibility of results using the criteria suggested by **vanbuuren2018a**. For instance, Figure 4.2 and 4.3 show iteration plots of outcomes variables (a and b) and the comparison between observed and imputed distribution of outcomes (d and c). In general, convergence plots looked fine as they mix not systematically after 20 iterations. The distribution of outcomes also seemed reasonable, and no ill behavior of estimates was observed.

Figure 4.3: PSID Imputation plots with relative mobility as exposure, 20 iterations

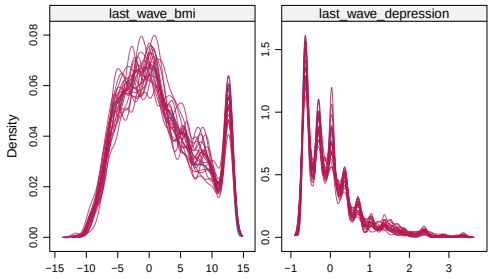
(a) Iterations



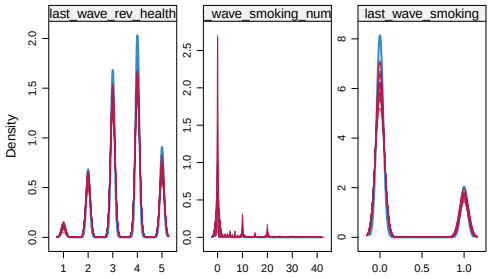
(b) Iterations



(c) Imputations vs Observed Values



(d) Imputations vs Observed Values



IPT weights (residualized models)

Table 4.7: NLSY97 Stabilized treatment weights (residualized)

			Percentiles			
Weight	Mean	SD	1st	25th	75th	99th
Continuous exposure						
Rank-rank	1.02	0.72	0.28	0.81	1.09	3.07
Upward mobility	1.02	0.48	0.34	0.82	1.09	2.80
Gini	1.02	1.01	0.32	0.84	1.05	3.04
Categorical (quintile) exposure						
Rank-rank	1.00	0.38	0.49	0.83	1.08	2.58
Upward mobility	1.00	0.34	0.54	0.82	1.10	2.27
Gini	1.00	0.41	0.45	0.80	1.10	2.56

Analyses based on exposure from 12 to 20 years old. Statistics based on 20 multiple imputed datasets.

Table 4.8: PSID Stabilized treatment weights (residual exposure)

			Percentiles			
Weight	Mean	SD	1st	25th	75th	99th
Continuous exposure						
Rank-rank	1.03	0.78	0.34	0.83	1.10	2.75
Upward mobility	1.03	0.90	0.45	0.87	1.08	2.11
Gini	1.00	0.28	0.43	0.89	1.06	2.00
Categorical (quintile) exposure						
Rank-rank	1.00	0.29	0.55	0.82	1.13	1.90
Upward mobility	1.00	0.25	0.59	0.86	1.10	1.73
Gini	1.00	0.24	0.53	0.88	1.09	1.81

Analyses based on exposure from 1 to 20 years old. Statistics based on 20 multiple imputed datasets.

Non-residualized models

For completeness, we show the results with non-residualized exposure treatments and IPT weight statistical descriptives for both the NLSY97 and PSID.

NLSY97

Table 4.9: Estimates of average continuous exposure on health indicators, NLSY97

	Health status	BMI	Depression	Smoking	Days smoking last month
Unadjusted models					
Rank-rank	−0.04	0.39***	0.00	0.13***	0.15***
	(0.03)	(0.11)	(0.01)	(0.04)	(0.03)
Upward mobility × -1	−0.04	0.30**	−0.00	−0.03	−0.02
	(0.03)	(0.11)	(0.01)	(0.04)	(0.04)
Gini	−0.02	0.16	−0.01	−0.12***	−0.12***
	(0.03)	(0.09)	(0.01)	(0.03)	(0.03)
Adjusted models					
Rank-rank	−0.03	0.28*	0.02	0.11*	0.12***
	(0.03)	(0.13)	(0.01)	(0.05)	(0.04)
Upward mobility × -1	0.04	−0.04	0.01	0.06	0.04
	(0.05)	(0.13)	(0.01)	(0.05)	(0.04)
Gini	0.04	−0.13	−0.00	−0.05	−0.08*
	(0.04)	(0.12)	(0.01)	(0.05)	(0.03)
Individuals	8810	8810	8810	8810	8810

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 12 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (days smoking last month). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.10: Estimates of average categorical (quintile) exposure on health indicators, NLSY97

	Health status	BMI	Depression	Smoking	Days smoking last month
Unadjusted models					
Rank-rank	−0.02	0.25***	0.00	0.08***	0.10***
	(0.02)	(0.06)	(0.01)	(0.02)	(0.02)
Upward mobility × -1	0.03	−0.23***	0.00	0.02	0.02
	(0.02)	(0.06)	(0.01)	(0.03)	(0.02)
Gini	−0.01	0.11	−0.01	−0.10***	−0.10***
	(0.02)	(0.07)	(0.00)	(0.02)	(0.02)
Adjusted models					
Rank-rank	−0.01	0.21**	0.01	0.09***	0.08***
	(0.02)	(0.08)	(0.01)	(0.03)	(0.02)
Upward mobility × -1	−0.01	−0.07	−0.01	−0.02	−0.01
	(0.03)	(0.10)	(0.01)	(0.03)	(0.03)
Gini	0.03	−0.07	−0.00	−0.07*	−0.07***
	(0.03)	(0.10)	(0.01)	(0.03)	(0.02)
Individuals	8810	8810	8810	8810	8810

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 12 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (days smoking last month). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.11: NLSY97 Stabilized treatment weights

			Percentiles			
Weight	Mean	SD	1st	25th	75th	99th
Continuous exposure						
Rank-rank	1.01	0.91	0.35	0.71	1.05	3.57
Upward mobility	0.99	0.63	0.34	0.74	1.05	3.30
Gini	1.05	1.78	0.29	0.70	1.08	4.29
Categorical (quintile) exposure						
Rank-rank	1.00	0.56	0.39	0.68	1.09	3.51
Upward mobility	1.00	0.55	0.47	0.69	1.08	3.10
Gini	1.00	0.55	0.47	0.67	1.07	3.02

Analyses based on exposure from 12 to 20 years old. Statistics based on 20 multiple imputed datasets.

PSID

Table 4.12: Estimates of average continuous exposure on health indicators, PSID

	Health status	BMI	Depression	Smoking	Cigarettes smoked
Unadjusted models					
Rank-rank	-0.14^{*}	0.58	0.02	0.13	0.10
	(0.07)	(0.30)	(0.03)	(0.11)	(0.11)
Upward mobility × -1	-0.14^{*}	0.47	0.02	−0.05	−0.03
	(0.07)	(0.28)	(0.03)	(0.14)	(0.12)
Gini	−0.01	0.18	−0.01	−0.12	-0.13^{*}
	(0.08)	(0.22)	(0.02)	(0.08)	(0.07)
Adjusted models					
Rank-rank	−0.06	0.34	0.03	0.10	0.04
	(0.08)	(0.28)	(0.03)	(0.12)	(0.11)
Upward mobility × -1	−0.07	0.58	0.07	−0.05	−0.09
	(0.10)	(0.32)	(0.04)	(0.14)	(0.14)
Gini	−0.02	0.27	−0.02	−0.15	-0.22^{*}
	(0.12)	(0.29)	(0.03)	(0.13)	(0.09)
Individuals	2273	2273	2273	2273	2273

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 1 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (cigarettes smoked). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.13: Estimates of average categorical (quintile) exposure on health indicators, PSID

