Income Mobility, Mortality and Health in the US

by

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It remains only to say, as usual, that any remaining errors are chargeable to me - with the exception of errors in this sentence, obviously!

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Introduction

Socioeconomic inequality in health and mortality has been a persistent regularity across societies. Even those with high economic prosperity and human development show systematic disparities in mortality and morbidity. These inequalities are not only persistent. Evidence shows health inequalities have also been widening in high-income countries in recent decades (Chetty et al., 2016; Mackenbach, 2012), especially in countries like the US.

Chetty et al. (2016) shows life expectancy between 2001 and 2014 has increased 2.34 years for men and 2.91 years for women in the top 5% of the income, only 0.32 years for men and 0.04 years for women in the bottom 5% of the distribution. This increase in mortality differences between better-off and disadvantaged people represents a fundamental challenge for health policy. Many of the factors usually attributed to health disparities – income and health care access – do not seem enough to explain health disparities across space and racial-ethnic groups.

In the search for new factors that can explain, at least in part, health disparities, a small group of scholars has been proposing the connection between income mobility (i.e., how flexible or rigid is the stratification system of society) and health as one of the potential determinants of health inequalities. There is some empirical evidence to support these claims. Using county-level and individual cross-sectional data, scholars have shown that higher mobility is systematically associated with lower mortality risks and better health (Daza & Palloni, 2018; Venkataramani et al., 2016; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020). That relationship seems more robust and more consistent than the relationship between income inequality and health (a topic widely studied), although considerably smaller than the impact of income. 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 this small literature and examines the robustness of the relationship between income mobility and health using empirical data and formal modeling. Using different data sources and modeling approaches, I look at the magnitude and variability of this association in the US 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 three strategies. First, I analyze aggregate data to assess the magnitude, robustness, and variability of the *association* of income mobility with mortality. Second, I extend those analyses using individual and longitudinal data to define clearly exposure to an income mobility regime and examine whether some of the potential pathways and mechanisms proposed in the literature are *supported* by the data. Finally, building on this evidence, I create an agent-based model to assess the conditions and plausibility of the potential mechanisms involved in the association between income mobility and health (Billari et al., 2006; Epstein, 2006; Manzo, 2014; Miller & Page, 2007; Page, 2015). This forces me to represent precisely (code) a set of mechanisms likely to bring about the observed patterns. These *virtual* representations, thus, help me explore the implications of the theory and ask *what-if* questions, in addition to providing a general framework to assess previous research and help design new studies. The goal is to go beyond statistical models (i.e., the detection of average differences that may reflect the aggregate statistical signature of *unspecified* underlying mechanisms) and define plausible (precise) mechanisms, and testable hypotheses.

This dissertation is organized into four chapters. First, I formulate and discuss the theoretical framework to explore the connection between income mobility and health. Then, I empirically examine the link between the flexibility of the stratification system, health, and mortality (Chapter 3 and 4). First, I use aggregate data to explore the association between county income mobility and

mortality in the US. Then, I employ longitudinal data to examine the association between exposure to county's income mobility, health indicators, and behaviors related to health (.e.g, smoking). In the last chapter, I propose an agent-based model to formalize the theory behind the link between income mobility and health, explore the population-level consequences of empirical and individual estimates, and assess how hard it is to retrieve these *effects* using data such as those used in previous studies. Finally, I discuss my findings and briefly outline future research ideas and suggestions.

The problem: health disparities and stratification

Theory without data is myth: data without theory is madness.

Phil Zuckerman

Socioeconomic inequality in health and mortality is one of the persistent regularities observed across societies since at least the turn of the 20th. 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) show the gap in life expectancy between the 1% richest and poorest 1% of individuals in the U.S. is about 14 years. These disparities persist across multiple measures of individual social and economic status (e.g., education, income, wealth, occupation) and seem to be widening in high-income countries (Mackenbach, 2012).

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), for instance, is that inequality in health is caused ultimately by social stratification processes, not the exposure to proximal risk factors such as smoking or a bad diet. In other words, 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 would be contingent, changing and continuing to exist, reproducing the association between socioeconomic status and health over time. To explain health disparities, thus, it would be necessary to identify some of the features of the stratification system that might influence health status.

According to Grusky (2014), 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 others. Correspondingly, health inequalities might be understood as a function of (1) social mobility and differences of people across social strata, (2) resource distribution or differences in access to material and non-material resources across economic groups, and (3) the value of those resources to avoid health problems (Mackenbach, 2012). All these processes and mechanisms might vary over time and across 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. Leigh et al. (2009) also 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 (Palloni et al., 2009). The remaining two stratification mechanisms pointed out by Mackenbach (2012) are related to the effect of allocation of resources on health, and its interaction with characteristics of the prevailing epidemiological regime.

The mechanisms that produce health disparities, however, are not limited to sorting, differential resource abundance, and resource productivity heterogeneity. It has also been argued that factors such as the distribution of income in the population (i.e., income inequality), the socioeconomic relative position of individuals, and the rigidity of the stratification system (i.e., income mobil-

ity) might have a *contextual* impact on individual health and mortality risk. This is the type of mechanism I would focus on in this dissertation, namely, income mobility.

To gain conceptual clarity and establish common ground with a rich research tradition, I first discuss briefly the literature linking income inequality and health, and focus mostly on theoretical and conceptual issues rather than methodological problems. Using a similar framework, I then discuss the link between income mobility and health, this dissertation's main topic.

2.1 Income inequality and health

The *income inequality hypothesis* states that a *contextual* factor such as income inequality affects individual's health independently from their resources (e.g., income). Income inequality is, by definition, an ecological variable (i.e., a population trait) that describes the scale of income differences across a population. There is no such thing as individual income inequality. That said, the association between income inequality and health can be generated by different mechanisms or processes. Evans et al. (2004) provide a useful typology that summarizes ways to produce 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, would not be only due to to material factors. *Scarcity* may impose a cognitive *bandwidth tax* that interferes with decision making and long-term planning, increasing the adoption of risky behaviors (Haushofer & Fehr, 2014; 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.

4. Finally, Evans et al. (2004) point out *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 violent crime. Thus, changes in other people's income can modify customs, ideals, politics, and the behavior of those whose own income remains unchanged.[1]

To formalize the relations conjectured by the *income inequality hypothesis*, I use the notation proposed by Wagstaff and van Doorslaer (2000) and express the expected individual relationships 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, where community refers to any geographic or administrative unit available (i.e., census tract, county, state, or region). f_i is a function through which inputs are transformed into health outcomes. This formula represents the independent effects of the terms using an unspecified functional form f_i.[2] We can also express these associations at a given community level c:

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

where c is an index of the aggregation level (e.g., county). Using this aggregate model would make it difficult to distinguish between some of the effects discussed by Evans et al. (2004). However,

[1] 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).

[2] It is possible to define a case where it is not the community's inequality that matter, 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).

it can provide clues on how contextual effects observed at the individual level *behave* at the community and population level. Given the complexity of the individual and contextual effects linking stratification and health, aggregation might not always be linear and straightforward. Health often reflects cumulative exposures over a long time. Thus, health at adult ages can be partly determined by exposure to biological and social factors early in life. The roots of health inequalities may well lie in inequalities experienced back then. Unfortunately, aggregate analyses might also be misleading as trends at multiple analysis levels may or may not be similar. Their causes may or may not be the same (i.e., ecological fallacy Snijders et al. 2011). Pickett and Wilkinson (2015), for instance, note that income inequality effects are more substantial in large areas because, in that context, income inequality serves as a measure of the scale of social stratification. Income inequality in small areas, in turn, is affected by the degree of residential segregation. Higher segregation would increase the inequality between areas and increase homogeneity within them (Chen & Gotway Crawford, 2012).

The main argument behind the *income inequality hypothesis* is that an unequal income distribution would damage the social fabric, reducing both social capital and mutual trust. The resulting stress and lack of public investments would 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, thus, would increase 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). Also, social comparison to higher-ranking friends and neighbors might cause stress and ill-health among more impoverished people than their reference group (Marmot, 2004; Wilkinson, 1992). The so-called *relative deprivation hypothesis*, though, assumes that adverse effects would outweigh the benefits of having more affluent neighbors who may pay for better neighborhood amenities. Another supplementary mechanism 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).

A different approach explains the link between income inequality and health using 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 impact public resources such as schooling, health care, social welfare, and working conditions (Lynch et al., 2004). Gilens (2012), for instance, provides a political interpretation of these processes and notes that income inequality increases the wealthy's political influence. If their policy preferences restrict public goods that provide health benefits (e.g., education, sanitation, policing), it may end up harming the most disadvantaged people's health. In sum, according to the neo-material perspective, once we account for material factors at the community level, the independent connection between inequality and health should disappear.

Can these hypotheses and mechanisms be extended to place's income mobility? If so, what consequences and patterns we should expect?

2.2 Income mobility and health

Unlike income inequality, mobility can be measured at the individual (inter and intragenerational socioeconomic changes over the life course) and aggregate level (earnings elasticity, rank-rank slope).[3] Thus, income mobility is not always an ecological or contextual variable like income inequality.

There is a large body of research about the impact of individual mobility on health and mortality. The work by Sorokin (1959) and the *mobility effects* literature, for example, suggest at least

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

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) would create chronic strain and generate a permanent negative impact on mental health and well-being. The *falling from grace* hypothesis, in turn, predicts that *only* downward intra-generational class mobility is detrimental to health, especially in social and historical contexts that foster high social expectations for economic success (Newman, 1999). Downward mobility often entails an involuntary loss of 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 health consequences, independently of the material conditions. Finally, the *acculturation* hypothesis (Blau, 1956) argues that mobility is neither a cause nor consequence of health, but those 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 adjusting for income or social class. In other words, class differences in stress exposure and access to personal and social resources would be the dominant mechanism affecting health, despite the resources that socially mobile individuals might bring from their previous social position.

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 income inequality, we can think of income mobility as a contextual factor determining health and health disparities. Recent evidence suggests that social mobility has a positive relationship with health and mortality even after adjusting for income at the community and individual level, and income inequality (Daza & Palloni, 2018; Venkataramani et al., 2016; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020). At a given level of inequality, people living in communities with a more flexible stratification regime seem to have lower mortality and better health. Theoretically, it is possible to

think about societies with different levels of inequality and social mobility. 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, what provides empirical grounds to estimate the independent effect of income mobility and inequality (Chetty et al., 2014).

2.2.1 Mechanisms

What mechanisms would explain the link between health and mortality and place's income mobility? 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 level and aggregate income inequality. In that sense, place's income mobility might be just a proxy for a host of community traits that could impact health.

But it also possible that credible prospects for upward income mobility might encourage people to invest more in their health, as higher returns to health investments are expected (Grossman, 1972; Venkataramani et al., 2015). Optimism about the quality of one's future may raise the desire to achieve good health, independently of the possibility of obtaining higher income by doing so (Venkataramani et al., 2015). A pessimistic interpretation of this mechanism, in turn, holds that a rigid stratification system fosters individual hopelessness, weakens aspirations, and, more generally, diminishes the value of adopting 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).[4] This is in line with Case and Deaton (2020)'s findings, who reported fastest-rising death rates of causes such as suicides, drug overdoses, and alcoholic liver disease in the US, especially among those without a bachelor's degree. These self-inflicted deaths have been labeled *deaths of despair* as they affect those who face economic, social, or psychological adversities, hopelessness, and lack of well-being.

Both positive and negative interpretations of this income mobility mechanism 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). In contrast, hopelessness implies that the extent of mobility should only produce benefits for individuals at the lower end of the income hierarchy.

This general interpretation of the consequences of income mobility for health borrows 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 health care prices, wages, and health productivity. For instance, increasing wage rates (shifting the marginal efficiency of investment) would increase returns from healthy days and increase the optimal health stock. However, the final result of an increase in income might be ambiguous because of the trade-off between higher returns of health (healthy days are more valuable) and higher opportunity costs associated with health production (time spent exercising). On the other hand, the level of education 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

[4]Time discounting characterizes how individuals' preference for a reward decreases with the delay to its receipt. 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).

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 positive outlooks and the value of skill acquisition that eventually affects 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. As 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. Thus, it is not extravagant to think that, along with the place's income mobility, the parent's mobility experience itself (whether they experienced mobility and how significant was the shift) before children turn 18 or when they leave the parental home, could be a more direct (and easy to measure) factor that changes the kid's expectations and adoption of healthy behaviors. Similarly, parents' average exposure to regimes of mobility depending on the places they lived in before a child is born might be critical in shaping future expectations and parenting styles that may ultimately affect the kid's adoption of health-impairing behaviors. This mechanism also opens the possibility that health would *impact* income mobility so that the deterioration of health in a community might stiffen the stratification regime due to health selection processes.

To simply formalize some of these ideas, I can write an expression using the nomenclature 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 c, y_i individual income, and I_c income inequality. As in 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. First, Equation 2.3 does not include a term for *intragenerational* mobility. The hypothesized intragenerational *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 (more flexibility in the stratification system). 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 can protect themselves against the anticipated threat of falling on the economic ladder. 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 socioeconomic mobility over the life course. It is also possible that individuals' experiences of occupation or SES mobility are 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 health are related. To keep our framework simple, however, we omit any explicit mechanism connecting individual health and individual experiences of income mobility (intragenerational).

Regarding the connection between m_i and M_c, it is reasonable to expect that *intergenerational* mobility m_i has a similar impact on decision making and long-term planning as contextual *income mobility* M_c, as place's income mobility would be just an aggregation of individual experiences of *intergenerational* mobility. This brings up the question of how persons define their perceptions about their place's mobility regime and how resistant these perceptions are over time. All the potential mechanisms described above are based on the idea that people can experience and perceive a given *mobility regime*. However, distinguishing in a clear way between the place's income mobility, the perception of place's income mobility, and individual *intergenerational* mobility experiences is not an easy task. There might be lagged effects – it takes time to align individuals' perceptions with actual changes in the stratification regime – and significant others' experiences

(i.e., family, neighbors, friends, acquaintances) can also impact how individuals' perceptions of a stratification regime are created (Hauser & Norton, 2017). It might be that the critical factor is the intergenerational mobility experiences of parents, rather than the individual. We expect all of these measures to be positively correlated, although not perfectly. Thus, we assume individuals can *correctly* perceive the contextual income regime where they live (i.e., empirical estimates of place's income mobility). We are aware that this assumption is limited. However, only additional research or simulation models implementing robust theory would help us to distinguish the consequences of these different measures when testing the hypotheses discussed above.

The link between M_c (community income mobility) and I_c (community income inequality) also deserves some remarks. First, the contextual conditions generated by income inequality might be a direct cause of income rigidity. For instance, nepotistic relations are more prevalent 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 health. Under this assumption, estimating mobility's independent contextual effect 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.

To illustrate the complexity of the effects we have discussed, we can imagine a counterfactual scenario where 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 steady-state income mobility. Suppose there is an intervention that improves educational attainment among those with fewer resources. We would expect that those changes would affect communities' income and its distribution in the long run. 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 current socioeconomic opportunities both due to intergenerational experiences of mobility or one of the 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 amplify 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 effect that is not due to income. In this *ideal* scenario, adjusting additionally for income inequality might 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 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 we use.

2.2.2 Outline general conjectures

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, provided we avoid post-treatment bias and overfitting (McElreath, 2020).

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 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. However,

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 income mobility differences 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.). Still, in other cases, inequality might be due to increases in the population size at the lower-income ranks. Social mobility might also occur at different levels of the income distribution. Unless we are able to define relevant theoretically relevant configurations formally, we cannot aspire to formulate precise predictions, models, or estimations. This is the motivation behind the idea of proposing a generative theoretical model of the impact of income mobility on health.

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.

In the next chapters, I explore some of these hypotheses using aggregate and individual data, and simulation models.

Income mobility, income inequality and mortality in the US

Working with aggregate data is dangerous at best, and disastrous at worst.

Aitkin and Longford

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

[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.

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 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;

[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.

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,

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

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, estimated by regressing child's income rank in her birth cohort national income distribution and parents' income rank in their parents' national income distribution.[5] The relative income mobility indicator ranges between -1 and 1, and larger values correspond to lower income mobility (higher rank-rank slope between parents' and child's income rank).

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 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)

[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.

[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).

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-

[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.

dispersion by adding a random effect at the observation level.[10] The general model is specified as follows:

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

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

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

[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 $\log(\lambda_i) = \log(\mu_i) - \log(\tau_i)$.

(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) 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

[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 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).

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.

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

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

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

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

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 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

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

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 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 influ-

enced 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.

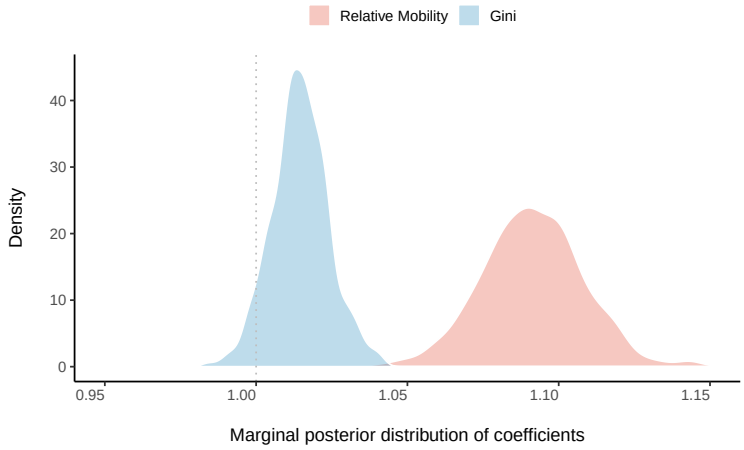
Table 3.2: County Level Poisson Models Relative Mobility
PC Prior $= Pr(\sigma > 1) < 0.10$, Men, CDC 2000-2014

	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

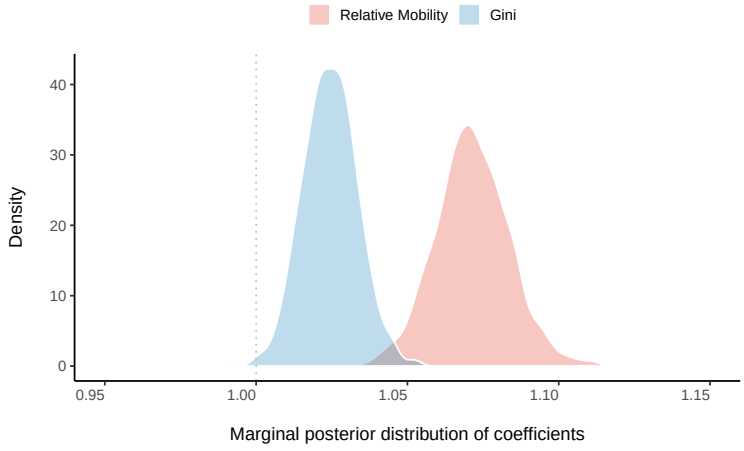
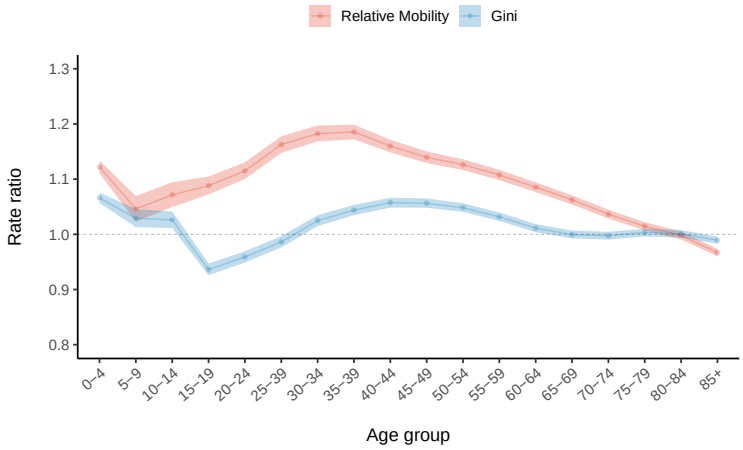


Figure 3.2: 95% Credibility Interval Posterior Distribution of $exp(\beta_m)$ and $exp(\beta_g)$ (Equation 3.1) by Age Group
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

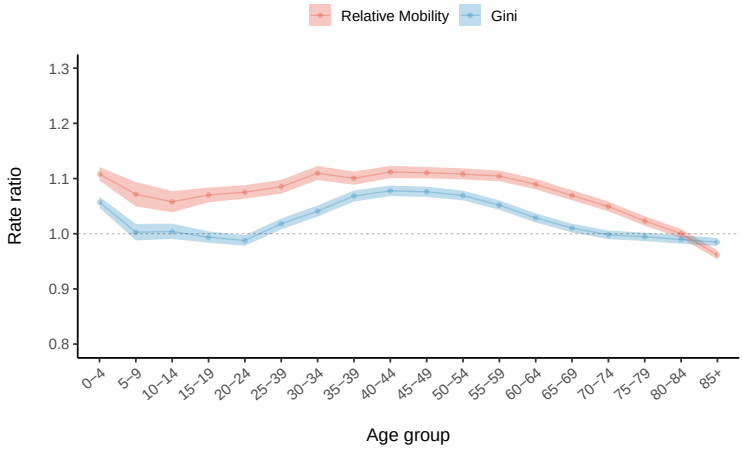
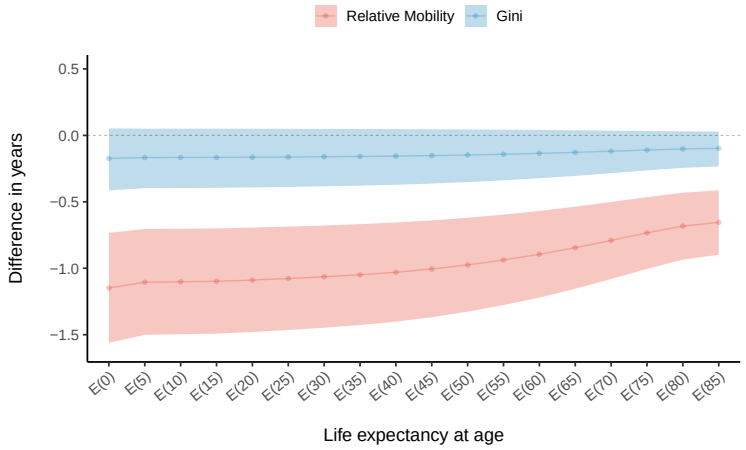