	Health status	BMI	Depression	Smoking	Cigarettes smoked
Unadjusted models					
Rank-rank	−0.07	0.37^{*}	0.01	0.06	0.06
	(0.05)	(0.18)	(0.02)	(0.07)	(0.07)
Upward mobility × -1	0.09	−0.33	−0.01	−0.01	−0.03
	(0.05)	(0.20)	(0.02)	(0.08)	(0.07)
Gini	0.01	0.08	−0.00	−0.08	−0.09
	(0.05)	(0.15)	(0.01)	(0.06)	(0.05)
Adjusted models					
Rank-rank	−0.04	0.16	0.03	0.07	0.05
	(0.05)	(0.17)	(0.02)	(0.07)	(0.07)
Upward mobility × -1	0.07	−0.33	−0.02	−0.15	−0.09
	(0.06)	(0.22)	(0.02)	(0.08)	(0.07)
Gini	0.07	0.06	−0.00	−0.03	−0.07
	(0.06)	(0.18)	(0.02)	(0.07)	(0.06)
Individuals	2273	2273	2273	2273	2273

Each coefficient represents a model. Coefficients and standard errors are combined estimates from 20 multiple imputed datasets. Analyses based on exposure from 1 to 20 years old. We estimate different models depending on the outcome: Ordinal regression (self-reported health), General linear model (BMI, depression), Logistic regression (smoking), Quasi-Poisson regression (cigarettes smoked). $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4.14: PSID Stabilized treatment weights

			Percentiles			
Weight	Mean	SD	1st	25th	75th	99th
Continuous exposure						
Rank-rank	1.02	0.81	0.36	0.69	1.07	4.39
Upward mobility	1.04	2.66	0.30	0.57	1.04	4.50
Gini	1.08	1.96	0.24	0.66	1.06	5.52
Categorical (quintile) exposure						
Rank-rank	1.00	0.61	0.41	0.69	1.07	3.40
Upward mobility	0.98	0.70	0.46	0.59	0.98	3.30
Gini	1.01	0.65	0.44	0.64	1.05	3.73

Analyses based on exposure from 1 to 20 years old. Statisticis based on 20 multiple imputed datasets.

A Generative Model for Income Mobility and Mortality

Two trends have characterized the distribution of socioeconomic resources and health conditions in the U.S. over recent decades. First, both socioeconomic inequality and upward mobility rates have been declining in America since the 1970s (Chetty et al., 2017; Gould, 2019). At same time, the life expectancy gap by socioeconomic status has been growing in recent years in the U.S., representing a fundamental challenge for health policy. Recent work by Chetty et al. (2016), for instance, shows that between 2001 and 2014, life expectancy has increased 2.34 years for men in the top 5% of the income distribution and 2.91 years for women, while only 0.32 years for men in the bottom 5% of the distribution and 0.04 years for women.

While inequality and upward mobility trends have clear economic consequences, they may also foster the divergence of health behaviors and outcomes. This hypothesis has gained ground not only because previous research shows that neither access to medical care nor socioeconomic factors fully explain observed geographic or income disparities in longevity, but because socioeconomic mobility rates, similarly to the life expectancy gap, vary considerably by geography (Chetty et al., 2014). Thus, some scholars have recently suggested that contextual income mobility — defined as individuals' ability to exceed their parents' income at the place of residence – may play an essential role in explaining health disparities in the U.S. (Daza & Palloni, 2018; Venkataramani et al., 2016; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020; Venkataramani, O'Brien, et al., 2020). Low-income mobility, for instance, may harm health by raising despair and diminishing the motivation to engage in healthy behaviors (Case & Deaton, 2020; Schilbach et al., 2016). These effects would be distinct to the consequences of income inequality for health, as individuals living in areas characterized by similar high degrees of income inequality may experience different probabilities of income mobility – and therefore may have different impacts on health outcomes.

Previous research provides some evidence on the connection between place's income mobility and health. Most of this research, however, uses either aggregate data (Daza & Palloni, 2018; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020) or individual cross-sectional surveys (Venkataramani et al., 2016). Recently, **daza2020a** found only partial support to the connection between exposure to county's income mobility during early childhood and adolescent and smoking during their thirties and forties using longitudinal datasets such as the Panel Study of Income Dynamics (PSID) and National Longitudinal Surveys of Youth (NLSY 1997). As previous research suggests (**daza2020a**; Daza & Palloni, 2018), the link between place's income mobility and health is not simple. The processes connecting health, income mobility and inequality might

involve individual, contextual, spatial, reciprocal effects, cumulative processes and feedback loops. Unfortunately, little attention has been paid to the formalization of the connection between the flexibility of the economic system, individual behavior, health and mortality, in order to assess the population consequences of the individual estimates provided by empirical studies, the challenges or problems associated with using aggregate data to estimate the effect of place's income mobility on health (e.g., ecological fallacy), and the data needed to estimate the predictions discussed in the literature.

In this paper, we take a step back from statistical models, and attempt to formalize the association between the exposure to given stratification system and health, and assess the nature of the patterns we could find under different stratification scenarios. Specifically, we develop an agent-based model (ABM), *Mortality and Income Mobility Agent-Based Model* (MIA), that generates intergenerational data to study the connection between income mobility, inequality, residential segregation, smoking and mortality. The purpose of the model is, thus, to formalize initial ideas about how place's income mobility might affect health outcomes, and under which conditions we could recover estimates of those effects.

The paper is organized as follows. First, we first briefly review theoretical mechanisms linking place's income mobility, adult health, and mortality. Second, we outline our research questions and justify the use of agent-based modeling to examine them. Third, we describe the implementation of each of the components of our model, and the experimental design we to examine our research questions. Finally, we present and discuss the implications of our results.

5.1 Potential mechanisms

In this section, we briefly discuss some of the potential causal mechanisms that might generate an association between place's income mobility and health. We particularly focus on 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 follow the interpretation of Chetty's work that suggests economic opportunity is a characteristic of places (Chetty et al., 2017). We add to that interpretation by proposing that prospects of income mobiltiy would 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 or individual mobility) – a problem studied in a large and distinguished body of research (Blane et al., 1999; Chandola et al., 2003; Fox et al., 1982; Illsley, 1955) – or 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 reasonable to expect that individuals' experiences of occupation or SES mobility would also be influenced by the prevailing aggregate regime of income mobility. These experiences may be just one of many other pathways through which aggregate income mobility and individual mortality are related.[1]

[1] At the individual level, the main effect of income mobility on health would operate through socioeconomic status

An association between places' income mobility and mortality could exist if communities with higher income mobility 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.

Why this might be the case? We describe potential pathways that might produce a link between income mobility and mortality. First, 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 might foster 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 (Schilbach et al., 2016). This is also in line with the hypothesis of *deaths of despair* proposed by Case and Deaton (2020). These authors reported the fastest-rising death rates of causes such as suicides, drug overdoses, and alcoholic liver disease in the U.S., especially among those without a bachelor's degree. These self-inflicted deaths are prevalent among those who face economic, social, or psychological adversities, hopelessness, and lack of well-being.

We can extend these mechanisms to the consequences of early conditions on health. 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 (Mackenbach, 2012; Meara et al., 2008). In addition, early conditions and upbringing of individuals matter greatly for adult health and mortality disparities (Case et al., 2002; 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 (Marmot, 2004; Sapolsky, 2005) may be rooted in antecedent health conditions sculpted early in life (Case & Paxson, 2011). 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 (Cunha & Heckman, 2009; Heckman, 2007; Knudsen et al., 2006; Shonkoff et al., 2009). Some of these traits involve the development of outlooks and attitudes that influence in-

and educational attainment. Although, it is also believed that mobility itself could affect health through the lack of or lots of stress when there is downward or upward mobility.

vestments 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, 1972, 2000).