Figure 3.3: 95% Credibility Interval of Predicted LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

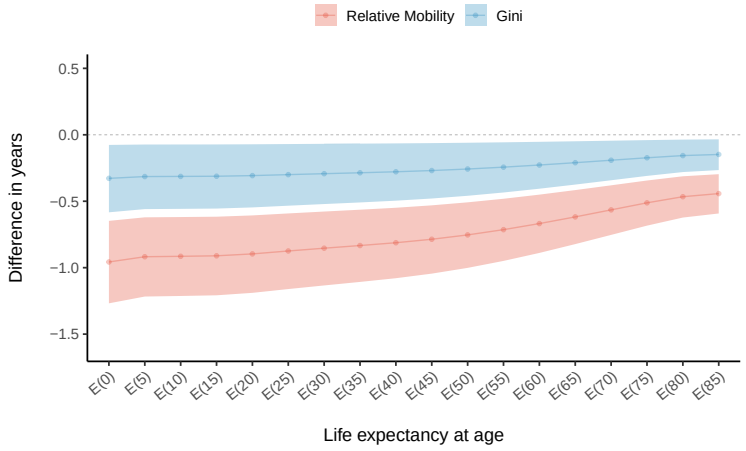
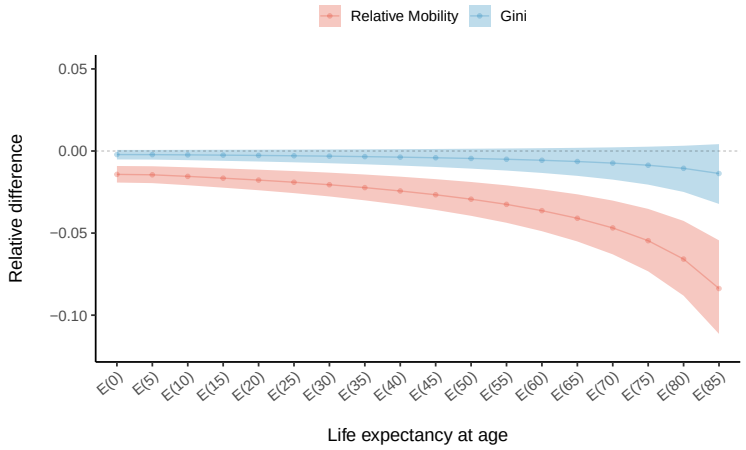


Figure 3.4: 95% Credibility Interval of Predicted Relative LE Differences by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men

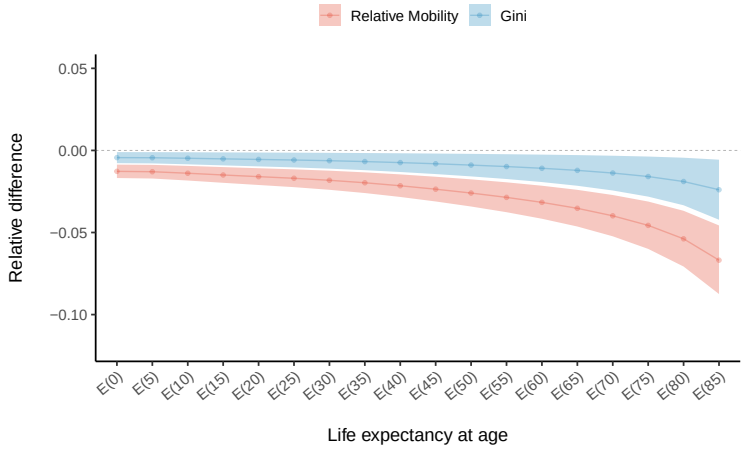
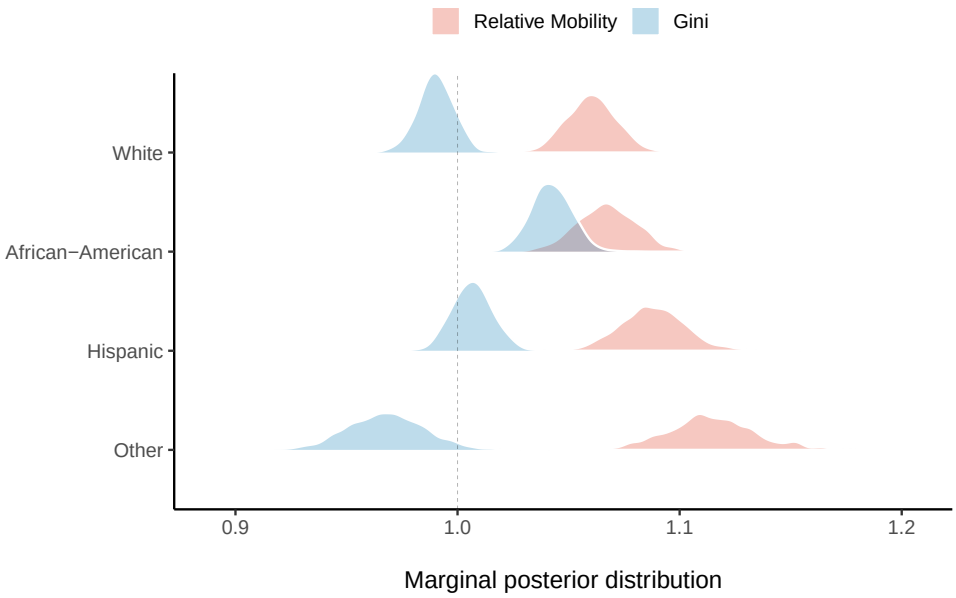


Figure 3.5: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta_{\text{Gini}})$ (Equation 3.1) by Race/Ethnicity and Gender

(a) Women



(b) Men

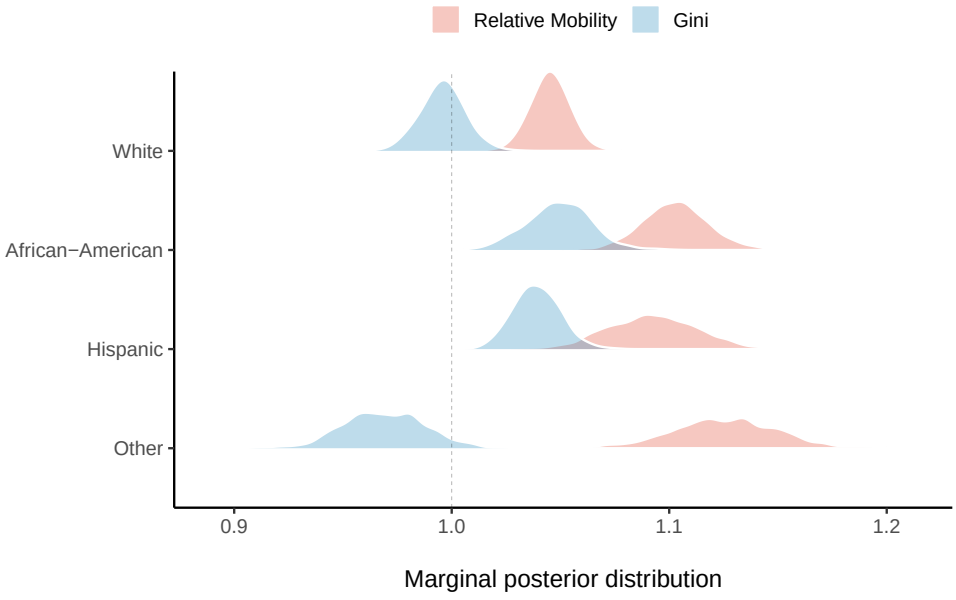
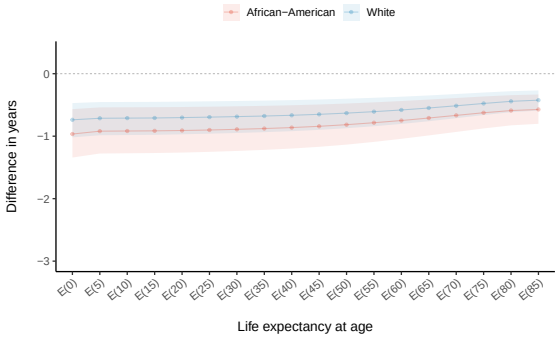
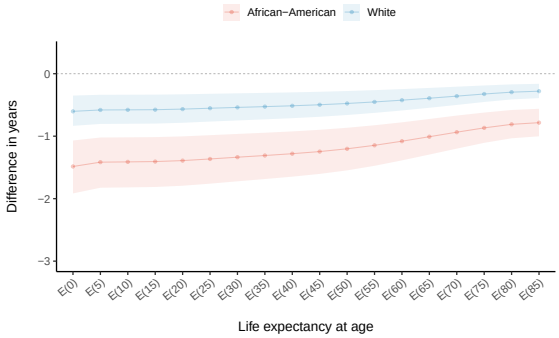


Figure 3.6: 95% Credibility Interval of Predicted LE Differences
by Age Group and Race/Ethnicity, Increase in One Standard Deviation

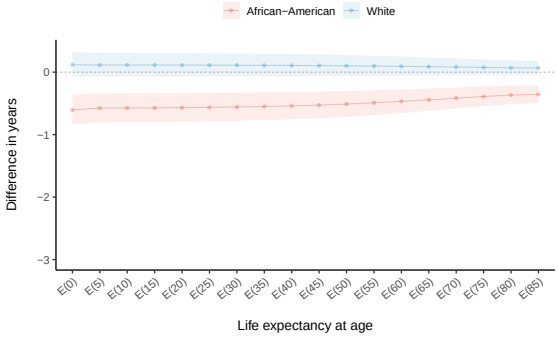
(a) Women - Relative Mobility



(b) Men - Relative Mobility



(c) Women - Gini



(d) Men - Gini

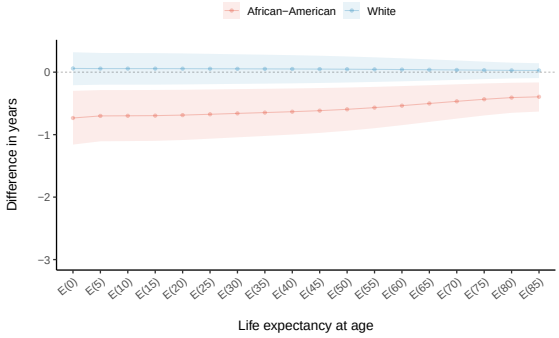
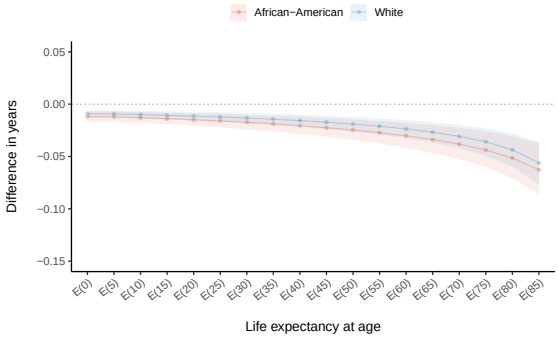
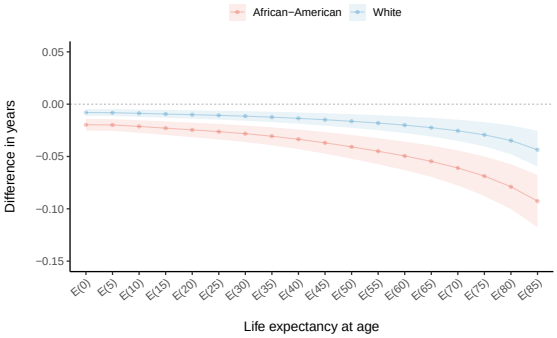