We know from empirical research that negative affect, chronic stress, subordination, bleak future outlooks associated with poverty lead to increases in time discounting (Haushofer & Fehr, 2014; Schilbach et al., 2016). 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 (Eigsti et al., 2006; Schlam et al., 2013). 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.

Finally, an association between aggregate income mobility and individual health and mortality may be the outcome of a composition effect, namely, that places with higher income mobility contain a population composition biased toward individuals who are both, more likely to experience mobility and to embrace health protective behavior. In this case, the association between the aggregate property of the stratification system and individual experiences of health and mortality would reflects the influence of individual residential mobility patterns (and associated selection processes). This mechanism could be also linked to the *neo-material* theory by Lynch et al. (2004), which suggests that the aggregate relation between income inequality and health is not necessary but contingent. In other words, communities with high income mobility would host a set of (unobserved) social and economic traits that might eventually promote good health and reduce mortality risks. It would not be income mobility itself whath would generate a reduction in mortality, but a set of associated community characteristics related to good health and reduced mortality risks.

5.2 Modeling strategy

Health mechanisms, like those described above, are difficult to identify using statistical models as they tend to produce the same summaries from data generated by different processes.[2] Simulation

[2]This issue is commonly named the *inverse problem* (McElreath, 2020). 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 can generate similar mean and variance summaries. In contrast to statistical

and *generative* models, in contrast, have the potential to help us learn from complex systems by offering simplified representations of the mechanisms that generate and preserve health inequalities (Railsback & Grimm, 2011; Smaldino, 2017; Speybroeck et al., 2013; Wolfson et al., 2017).

In this paper, we take a step back from statistical models and propose a computer simulation that implements the mechanisms discussed in the literature in order to assess the consequences of the socioeconomic mobility regime for health, and examine the behavior of the mechanisms proposed by exploring *what-if* questions and scenarios. Specifically, we create a *low-dimensional realism* model where micro-level behaviors are assumed or known, and simulation is used to explore how a system behaves (Edmonds et al., 2019).[3] Thus, the scope of the model is mainly theoretical and exploratory.

In terms of design, we focus on three critical aspects on how empirical data are generated. First, we take into account *space* by allowing agent to reside in a county or neighborhood. Thus, we can explore the hypothesis that place's income mobility (not intra or inter-generational mobility) might impact health and mortality. Agents would interact directly through the relationship with their parents and kids, but also indirectly by sharing characteristics of their county of residence. Second, individual preferences on place would not be random, but endogenously defined allowing variability in the exposure individuals have to their place's characteristics (i.e., residential segregation). The endogenous definition of place's preferences would impact contextual characteristics agents are exposed, and the economic rigidity or flexibility of the stratification system. Finally, and unlike what can be realistically done with empirical data, we can track with precision each agent trajectory and their exposure to a given income mobility context.

These aspects make attractive the use of an agent-base model as gives us enough flexibility to implement interaction, space, and individual measurement in a relatively simple way. While agent-based modeling cannot by itself solve the issues of estimation and identification using empirical data, it offers the opportunity to rigorously implement theoretical and empirical insights to experiment with a system and identify the limitations of previous studies.

The purpose of our model is to formalize a set of ideas about the implications of income mobility on health, and consequently, it represents a first step in developing a further theoretical exposition of the link between aggregate income mobility and health. In particular, we first use our model as a micro-simulation tool to translate empirically estimated effects of income mobility on smoking, into absolute differences in life expectancy. We choose smoking because previous empirical research using longitudinal data shows the most systematic effect of income mobility early exposure on health outcomes and behavior is smoking (**daza2020a**). In addition, smoking has well-known consequences for mortality risk and those can be easily incorporated to our model. Due to the low-dimensional realism of MIA, though, our micro-simulation would only provide an approximation to the aggregate consequences of income mobility for longevity under different scenarios.

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models, generative models explicitly define causal connections and mechanisms when simulating a system or behavior. This process forces us to express our ideas and theories in formal and clear way.

[3]This strategy is what Wilensky and Rand (2015) refer to as *exploratory modeling*.

Second, we examine under which conditions or scenarios key parameters of the link between place's income mobility and mortality can be retrieved at the aggregate and individual level. For instance, we can identify conditions in which an ecological fallacy might occur, so that the aggregate association commonly observed in empirical studies, would not be due to individual effects of income mobility on health, but to other processes such as residential segregation or income composition of neighborhoods.

Finally, we explore the interaction between income mobility, segregation, and mortality by exploring under which conditions the consequences of place's income mobility would be the highest or lowest. To be completed when I have a clearer idea on what I am doing.

5.3 MIA: Mortality and Income Mobility Agent-based Model

We design the *Mortality and Income Mobility Agent-based Model* (MIA) to simulate a simplified data-generating process of the interaction between income mobility and mortality, based on the mechanisms proposed in the literature. MIA consists of four essential parts: demography dynamics (mortality and fertility), income residential mobility, income generation and mobility, and smoking behavior. We now describe the implementation of each of those components in detail. The results of the verification and testing of these procedures are available in the *Methodological Supplement*.

5.3.1 Demographic processes

Age-specific mortality rates and birth rates in the US define the population dynamics of the model (for more details see Table 5.2). Because the model does not include any information about gender, we divided birth rates by an adjustment factor to make the population close to stationary (zero growth rate).[4] While fertility rates are constant across agents, age-specific mortality rates might be affected by the agent's income and smoking behavior following the expression:

$$ {}_a m_{x_i} =_a m_{x_b} * exp(\beta_{m_k} \text{incomeType}_i + \beta_{m_{smk}} \text{smokingStatus}_i) \tag{5.1} $$

Where ${}_a m_{x_b}$ represents the baseline age-specific mortality rates, β_{m_k} is the natural logarithm of the hazard ratio of income group k (versus the reference category), and $\beta_{m_{smk}}$, the natural logarithm of hazard ratio of smokers (versus non-smokers). While β_{m_k} coefficients intend to reproduce life expectancy gaps observed in previous research (Chetty et al., 2016), $\beta_{m_{smk}}$ comes from models using the U.S. National Health Interview Survey (Jha et al., 2013). When both β_{m_k} and $\beta_{m_{smk}}$ are equal to zero, agents' life expectancy across different generations is about 78.4 years (see section *Demographic dynamics* in the *Methodological Supplement*). As expected, that value is close to the life expectancy of 78.6 reported by Kochanek et al. (2019) for the US in 2017 using the same mortality rates. The average number of off-springs, in turn, is 1.00 (about 37% of agents are childless), and the population size is relatively uniform over time. It is important to note that

[4]The fertility adjustment factor was derived through calibration.

income and smoking are the only direct effects for mortality risks, and in this version of the model current characteristics of the place where agents live do not impact directly mortality. Table 5.1 describes all model parameters with their initialization values.

5.3.2 Residential mobility

Agents live in counties (or neighborhoods). Although the first generation of agents in our simulation (G_0) is located randomly in space, they can move to a different county when over 18 years old. To assess the consequences of reinforcing selection processes on health, we generate income segregation by adapting the Schelling segregation model (Schelling, 2006). As the famous Schelling's model shows, a simple rule of satisfaction can generate residential segregation even when agents are tolerant to live in a neighborhood with people of different *color* (Wilensky & Rand, 2015). Although Schelling's model is dynamic (agents move to different neighborhoods until equilibrium is reached), population in that model is constant (nobody is born or die), only two groups (colors) are allowed, and every agent stops moving when satisfied. In our version, instead, agents decide at rate mob_r whether to move or stay in their county. With probability mob_{rand}, agents move to a random county that has not reached its population limit.[5] With probability $1 - mob_{rand}$, agents assess whether to move or not based a similarity tolerance threshold mob_{thr} (e.g., 20%). When the proportion of people with agent's income group is lower than the tolerance threshold, agents move to a random county from a pool of counties that has not reached its population limit. Otherwise, they decided to stay in the current county. Young agents (less than eighteen years old) are not allowed to make moving decisions, and they just follow their parents' county of residence.

This mechanism generates income segregation as shown in the section *Verification and calibration* in the *Methodological Supplement*. We also adjusted the residential mobility parameters so that to reproduce the levels of income segregation reported by **jargowsky2017a** using the neighborhood sorting index. According to **jargowsky2017a**, the U.S. in 2020 has a NSI of 0.396.

5.3.3 Income generation and mobility

Agents are given an income category k (e.g., low, medium, high) by age eighteen. After the first generation (G_0), income categories are assigned based on two components. The first consists of the intergenerational transmission of resources from parents to children. This process is defined by the probability of inheriting agent's parent income group or p_k. The rest of income groups would have probability $\frac{1-p_k}{2}$, so that we create a vector P that adds up to one. This first intergenerational component allows us, for instance, to define a baseline income mobility regime: no income mobility at all, or 60% chance of inheriting agent's parent income group. If the parent's income group is $k = 1$, P would be $\left[p_k, \quad \frac{1-p_k}{2}, \quad \frac{1-p_k}{2}\right]$.[6] These probabilities are then used to assign agents to an income group. The income final values (currency) are sampled from the the National Health

[5] For instance, more than 20% the expected county population N_c, where N_c is the population at time t divided by the number of counties.

[6] If income group $k = 2$, P would be $[\frac{1-p_k}{2}, p_k, \frac{1-p_k}{2}]$, when $k = 3$, P would be $[\frac{1-p_k}{2}, \frac{1-p_k}{2}, p_k]$.

Interview Survey 2019 (NHIS) distribution of family income by tertiles.[7] Once income is defined, it remains constant throughout the agent's life (i.e., permanent income).

The second component of income definition is contingent on where an agent lived during their early live. Specifically, the probability of being in a given income category is affected by the average exposure to residence county's income composition until age 18. Thus, the average exposure would depend on the residential mobility regime in action (i.e., how parents move across counties), and the income composition of counties where their parents moved.[8] In other words, the vector P is adjusted using the distribution of total years exposed to counties with a predominant income group k. We define a vector C with the fraction of exposure in counties with a dominant income class, where dominant refers to the largest class in a county. A person, thus, would spend y_1 years of their first 18 years in counties where the largest class was $k = 1$, y_2 years in counties where the largest class was $k = 2$, and $18 - y_1 - y_2$ years in counties where the largest class was $k = 3$. The probabilities of belonging to each class p_{y_k} are proportional to the fractions:

$$C = \left[\frac{y_1}{18}, \quad \frac{y_2}{18}, \quad \frac{18 - y_1 - y_2}{18} \right] \tag{5.2}$$

We combine vectors P and C using the function:

$$I_k = P(1 - w) + Cw \tag{5.3}$$

Where I_k is a vector with the probabilities of being in each income group k, and w represents the relative importance of the contextual component in agent's income generation against the individual component. Using the weight w allows us to control the relative importance of both income generation mechanisms, so that when $w = 0$, I_k would be just equal to P, while when $w = 1$, I_k would be equal to C.

In sum, our model simplifies the income generation process by using two factors: the parent's income and the average exposure to counties' resources during early life. County's resources are represented by their income composition. They depict a set of opportunities to which agents are exposed during early life. Conceptually, these resources would affect education attainment and the development of skills useful during the adult work life. However, in our model, income assignment accounts for all those effects. In the same vein, family transmission of skills, cultural capital, or habits are only represented through the association between parent's and child' income. Importantly, both family transmission and early exposure can, in theory, offset each other. Coming from a poor family can be compensated, at least in part, by living in affluent counties during early years, and vice-versa. That compensation process, although, would be unlikely in a context where income segregation is common.

Our segregation implementation by itself does not generate individual income mobility if

[7] The Gini coefficient of the NHIS 2019 income distribution is 0.42.

[8] Agents under eighteen are not allowed to make moving decisions, and they follow their parents' county of residence.

agents do not change their income based on the characteristics of the place where they live. By combining family (P) and county (C) *resources*, we establish a feedback loop between income and county of residence so that income is assumed to depend on the neighborhood, while the neighborhood in adulthood is assumed to depend on individual income.

These income generation mechanisms define the income mobility regime of our model. We track the income mobility regime using aggregate measures of mobility. On the one hand, we use *relative mobility* to measure how the ranking of adults against their peers is (or is not) tied to the ranking of their parents against their peers. That is, how did adults who rank high or low in the income distribution also have parents who ranked high or low? Chetty et al. (2014) uses an OLS model to estimate relative and absolute income mobility, under the assumption of linearity between parent and kid income ranks or percentiles:

$$R_{ic} = \alpha_c + \beta c P_{ic} + \varepsilon_{ic} \tag{5.4}$$

Where R_{ic} is the national income rank among children in a given cohort or generation of child i who grew up in county c. P_{ic}, instead, is the parent's rank in the income distribution of the parents of the corresponding generation of kids. β_c represents the relationship between national parent and kid income ranks. However, it is not equivalent to the rank-rank correlation within a county - or intensity of the linear relationship between ranks - because ranks in a small area would not be necessarily uniform when using national ranks. Computing ranks within counties would create uniform distributions so that β_c would be equal to $Corr(P_{ic}, R_{ic})$. Thus, the interpretation of relative income mobility proposed by Chetty et al. (2014) to small areas (i.e., counties), so-called rank-rank slope, would be the difference between the expected ranks of children born to parents at the top and bottom of the income national distribution: $\bar{r}_{100,c} - \bar{r}_{0,c} = \beta_c$. Unlike the correlation, the size of the coefficient β_c will depend on the standard deviation of the parent and child rankings at the national level:

$$\hat{\beta}_c = r \frac{\sqrt{\sum (R_{ic} - \bar{R}_c)^2}}{\sqrt{\sum (P_{ic} - \bar{P}_c)^2}} \tag{5.5}$$

β_c will be higher than correlation when the standard deviation of the parent's rank P_{ic} is lower than the standard deviation of the kid's rank R_{ic}. In a context of segregation, parent's rank would tend to be homogeneous (lower standard deviation), while kid's rank would tend to vary more that P_{ic} due the income generation probabilities used in our model (vectors P and C defined above). I have to explore the consequence of this way to measure relative income mobility at the aggregate level. As a robustness test, Chetty et al. (2014) estimated relative mobility based on parent and child ranks in the local income distribution instead of national ranks, showing their estimates are highly correlated with their baseline estimates based on national ranks. As can be seen in Figure X in the 5.9, comparing different scenarios using MIA we confirm the relationship observed in equation 5.5. If income mobility is zero, the correlation between the ran-rank correlation (within county) will be only moderately related

On the other hand, Chetty et al. (2014) define upward income mobility as the average rank of children with parents at the 25^{th} percentile in the national distribution:

$$\bar{r}_{25,c} = \alpha_c + 0.25\beta_c \tag{5.6}$$

This is not a true measure of absolute mobility because it is based on ranks. Therefore, it measures displacement of someone in a distribution. However, because the measure of absolute mobility is county-specific, and the county's income would not have a big impact on the national income distribution, the measure provides a reasonable a way to describe patterns of upward mobility. We supplement that measure by computing *absolute mobility* (A) using the fraction of sons who are in a income class higher than their parents.