Figure 3.7: 95% Credibility Interval of Predicted LE Relative Differences by Age Group and Race/Ethnicity, Increase in One Standard Deviation

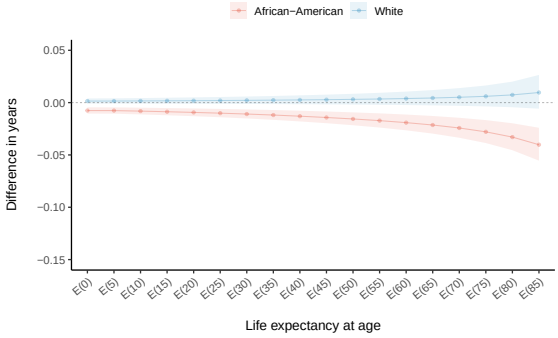
(a) Women - Relative Mobility



(b) Men - Relative Mobility



(c) Women - Gini



(d) Men - Gini

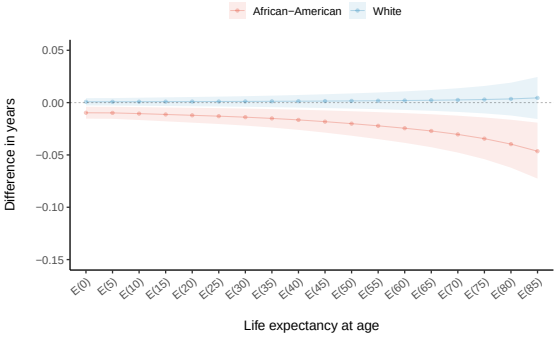
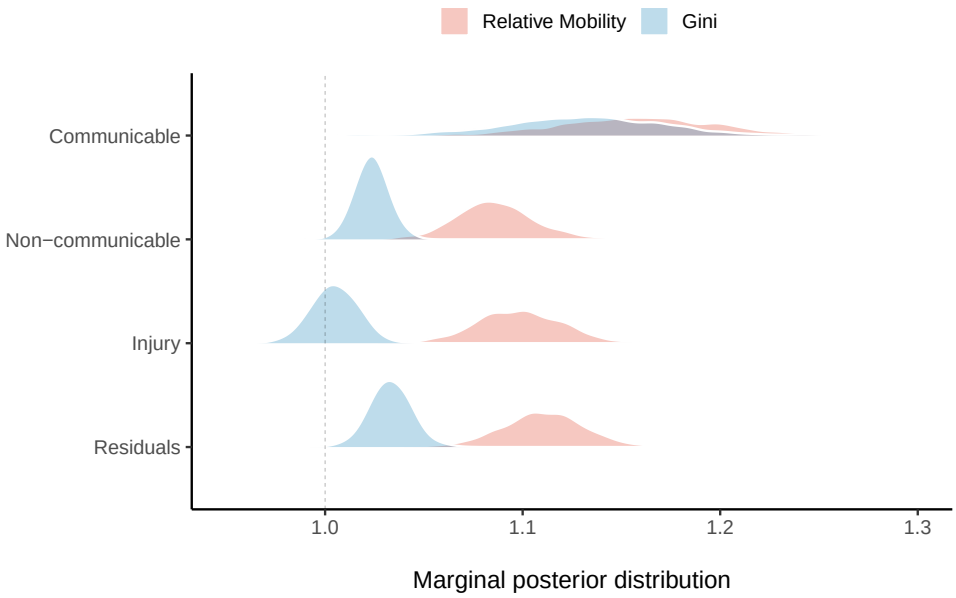
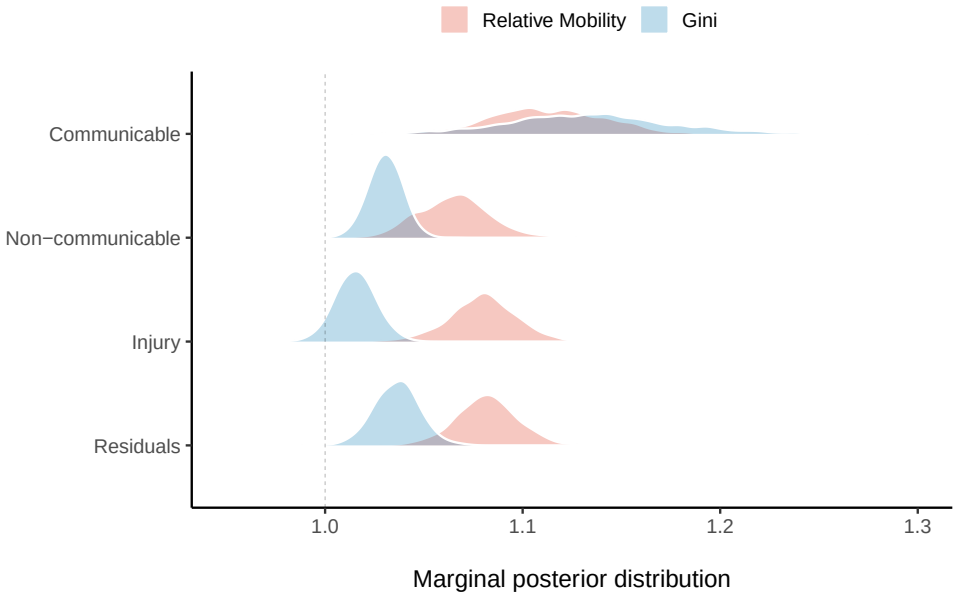


Figure 3.8: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta\text{Gini})$ by Cause of Death and Gender

(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%)

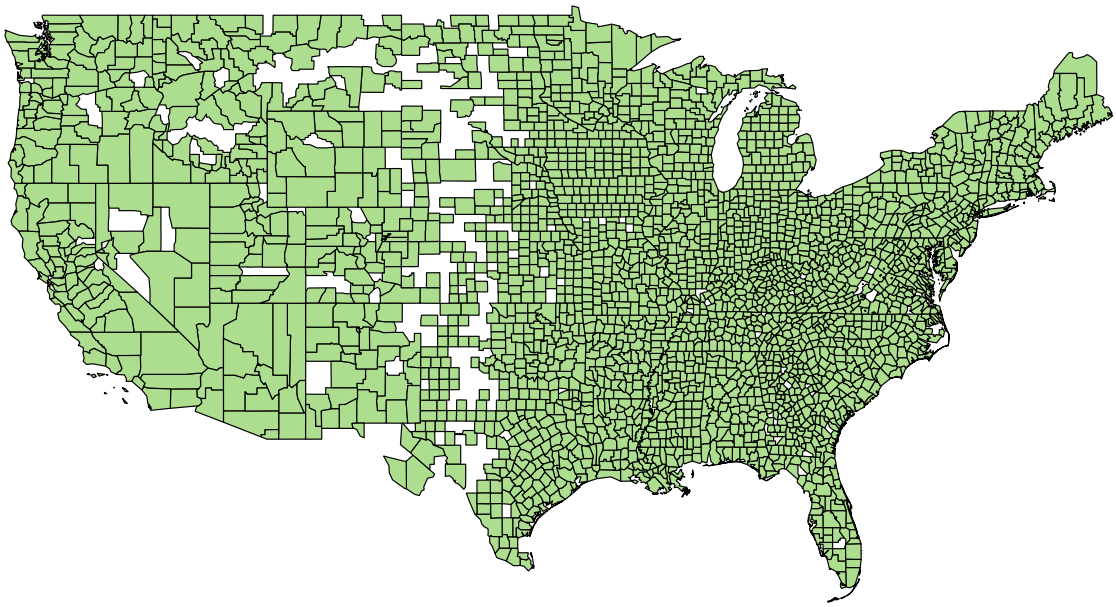
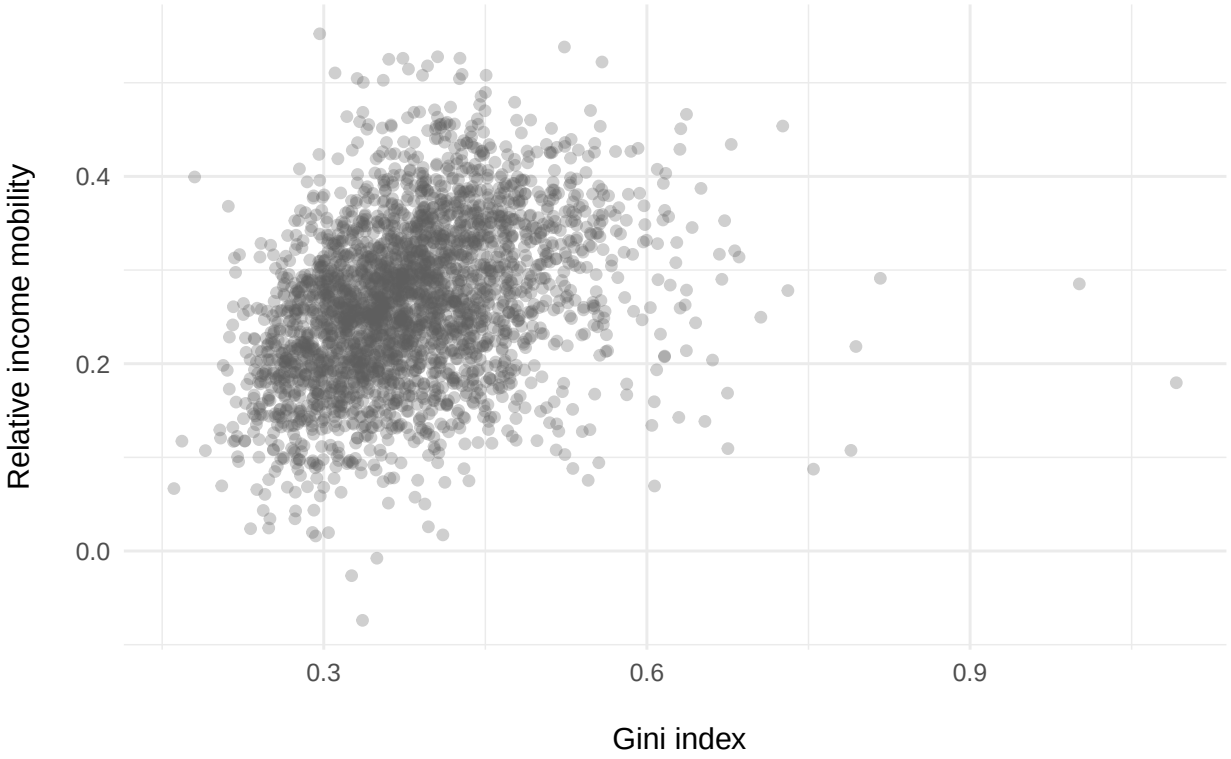


Table 3.3: Descriptive Statistics

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:

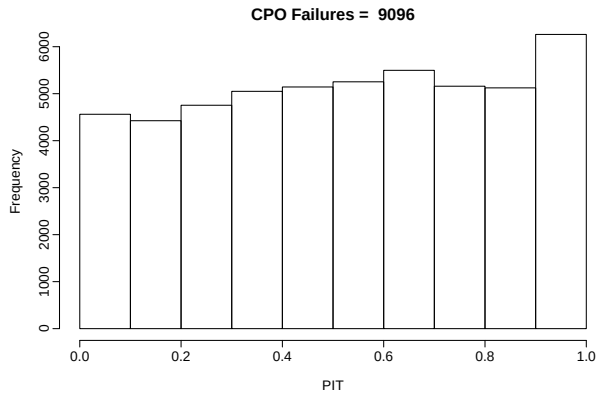
$$PIT_i = p(y_i^{new} \leq y_i | y_{-i})$$

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.