5.3.4 Smoking

Smoking is the key behavior that determines mortality risk in our model. Smoking, in turn, is the result of both individual income and income mobility. This strategy is based on the Grossman Model (Grossman, 1972), according to which those with higher discount rates invest less in their health, and on the evidence that shows that time discounting is related to health behaviors such as physical activity, substance use, and smoking (Barlow et al., 2016; Story et al., 2014). In the case of smoking, cigarette smoking's health costs come at a delay, while the benefits are immediate. Thus, time discounting seems to be an important mediating factor linking environmental, social, life-course factors, and health. In other words, smoking would be a mediator through which income mobility operates as there is no direct income mobility effect on mortality risk.

In our model, smoking behavior was implemented using a logistic model so that agents at age 30 decide whether or not to smoke. This simple implementation aims to include information from previous empirical research on the independent connection between early exposure to income mobility (childhood and adolescence) and current smoking during early adulthood (thirties and forties). Smoking status at age 30 is defined using the following logistic model:

$$Pr(Y_i) = \frac{e^{\alpha_s + \beta_{s_k} \text{incomeType}_i + \beta_{s_{imob}} \text{incomeMobExposure}_i}}{1 + e^{\alpha_s + \beta_{s_k} \text{incomeType}_i + \beta_{s_{imob}} \text{incomeMobExposure}_i}} \tag{5.7}$$

Where $Pr(Y_i)$ – the probability of being an active smoker by age 30 – is a function of the agent's income group (k), and income mobility exposure. β_{s_k} coefficients were estimated using a subset of the National Health Interview Survey 2019 (NHIS). By considering only respondents between ages 30 and 50, we created income tertiles and defined current smoking as people who reported smoking at least 100 cigarettes during their lifetime and who reported smoking every day or some days at the time of the survey. We then estimated β_{s_k} coefficients to be plugged in Equation 5.7.[9]

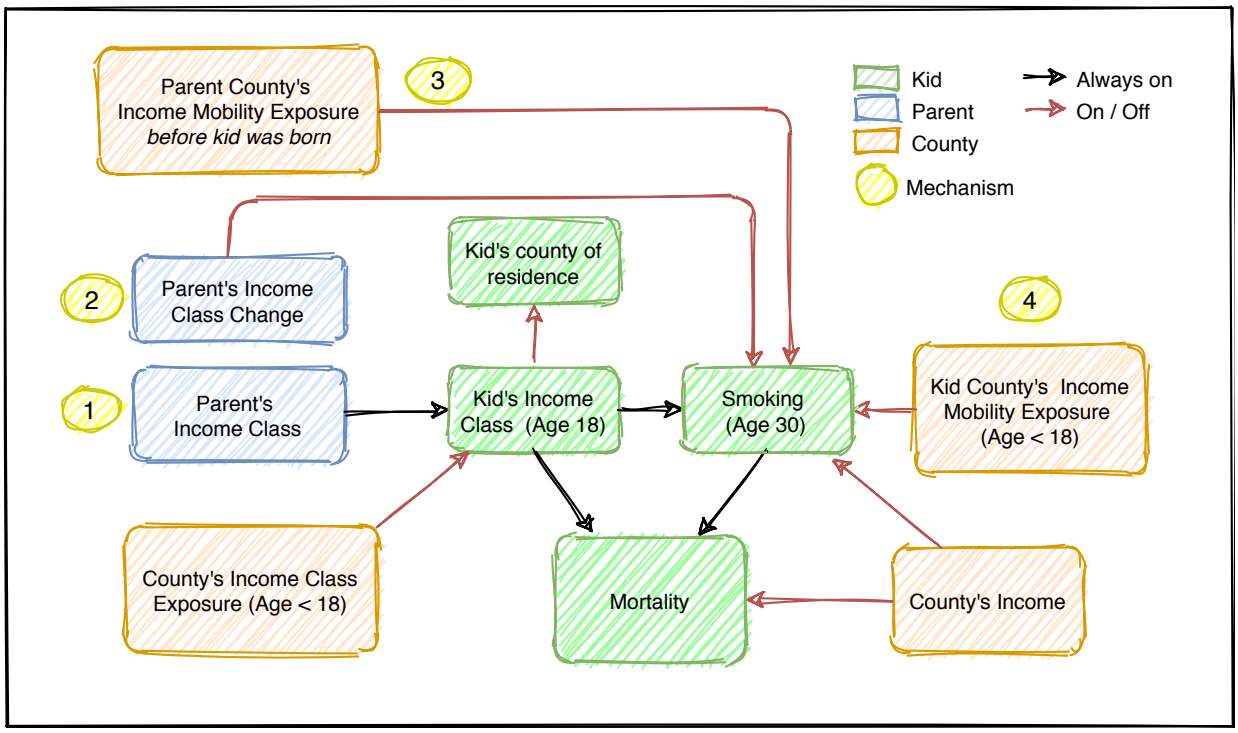
[9]The specific models and code to reproduce this analysis are available in https://github.com/sdaza/dissertation/tree/master/ch04. Additional information on the NHIS 2009 is available in https://www.cdc.gov/nchs/nhis/index.htm.

The income mobility exposure coefficient $\beta_{s_{imob}}$, in turn, comes from the effect of early income mobility exposure on current smoking reported by **daza2020a** using the National Longitudinal Survey of Youth 97 (NLSY97). **daza2020a**'s estimate comes from a logistic model, so that it can be directly plugged into our function. The final smoking status is generated using the probability (p_{s_i}) estimated from Equation 5.7. Agents become smokers if values from a uniform distribution – `uniform(0, 1)` – are less that p_{s_i}. We assume that no smoking occurs before age 30, and once agents decide to smoke, they do not quit. Although those assumptions are unrealistic, they allow us to directly incorporate information from empirical results into our model.

5.3.5 Agents attributes and relationships

Figure 5.1 display key agents' attributes and relationships. Factors in green variables represent the target agent or kid, in blue parents' variables, and in orange county-level factors. Black arrows are relationships always on, and red arrows can be switched off. Circles represent mechanisms or key effects, and compositional effects are omitted (e.g., how agents contribute to the county average).

Figure 5.1: Agent's attributes and relationships



Before age 18, kids live with their parents in the same county. By age 18, an income class is assigned based on the parent's income class and the county's income composition where they live (i.e., parent's county). The number of income classes could vary, let us say we have three classes. Here we apply the function $I_k = P(1-w) + Cw$, where P is the vector with the probability to have the same income as the parent's income class. For instance, if the parent is in class 1, P would be

$[0.6, 0.2, 0.2]$, meaning that the probability to be in the same class is 0.60, and 0.20 for the rest. This is represented by the arrow between *parent's income class* and *kid's income class*.

Kids are also exposed to the income class composition of the place where they live (parents' county) or *county's income class exposure*. That would be C, or the fraction of exposure in counties with a dominant income class (see above for more details). w would weight both components (e.g., $w = 0.5$ would be an arithmetic average). These are the two key factors that define *individual* income mobility. Once the income class is assigned based on probabilities I_k, we define the final income by sampling from an observed income distribution (IPUMS, US, 2018) for class k (tertiles, quartiles, or quintiles). Each county's rank-rank slope is computed based on the rank of residents' parent's income and resident's income (kid). So, the income mobility measurement at the county level follows kids, not parents. I have to check this because it looks like Chetty is not doing that. Income mobility of a place will get "fixed" when it is measured, regardless of where the kids decide to live. Since age 18, agents can move to different counties - with their kids who are less than 18 years - randomly or based on their income class (i.e., segregation, the arrow from *kid's income class* to *kid's county of residence*). Kid's income class increases / reduces mortality risk and the propensity to smoke by age 30. Smoking affects mortality risks. Mortality and smoking are also affected by county's resources (the arrow from current county's income to mortality and smoking). The impact of individual income mobility would only be given by changes in income (no stress related to income mobility changes). The effect we estimated using the NLSY and PSID is represented by the arrow from *county's income mobility exposure* to *smoking*. Based on our conversation yesterday, I added a connection between *parent's income class change* (parent's income class minus grandparent's income class), so that a positive (parent's jump to a better class) would impact agent's chances to smoke. It's important to note that mortality (life expectancy) is measured with a lag (LE comes from completed cohorts, the average of age of death). But, as the mortality regime is the same over time, it shouldn't be an issue.

5.3.6 Model setup and measurement

When the simulation starts, each agent has a set of states and transitions triggered and updated over time. Figure 5.2 display agent's states and transitions. Most of the events in MIA are stochastic. Duration times (rates) are drawn from exponential distributions, and the selection of categories (e.g., income or definition of smoking status) are randomly defined using uniform distributions. Model time is continuous, meaning there is no fundamental clock *tick*.[10]

The model tracks agents' exposure to county income composition and income mobility by recording the difference between $t + c$ (current time) and t (time of previous record), Δt_r, and agent's county income and income mobility at time t. Exposure is recorded each simulation year

[10]We implement MIA using AnyLogic Personal Learning Edition 8.6.0. Rather than specifying the smallest time increment (a *tick*), AnyLogic uses an event-driven scheduler. For example, an event scheduled for ten model-seconds in the future will always occur before one scheduled for 11 model-seconds, and after one scheduled for nine model-seconds. Multiple events may take place at the same time during simulation. In this case, we set up Anylogic so that the events occur in random order. All relevant model parameters are updated at each event. All the files and code to reproduce our analysis are available at https://github.com/sdaza/dissertation/tree/master/ch04.

and when agents move to a new county. By age 18, for instance, average income mobility exposure is estimated using:

$$\frac{\sum_{i=0}^{18} \text{county's income mobility}_i}{18} \tag{5.8}$$

A similar formula is used for tracking the exposure to income groups. Average exposure stats are then used as input in some of the functions discussed in previous sections. More intensive county's characteristics, such as the Gini coefficient and rank-rank slope (income mobility) are computed every ten years. Life expectancy, in turn, is estimated by averaging the age of death of entire generations (i.e., when everyone from a given generation died).

The initial setup includes 60 counties and 150 agents per county. That generates a total population of around 25,000 alive agents (see section *Demographic dynamics* in the *Methodological Supplement*). When collecting individual data from our model, we selected only three generations (e.g., 30, 35, 40) to avoid too big datasets. Those generations are created after 1,000 simulation years.

5.4 Experimental Design

The experimental design would be something like this:

1. To examine how we can create a relationship between aggregate income mobility and life expectancy (county-level analysis) and mortality (individual-level analysis) without any exogenous effect of income mobility on health. That is, removing mechanism 2 and 3 in Figure 5.1. Mechanism 1 would be, thus, a sort of baseline. In a system like the one we created, when removing mechanisms 2 and 3, the only possible effect of income mobility on health would be through income (individual and county's composition) or an unobserved confounder. For instance, we can add heterogeneity in the residential movement rate (or threshold) so that poorer individuals get stuck in their counties, following the research by **sharkey2013a**. The idea is to create an (artificial) relationship between income mobility and mortality. Places with higher income mobility (lower rank-rank slopes) are those with more income heterogeneity. Of course, once we adjust by income, the effect would disappear. Still, I have to identify a consistent way to create these relationships. If I can do it, the conclusion would be that it is possible to observe a spurious relationship between income mobility and mortality when heterogeneity exists in the residential mobility regime. Heterogeneity would be equivalent to an unobserved confounding variable for the relationship between income mobility and health (as we adjust for that variable, we should be able to recover the real relationship). This might be deduced or solved using a DAG, no need to create a simulation model.

2. To examine mechanism 2, likely to be the most important effect (it's a direct experience of income mobility). See the consequences of that mechanism under different scenarios (no segregation, homogeneous segregation parameters, heterogeneous segregation parameters).

3. Something similar regarding mechanism 3.

4. Combine mechanisms 2 and 3 and explore the magnitude of their aggregate consequences.

5. That would be it.

5.4.1 Ecological fallacy

1. Baseline (no effect of aggregate income mobility)
2. Average life expectancy of complete cohorts using different *pk* and no segregation
3. Same *pk* with segregation
4. Same *pk* with segregation *w* set to a value (i.e., 0.40)

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 the environment during their 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.

We explore the aggregate and individual associations between income mobility and mortality (LE).

5.4.2 Segregation and income mobility

(Sharkey & Torrats-Espinosa, 2016) The variability of income mobility across places suggests a causal effect of places on economic mobility. However, minimal progress has been made in explaining what it is about those places that increases or reduces the chances for residents to move upward in the income distribution. In an initial attempt to shed light on the mechanisms for upward mobility, Chetty et al., 2014 examine several characteristics of counties and commuting zones. The authors find that both commuting zones with the highest levels of absolute upward mobility and counties with the largest positive causal effects on earnings in adulthood have, on average, lower rates of residential segregation by income and race, lower levels of income inequality, better schools, lower rates of violent crime, and a larger share of two-parent households. However, as the authors acknowledge, these associations are a first step and should not be thought of as causal

Table XX shows all relevant combinations of the X MIA scenarios we explored to answer our research questions. Resulting in X experimental scenarios.

5.5 Results

5.6 Conclusion and discussion

5.7 Tables

Table 5.1: MIA Parameters

Parameter	Anylogic Name	Symbol	Baseline value	Description
Population dynamics				
Mortality	`f_baselineMortalityRate`	m_x	See Table 5.2	Age-specific mortality rates per year.
Income coefficient	`p_mortalityCoeffIncType`	β_{m_k}	`{0.3, 0.0, -0.3}`	Coefficients of income group k on mortality risk.
Smoking coefficient	`p_mortalityCoeffSmoking`	$\beta_{m_{smk}}$	1.099	Coefficient of the effect of smoking on the mortality risk for all causes of death (Jha et al., 2013, Table 1, pg. 346). The coefficient comes from models adjusting by age, educational level, alcohol consumption, and body-mass index.
Fertility	`f_baselineFertilityRate`	f_x	See Table 5.2	Age-specific fertility rates per year.
Residential mobility				
Moving decision rate	`p_movingDecisionRate`	mob_r	0.30	Average number decisions to move per year when agents are eighteen years old or older. Younger agents (age < 18) would move only if parents do.
Move randomly	`p_randomMobilityProb`	mob_{rand}	0.10	Probability agents move to a random county that has not reached its population limit.
Moving threshold	`p_movingThreshold`	mob_{thr}	0.33	The lowest proportion of agents with the same agent's income category k before moving to a different county.
Population limit ratio	`p_populationMaxRatio`	pop_{limit}	1.20	Ratio at which counties can grow with respect to the expected population. To compute the maximum population of a county at time t, the population at time t is divided by the number of counties and multiply by 1.20.
Income				
Income group	`p_incomeType`	k	1-3	Agents' income category or tertile (e.g., low, medium, high).
Income	`p_income`	$income_k$	0 – 220,000	Agents' income. It is defined using the observed income distribution from the the National Health Interview Survey 2019 (NHIS) by tertile.
Parent income group	`p_parentIncomeType`	$parent_k$	1-3	Parent's income category or tertile.
Parent's income	`p_parentIncome`	$pincome_k$	0 – 220,000	Parent's agent income.