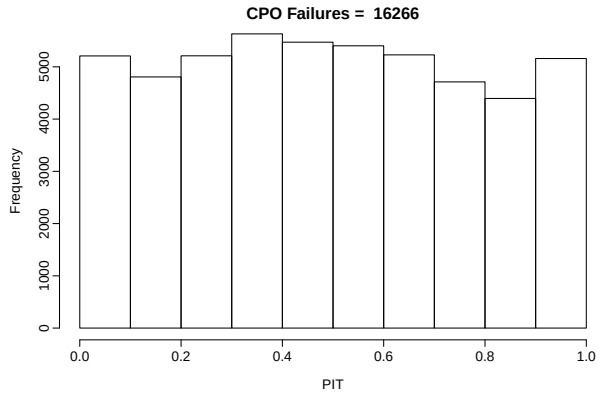
Figures 3.11 and 3.12 show the histogram and the uniform Q-Q plot of PITs for females and males. As can be seen, the distribution of the PITs is close to a uniform distribution, suggesting that the model reasonably fits the data.

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

(a) Women



(b) Men

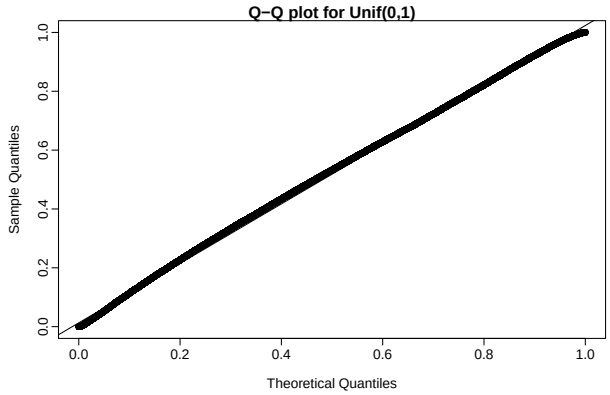


3.7.3 Prior sensitivity analysis

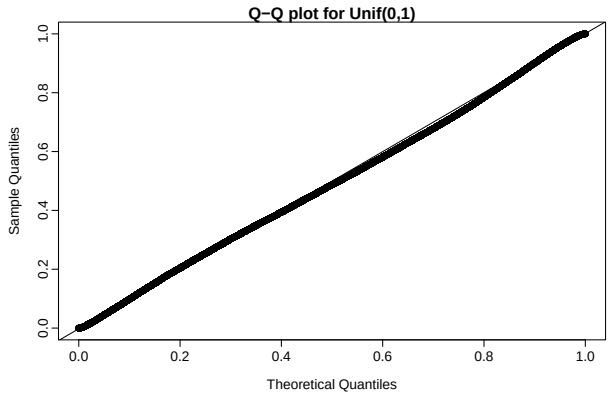
We perform prior sensitivity analysis. We start using the `R-INLA` default priors. `R-INLA` uses a precision measure for the variance of the posterior distribution parameters defined as $\tau_\varepsilon = \frac{1}{\sigma_\varepsilon^2}$. The default prior distribution for a fixed parameter is a normal 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

Figure 3.12: Q-Q Plot PIT
Model *Covariates* in Tables 3.1 and 3.2

(a) Women



(b) Men



(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.

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.

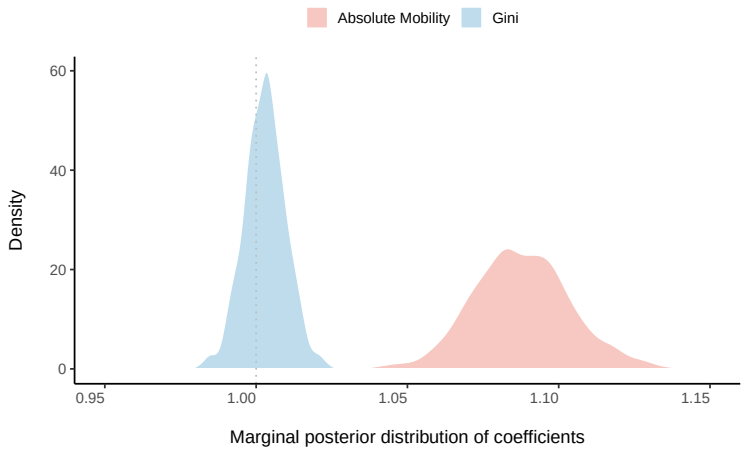
Absolute upward income mobility ranges from 0 to 1, and higher values correspond to large income mobility. We multiply the absolute upward mobility score by -1 so that the interpretation and expected association of relative and absolute income mobility were the same (i.e., increases in income mobility 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

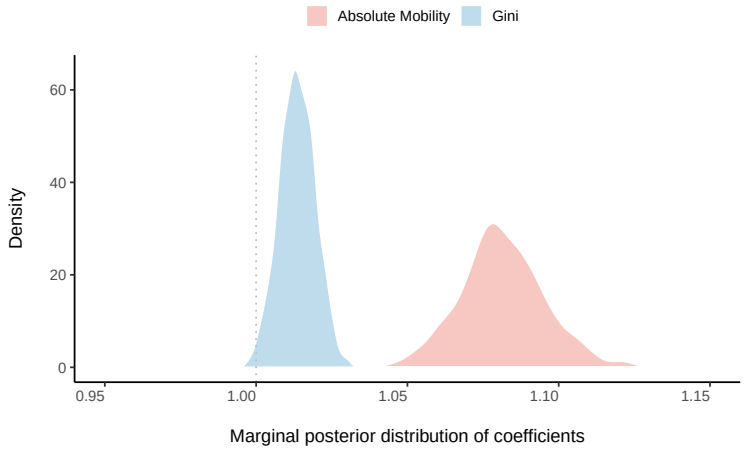


Table 3.6: County Level Poisson Models Absolute Mobility
PC prior $= Pr(\sigma > 1) < 0.10$, Women, CDC 2000-2014

	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.

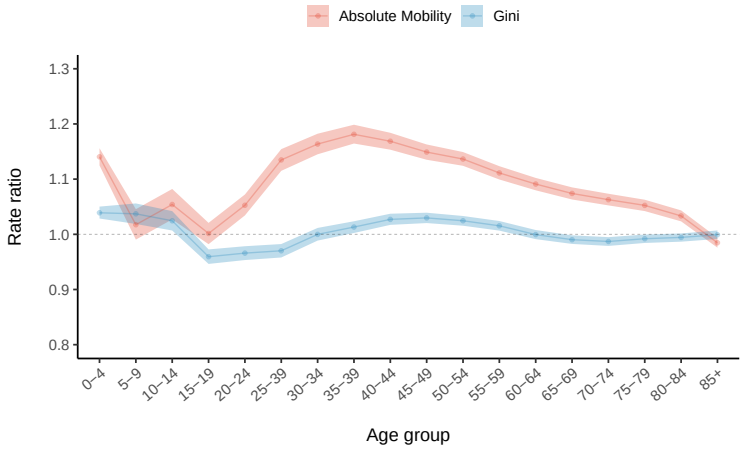
Table 3.7: County Level Poisson Models Absolute Mobility
PC Prior $= Pr(\sigma > 1) < 0.10$, Men, CDC 2000-2014

	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

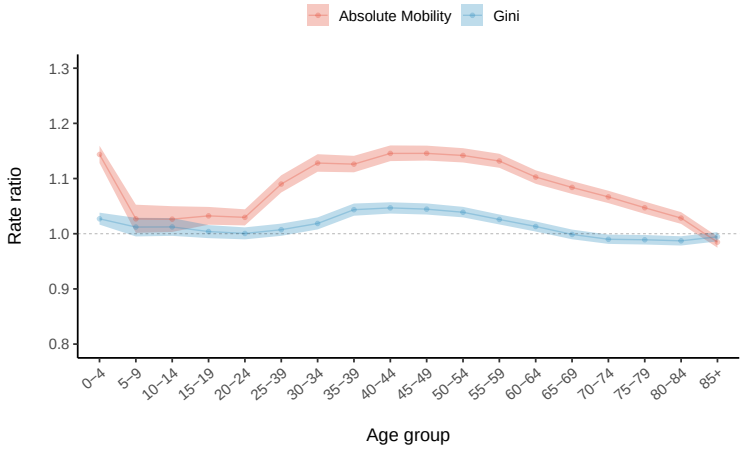
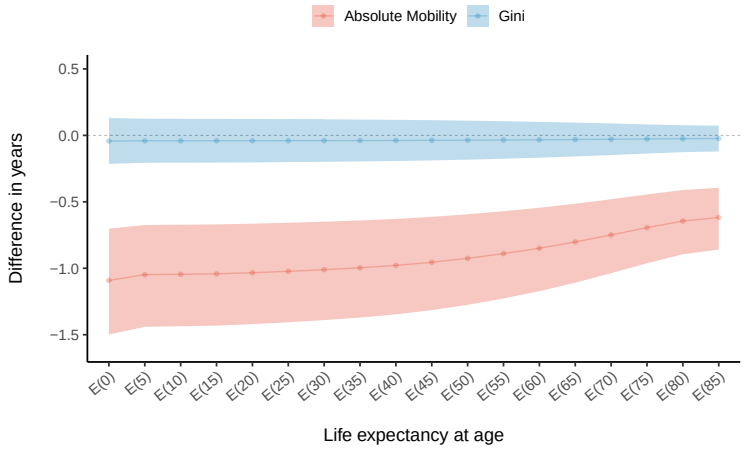


Figure 3.15: 95% Credibility Interval of Predicted LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.6 and 3.7