Continued on next page

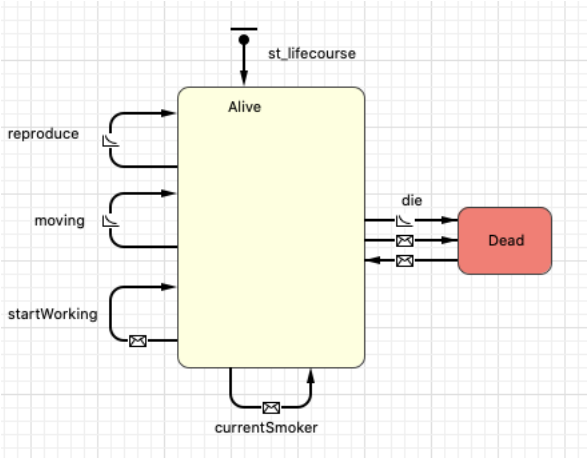
Continued from previous page

Parameter	Anylogic Name	Symbol	Baseline value	Description
Probability inheriting agent's parent income class	`p_probSameParentIncType`	p_k	$\frac{1}{3}$	Probability of inheriting agent's parent income group $parent_k$.
Relative important of contextual income group exposure	`p_weightVectorCountyIncExp`	w	0.40	Weight to combine vectors P and C (see main text for details). When $w = 0.5$, the estimate is equivalent to the arithmetic mean.
Smoking				
Income mobility coefficient	`p_smokingCoeffMobExp`	$\beta_{s_{imob}}$	0.5	Defined by dividing the z-score coefficient reported in **daza2020a** by the standard deviation of relative income mobility (rank-rank slope) across counties.
Income type coefficients	`p_smokingCoeffIncType`	β_{s_k}	{`0.00, -0.64, -1.54`}	Coefficients of income group on smoking. These coefficients where estimated using the National Health Interview Survey 2019 (NHIS). The intercept of the model is -1.043.
Other				
Population per county	`p_peoplePerCounty`	pop	100	Number of agents per county
Number of counties	`p_numberCounties`	$counties$	60	Total number of counties.
Last complete generation	`p_lastGeneration`	G	30, 35, 40	Last generation being tracked completely before stopping the simulation.
Measurement of stratification indicators	`p_recurrentTimeStratData`	T_m	10 years	Time between stratification measures (Gini coefficient and rank-rank correlation).

For distributions in the value column, these are re-sampled for each agent.

5.8 Figures

Figure 5.2: Person State Chart



Methodological Supplement

The code of our model, analysis, and plots is available at https://github.com/sdaza/dissertation/tree/master/ch04.

Verification and calibration

We verified and tested all the key processes of MIA to ensure the data generated by the model are not artifacts or due to coding error. We also checked MIA produced data similar to the pattern observed in empirical studies.

For verification, MIA was setup using: (a) 60 counties with an initial population of 150 agents, (b) three income groups or tertiles (e.g., low, medium, and high income), (d) a population limit per county of 20% the expected population at time t, (e) generation 40 as the last complete generation before finishing the simulation, (f) decision moving rate $mob_r = 0.30$, and (g) 30 replicates per scenario.

Demographic dynamics

To examine MIA's demographic behavior, we collected individual information for generations 30, 35, and 40, over 30 replicates. Figure 5.3 summarizes the key demographic processes implemented in MIA using the baseline mortality and fertility rates (Table 5.2). We use parameters in order to create a stationary population (no systematic population growth or shrinking, (Preston et al., 2000)). As expected, the average number of off-springs per agent is practically 1. The distribution of age of death follows the shape expected given the US's mortality rates with an upper cut-off at 110 years (all agents must die by age 110). Simulated life expectancy for complete cohorts concentrates around 78.3 years. This value is close to 78.6, the life expectancy estimated by

Kochanek et al., 2019 for the US in 2017. The dynamic of the population is close to stationary as sizes are relatively stable and uniform over the simulation, although uncertainty increases over time.

Residential mobility and segregation

The segregation mechanism is an adaptation of Schelling's segregation model (Schelling, 2006). Agents live in counties and at rate mob_r decide whether to move or stay in the current county. With a probability mob_{rand}, agents decided to move to a random county or based their decision on the proportion of people with the same income group living in the county (e.g., low, medium, high) and a similarity tolerance threshold mob_{thr} (e.g., 20%). Suppose the proportion of people with income group k is lower than the tolerance threshold mob_{thr}, agents would move to another random county, excluding those that have reached their population limit so that to avoid extreme concentration of agents within counties.

We use the neighborhood sorting index or NSI to measure income segregation (Jargowsky & Kim, 2005). The NSI compares the income variation across all neighborhoods (or counties) in a metro area with the income variation across all households in that metro area. If agents are segregated across counties by income, the income variation across counties will be similar to the income variation across agents, and the NSI will equal almost 1. If all counties are perfectly economically integrated (i.e., each county is a microcosm of the entire population), the NSI will be almost 0.

Thus, the NSI is a measure of the neighborhood's heterogeneity, normalized by income variance. It measures segregation by showing how much aggregating data lose information about variation in individual income. However, it fails to capture larger-scale features of neighborhoods' spatial arrangement. The NSI is not affected if all high-income neighborhoods are clustered in one part of the metropolitan area or scattered randomly around the map. For the proposes of our model,

that limitation is not problematic.

To examine the changes in the levels of income segregation, we used a decision to move rate (mob_r) of 0.30 per year.

If movement is random, that rate generates on average about 5 moves per agent. Once the segregation mechanism is in action, the number of moves is reduced considerably as agents do not always have incentives to change of county.

We explored different scenarios: all moves are random, only 10 % of moves are random with tolerance thresholds 0.21, 0.23, 0.28, 0.32, and 0.35. Figure 5.4 shows the dynamic of the NSI after 40 generations simulated over different scenarios (around 1500 years per replicate). The plots reveal that the NSI changes dramatically due to relatively small changes in the moving threshold: from an average of 0.06 when movement is completely random, to 0.51 when the moving threshold is 0.35. These differences are related to how the segregation model was implemented, the number of income groups and counties.

The standard deviation of the NSI increases as the tolerance threshold mob_{thr} goes up. Still the trajectory of the NSI is relatively stable over time, where the proportion of the standard deviation over the average NSI across replicates and time, goes from 12% ($mob_{thr} = 0.35$) to 30% ($mob_{thr} = 0.21$). The average number of moves over agent's life also changes depending on the mob_{thr} used. As expected, the highest number of moves is observed when movement is completely random (23.3 moves on average), while the fewest moves (2.6) when mob_{thr} is the lowest (0.21). The average of moves during childhood (before eighteen years old) goes from 0.8 to 1.7 when movement is not completely random.

According to **jargowsky2017a**, the economic segregation in the US by 2010 was 0.396. In order to reproduce those levels of income segregation, we use a tolerance threshold mob_{thr} that generate NSI levels close to 0.39.

Income mobility and segregation

Income generation and mobility mechanisms consists of two components: (1) a baseline intergenerational relation between between parent's income and children's income, and (2) the impact of exposure to county's income. We first verify the implementation of the individual intergenerational mobility mechanism, and how it interacts with income segregation at the aggregate level. Then, we check the implementation of the contextual effect of county resources on income generation and mobility. To calibrate, we compare the distributions generated by MIA with Chetty's mobility data at the county level in the US (Chetty et al., 2014).

I have to show the following:

Income distribution individual and county

Trajectory of income mobility, income, inequality over time for:

for same prob: 0.33, 0.40, 0.50 no income segregation

for same prob: 0.33, 0.40, 0.50 income segregation

Distribution income, income mobility, inequality by county with different scenarios.

Table X shows..

Mortality and smoking by income

Here I have to show differences in life expectancy by income so that to emulate income gaps observed in empirical data.

The same with smoking differences. Smoker should have a much higher mortality risks, and those in lower income groups should have a higher chance to be smokers. The intercept on smoking

should be a higher one.

Then I can estimate the expected years lost due to smoking by income.

Tables

Table 5.2: Mortality and fertility rates

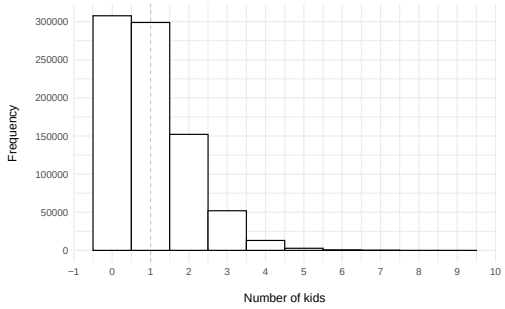
Age group	Mortality rate[1]	Fertility rate[2]
0	567.0	0
1	24.3	0
5	11.6	0
10	15.5	0.2
15	51.5	17.4
20	95.6	68.0
25	121.0	95.3
30	145.4	99.7
35	173.8	52.6
40	218.4	11.8
45	313.2	0.9
50	488.0	0
55	736.5	0
60	1050.2	0
65	1473.5	0
70	2206.9	0
75	3517.8	0
80	5871.7	0
85	13573.6	0

[1] Rates are per 100,000 population in the US (Kochanek et al., 2019, Table 2, pg. 24).
[2] Rates are births per 1,000 women in the US (Martin et al., 2019, Table 2, pg. 13).

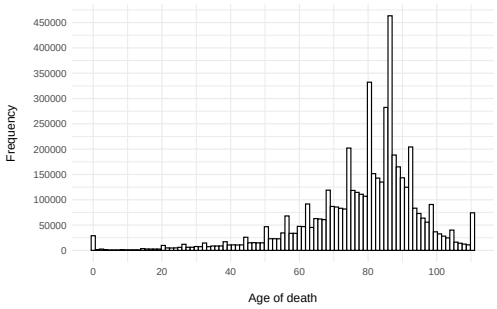
Figures

Figure 5.3: Population Dynamics (30 replicates)

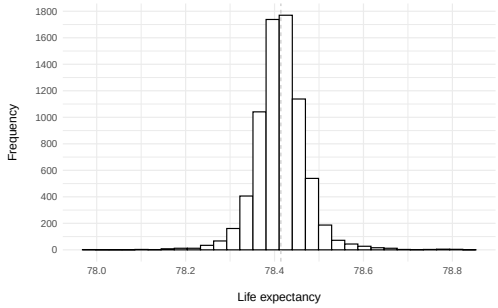
(a) Number of kids



(b) Age of death



(c) Life expectancy



(d) Population size

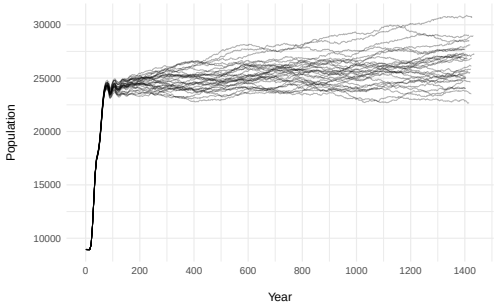
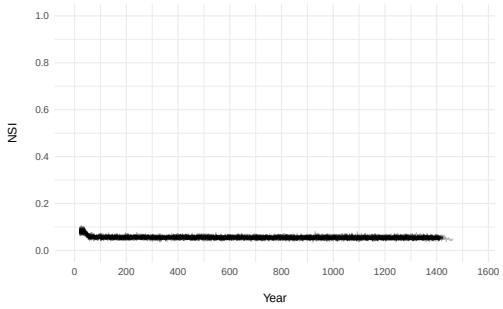
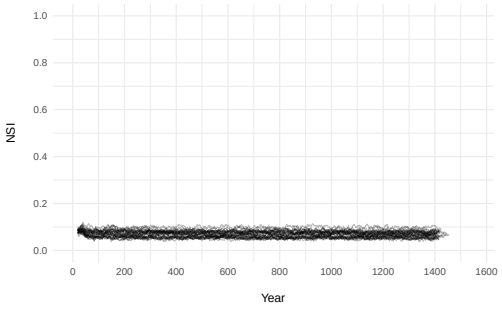


Figure 5.4: Neighborhood sorting index (NSI) by year (30 replicates)

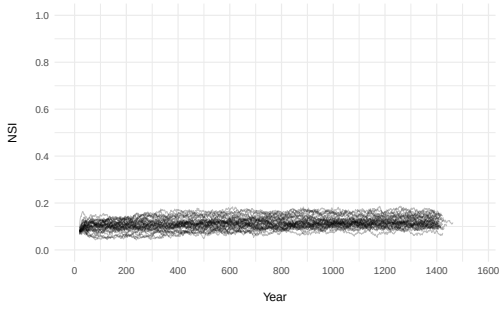
(a) Random



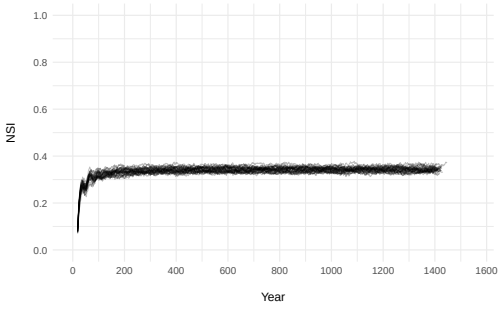
(b) Threshold = 0.21



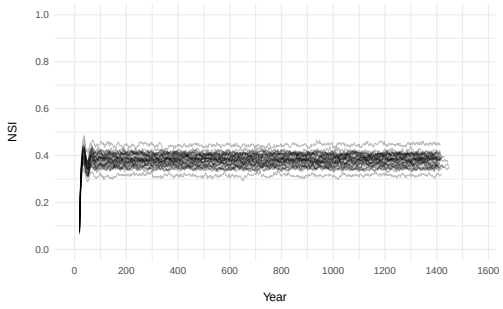
(c) Threshold = 0.23



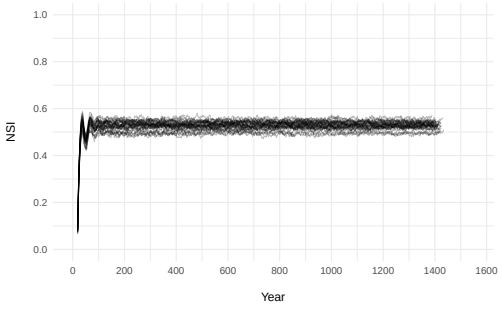
(d) Threshold = 0.28



(e) Threshold = 0.32



(f) Threshold = 0.35



Conclusion

Here I should write the conclusion. Grossman (2000)

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