(a) Women



(b) Men

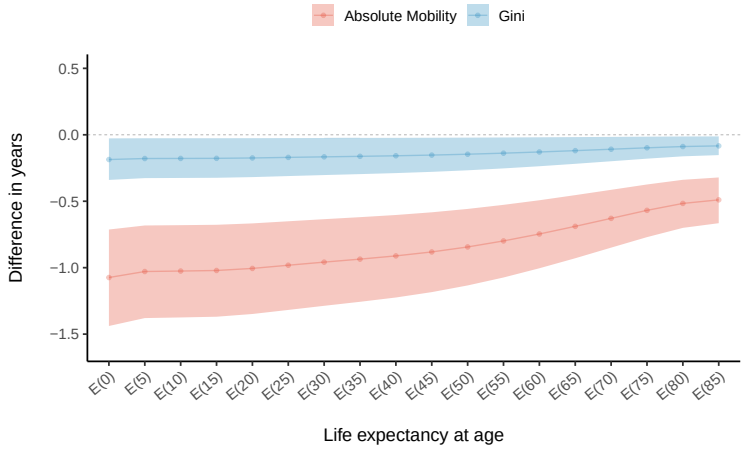
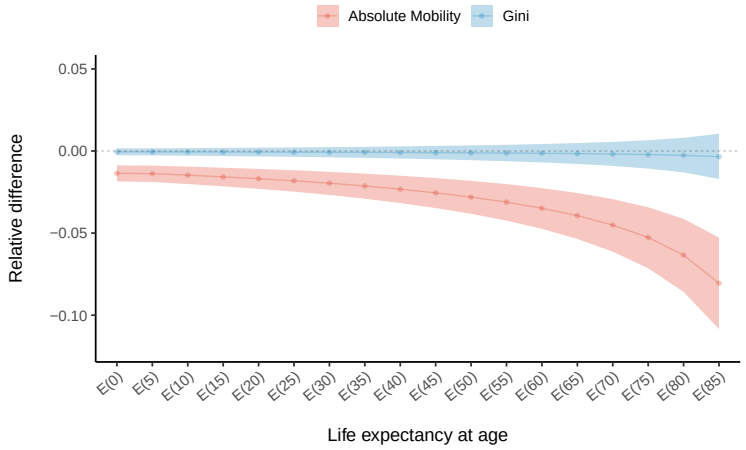


Figure 3.16: 95% Credibility Interval of Predicted Relative LE Differences
by Age Group, Increase in One Standard Deviation
Model *Covariates* in Tables 3.6 and 3.2

(a) Women



(b) Men

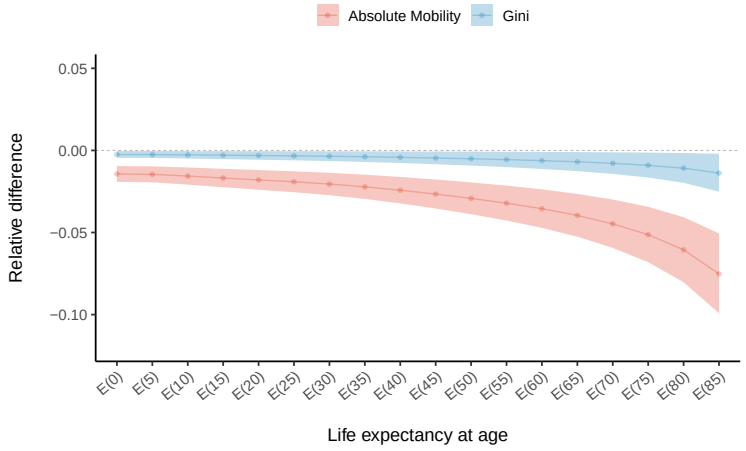
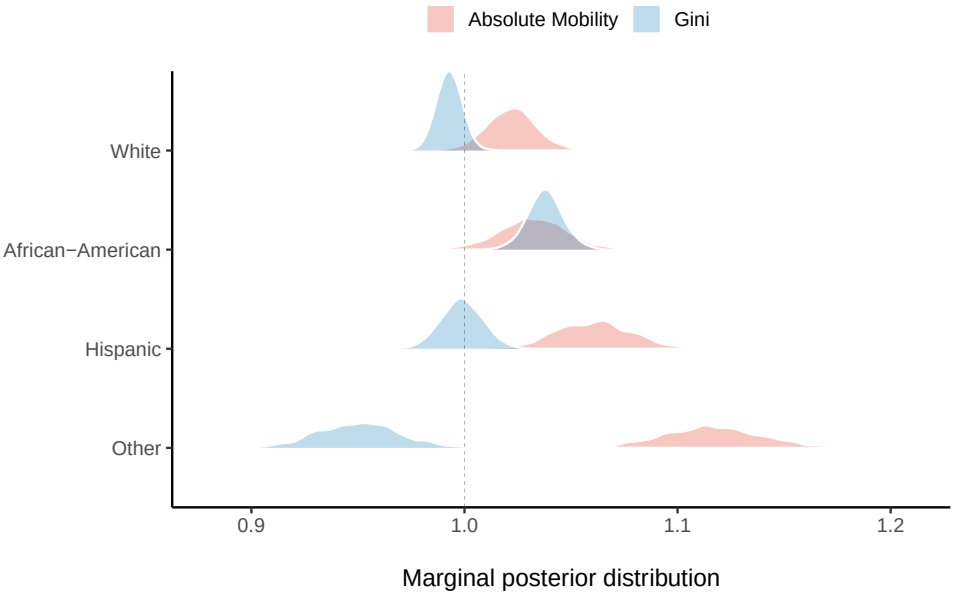


Figure 3.17: Posterior Distribution $exp(\beta_m)$ and $exp(\beta_g)$
Model *Covariates*

(a) Women



(b) Men

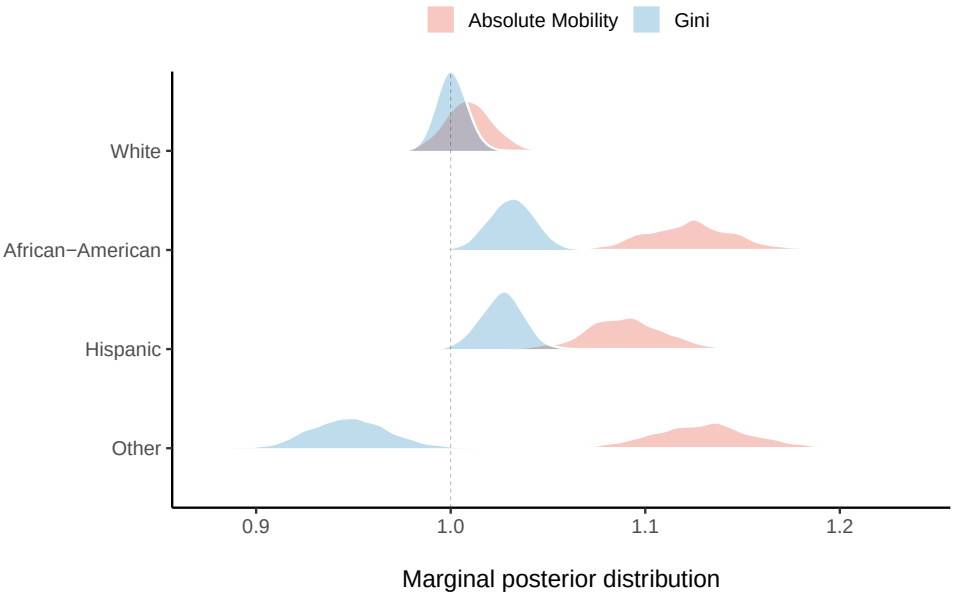
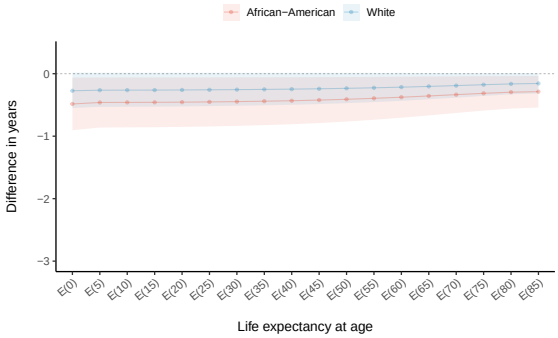
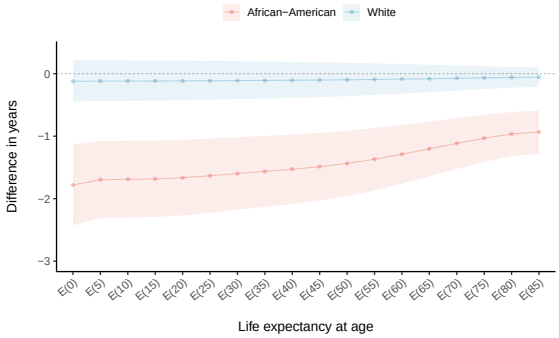


Figure 3.18: 95% Credibility Interval of Predicted LE Differences
by Age Group and Race/Ethnicity, Increase in One Standard Deviation

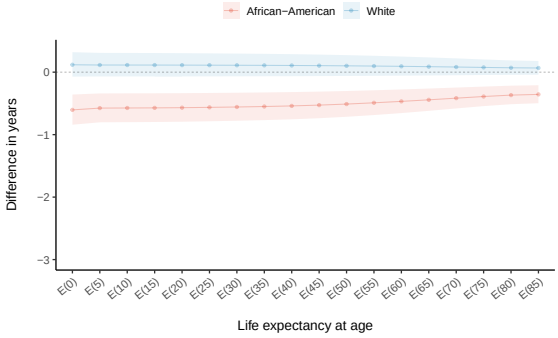
(a) Women - Absolute Mobility



(b) Men - Absolute Mobility



(c) Women - Gini



(d) Men - Gini

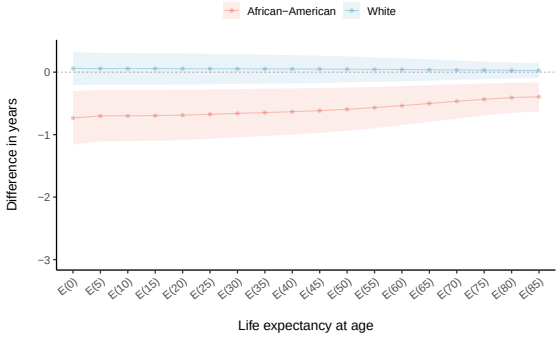
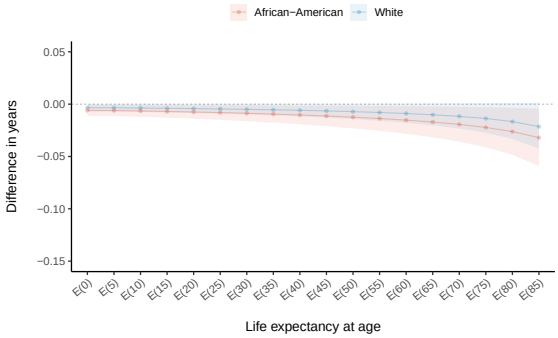
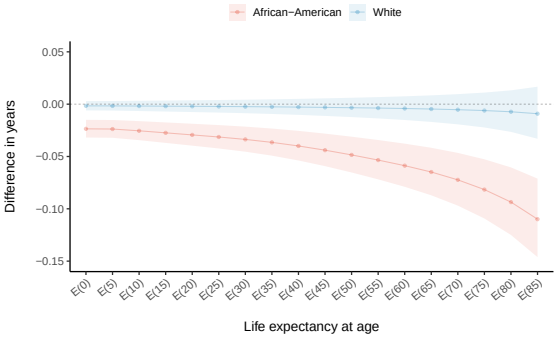


Figure 3.19: 95% Credibility Interval of Predicted LE Relative Differences by Age Group and Race/Ethnicity, Increase in One Standard Deviation

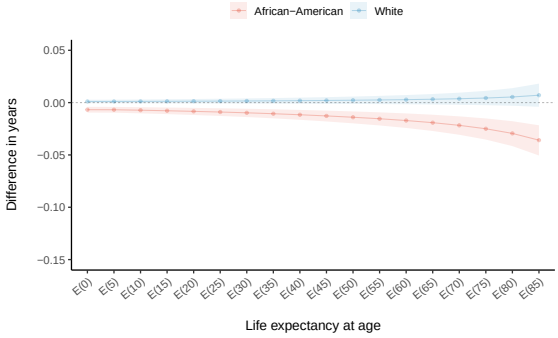
(a) Women - Absolute Mobility



(b) Men - Absolute Mobility



(c) Women - Gini



(d) Men - Gini

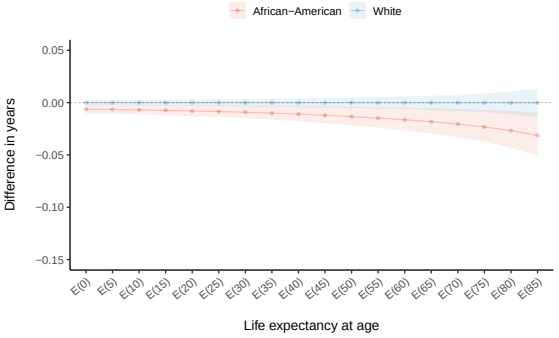
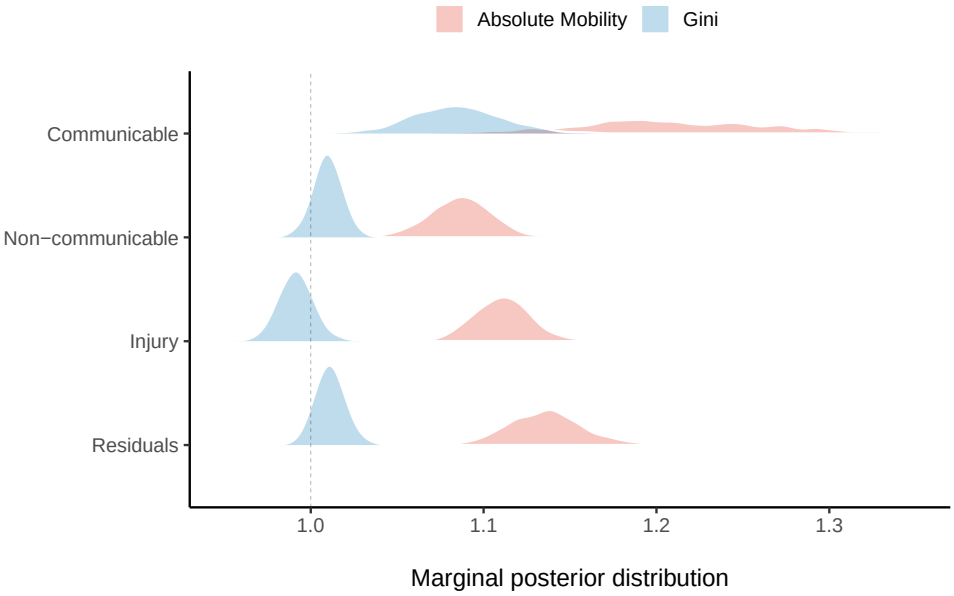
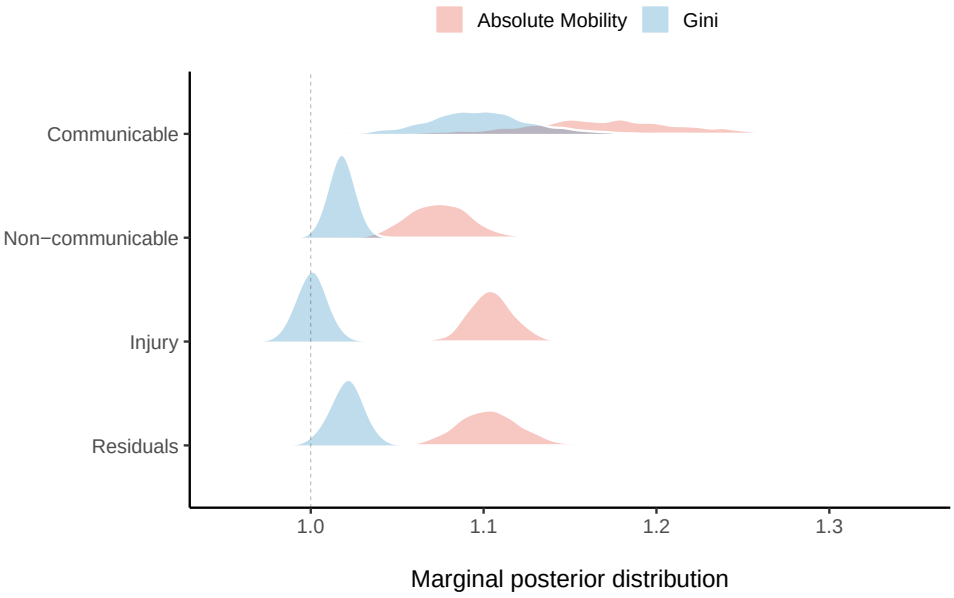


Figure 3.20: Posterior Distribution $exp(\beta_{\text{Mob}})$ and $exp(\beta\text{Gini})$
by Cause of Death and Gender

(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 income mobility and health in the US

Exploring the unknown requires tolerating uncertainty.

Brian Greene

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

[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.

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 (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 includes statistics of the income distribution and two indicators of income mobility derived from measures of the association between income 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 slope of a regression model between children's national income rank – within a birth cohort – and their parents' national income rank.[5] For the relative income mobility indicator larger values correspond to lower income mobility (i.e., higher rank-rank slope 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

[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 outmigrated as adults.

[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 sensitive to changes in inequality across generations. It is also important to note that the rank-rank slopes are not necessarily equivalent to the rank-rank correlation (i.e., Spearman's correlation) within a county. The ranks are computed using the national distribution income not the county distribution. (Chetty et al., 2014), however, argue that both measures are highly correlated at the place level (i.e, counties or commuting zones).

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 the timing of early exposure to the place's 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

[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.

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 under-represented (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 *Method-*

[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

ological 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 12th 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 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*

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 van Buuren (2018) 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.

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

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

[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.*

NOCB methods. About 44% of the PSID sample moved to a different county during the exposure 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 the 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[17] on the average exposure to a rigid stratification environment implies an increment of 2% ($exp(0.02) = 1.02$) in the odds of reporting excellent health (versus bad health)[18]. 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 slope 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 slope 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.

[17] The standard deviation of the rank-rank slope at the county level is 0.086.

[18] 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.

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 estimates 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 3rd 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.[19]

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.[20] 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 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.

[19] 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.

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

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 that 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 slope). 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 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 that 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 involve 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 the observed data, despite adjusting for an extensive set of observed covariates. Second, although we use different specifications and the 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 imputations. 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 the 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

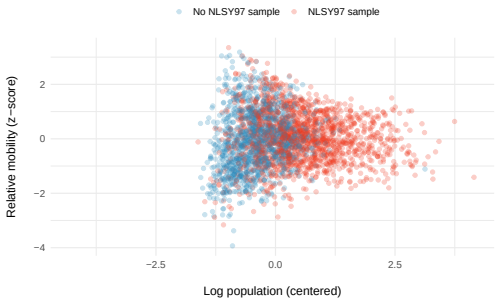
The code to create analytic data sets, models and plots is available at: https://github.com/sdaza/dissertation/tree/master/ch03. Some of the variables in the paper are restricted and obtained under special contractual arrangements to protect the anonymity of respondents. These data are not available 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

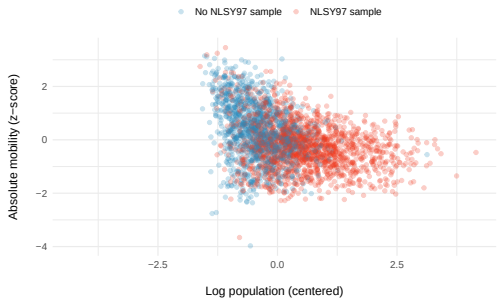
Figure 4.1 displays counties by the log of population and measures of income mobility and inequality, highlighting in red the counties included in the NLSY sample.

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

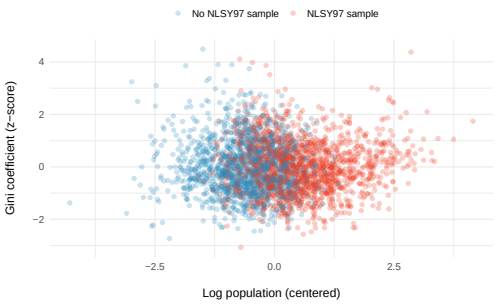
(a) NLSY97 Relative mobility



(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

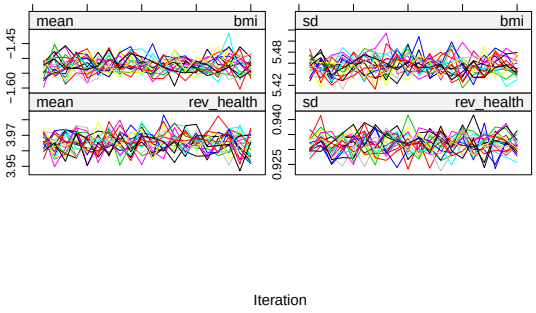
Note: Statistics based on non-imputed data. SD = Standard deviation. Observations correspond to respondents in the case of time-invariant and outcome variables, and person-years (N times exposure) for time-variant variables. Outcomes were measured in 2017.

Imputation

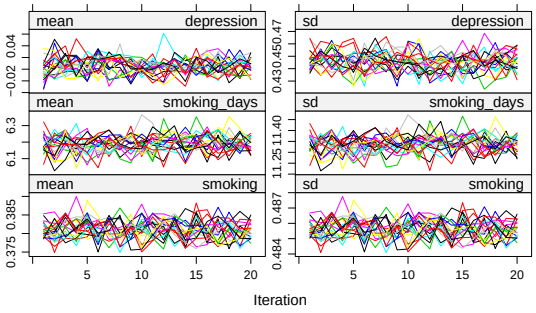
We employed multiple imputation for item non-response and attrition. For each exposure variable (e.g., relative and absolute income mobility), we ran multilevel models to impute values for both

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

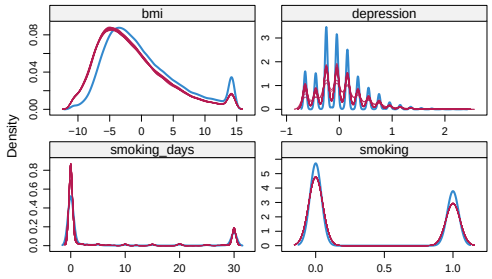
(a) Iterations



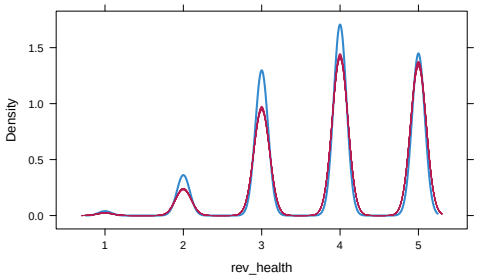
(b) Iterations



(c) Imputations vs Observed Values



(d) Imputations vs Observed Values

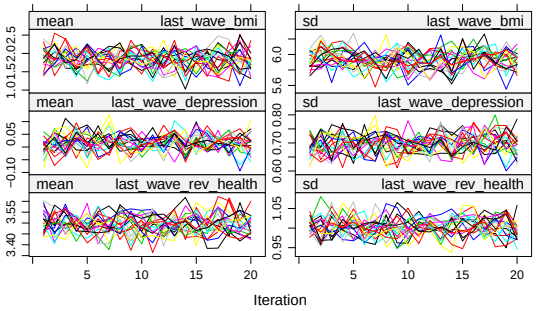


time-variant and invariant covariates. We produced 20 complete data-sets and pooled the results using Rubin's Rules (van Buuren, 2018).

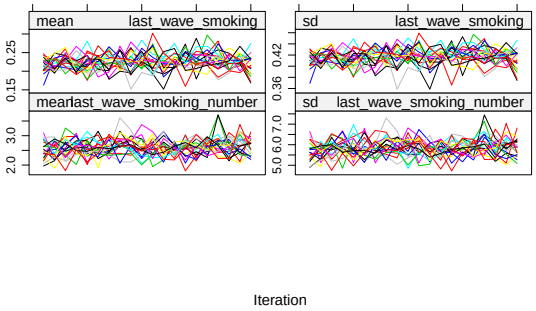
Multiple imputation model specifications are available in our code repository. For each exposure variable, we produced 20 complete datasets per data source (PSID and NLSY9). Different imputed data-sets were created for residualized and non-residualized, continuous and categorical exposure (360 datasets in total). We assessed convergence and feasibility of results using the criteria suggested by van Buuren, 2018. 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

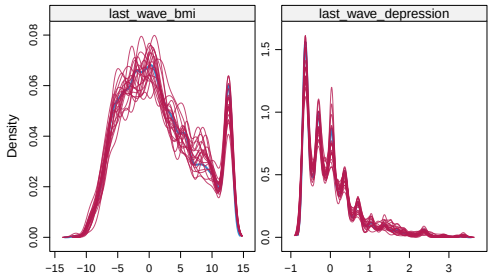
(a) Iterations



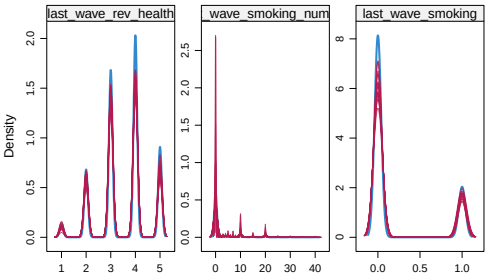
(b) Iterations



(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.

Exploring the link between place's income mobility and mortality using an agent-based model

Art is a lie that makes us realize truth, at least the truth that is given us to understand.

Pablo Picasso

Two trends have characterized US socioeconomic and health trends in recent decades. On the one hand, while income inequality has increased since the 1970s (Gould, 2019), upward income mobility has declined since the 1940s (Chetty et al., 2017). On the other, life expectancy disparities across socioeconomic groups have widened. Chetty et al. (2016), for instance, shows life expectancy between 2001 and 2014 has increased 2.34 years for men and 2.91 years for women in the top 5% of the income distribution, while only 0.32 years for men and 0.04 years for women in the bottom 5% of the distribution. This increase in mortality differences between better-off and disadvantage people represents a fundamental challenge for health policy.

While current inequality and upward mobility have clear economic consequences, how these new socioeconomic conditions may affect health and mortality is less evident. At first glance, arguing that changes in income inequality and mobility may foster health behaviors and outcomes disparities across social groups does not seem eccentric. In fact, the hypothesis about the link between

socioeconomic mobility and health has gained ground, not only because previous research shows neither access to medical care nor socioeconomic factors fully explain the observed geographic or income disparities in longevity, but because socioeconomic mobility rates, as life expectancy does, vary considerably by geography (Chetty et al., 2014). Some scholars have suggested that place's 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 consequences would be different from the ones associated with income inequality, as individuals living in areas with similar degrees of income inequality may experience different income mobility regimes and impacts on health outcomes.

Previous research provides some evidence of 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, using two longitudinal datasets, the Panel Study of Income Dynamics (PSID) and National Longitudinal Surveys of Youth (NLSY 1997), a researcher Daza and Palloni (2021) found only partial empirical evidence supporting the connection between exposure to county's income mobility during childhood and adolescent and smoking in early adulthood

The link between place's income mobility and health, however, is not a simple one. The processes connecting health, income mobility and inequality involve individual, contextual, spatial, reciprocal and cumulative effects as well as feedback loops. Unfortunately, little effort has been spent in formalizing the connection between the flexibility of the economic system, individual behavior, health and mortality. In this paper, we argue that formalizing the connection between income mobility and health is worthwhile because it allows us to assess the population consequences of individual estimates retrieved from empirical studies. It also enables us to identify

problems associated with aggregate or cross-sectional data when estimating the effect of place's income mobility on health. Finally, it is an exercise that will provide guidelines tp identify the type of data needed to test conjectures proposed in the literature.

Thus, we move away from statistical models and propose an initial formal model for the association between exposure to a given stratification system and health. 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 population-consequences of empirical estimates, and to identify under which conditions we can retrieve estimates of the effect of income mobility on health.[1]

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

5.1 Potential mechanisms

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 Chetty's interpretation suggesting that economic opportunity is a characteristic of place (Chetty et al., 2017). We add to that interpretation by proposing that prospects of income mobility would independently affect health and mortality.

Thus, we *do not focus* on the connection between individuals' lifetime income mobility expe-

[1]We model smoking behavior because previous empirical research using longitudinal data shows that the most systematic effect of exposure to income mobility regimes on individuals' health behaviors is via smoking (Daza & Palloni, 2021). In addition, smoking has well-known consequences for mortality risks that can be easily incorporated to our model.

riences 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 on the link between an *aggregate* property of the stratification system, on the 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.[2]

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 future 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 who 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? There are number of mechanisms that could produce this result. First, communities with low-income mobility may distort opportunities and incentives, reinforce unequal allocation of favorable traits and resources, 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 the way parents socialize children and favor (discourage) the adoption of positive outlooks and the value of skill acquisition. Rigid or weak income mobility

[2] At the individual level, the main effect of income mobility on health would operate through socioeconomic status and educational attainment. Moreover, it is also believed that mobility itself could affect health through the lack of or excessive stress when there is downward or upward 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 experiencing economic, social, psychological adversity, 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 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, 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, and bleak future outlooks associated with poverty lead to increases in time discounting (Haushofer & Fehr, 2014; Schilbach et al., 2016). 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 shapes 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 the individual's ancestral income mobility experience, particularly parental and possibly grand parental mobility.[3]

Finally, 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 are both more likely to experience mobility and to embrace health protective behaviors. In this case, the association between the aggregate property of the stratification system and individual experiences of health and mortality would reflect 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 (unob-

[3]One can argue that more important than the contextual income mobility of children is the mobility experienced by parents, or grand parents, as those experiences might strongly shape socialization and, in particular, time preferences of the next generations.

served) social and economic traits that might eventually promote good health and reduce mortality risks. It would not be income mobility itself what 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 conventional statistical models as they tend to produce the same observed results from data generated by different processes.[4] Simulation and *generative* models 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 step away from statistical models and propose a computer simulation that implements some of the mechanisms discussed above to assess the influences of socioeconomic mobility regimes on health. Specifically, we create a *low-dimensional realism* model where micro-level behaviors are assumed or known, and simulation is used to explore how the system behaves (Edmonds et al., 2019).[5] The scope of the model is mainly theoretical and exploratory.

The model represents three critical processes that may generate empirical data. First, it takes into account *space* by allowing individual agents to reside in a place, county or neighborhood, so that we can explore the hypothesis that place's income mobility (not intra or inter-generational mobility) might impact health and mortality. Agents can interact directly through the relationship with their parents and children but also indirectly by sharing the characteristics of their place (county) of residence. Second, individual preferences on residence's place are endogenously defined thus inducing selection of agents to exposure to places' characteristics (i.e., segregation). This endogenous definition of place's preferences could impact both the contextual characteristics agents are

[4]This issue is commonly referred to as 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 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 a formal and unequivocal way.

[5]This strategy is referred to as *exploratory modeling* (Wilensky & Rand, 2015).

exposed to and the economic rigidity or flexibility of the place's stratification system. Finally, and unlike what we usually do with empirical data, the simulation model tracks with precision each agents' trajectory and their exposure to income mobility contexts. This facilitates exploration of the influences that different data collection strategies may pose to retrieve the effects of interest using statistical models (Daza & Kreuger, 2019).

Because the agent-based model can explore and examine an artificial system closely mimicking real ones, it possesses a key advantage that endows with greater power than conventional hypotheses testing strategies: it can handle features like individuals' interaction, space and time dimensions, and feedback loops in a relatively simple and flexible manner. Because of this, the agent-based model offers the opportunity to rigorously test hypotheses, identify limitations of previous studies, and guide the design of future research. Its limitation is that the model cannot by itself resolve issues of estimation and identification when using empirical data. This is not a task that the model can handle.

The agent-based model we implement here formalizes a set of ideas about the implications of income mobility for health. In particular, we explore two research problems and in each case we suggest solutions and formulate new conjectures about the link between income mobility and health. First, we wish to assess the population-level consequences of empirical estimates of the individual link between exposure to income mobility and smoking. For this, we translate empirically estimates of the *relative* effect of income mobility on smoking, into absolute differences in life expectancy at the population level. We choose smoking behavior because previous empirical research using longitudinal data shows that the most systematic effect of exposure to income mobility regimes on individuals' health behaviors is via smoking (Daza & Palloni, 2021). In addition, smoking has well-known consequences for mortality risks that can be easily incorporated to our model. We are cognizant that by considering only one health-related behavior we are likely grossly underplaying the influence of income mobility. Thus, the low-dimensional realism of MIA, this *micro-simulation* exercise will only provide an approximation to the total aggregate consequences of income mobility for longevity

Second, we aim to examine under which conditions can one retrieve key parameters controlling the link between place's income mobility and mortality when using individual and aggregate data with different measures and modeling strategies. For instance, we can identify the conditions under which empirical results can be flawed due to ecological fallacy so that commonly reported associations in previous empirical studies might not be due to individual effects of income mobility on health, but to processes such as residential segregation, income composition of neighborhoods, or heterogeneity in agents' income mobility. This will enable us to assess the validity of previous research and to point to future data collection, measurement, and inferential strategies.

5.3 MIA: Mortality and Income Mobility Agent-based Model

We created 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 some of the mechanisms proposed in the literature. MIA consists of four essential modules: demography dynamics (mortality and fertility), residential mobility, income generation and mobility, and smoking behavior. Below, we describe the implementation of each of those components, in addition to the general model setup and data collection. We verified and tested each of MIA's modules. The results of those verification procedures are discussed 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 (see Table 5.4 in the *Methodological Supplement*). Age-specific mortality rates are defined every agent's birth date based on the following formula:

$$ {}_{a}m_{x_i} =_{a} m_{x_b} * exp(\beta_{m_k}\text{incomeType}_i + \beta_{m_{ie}}\text{incomeExposure}_i + \beta_{m_{smk}}\text{smokingStatus}_i) \qquad (5.1) $$

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), $\beta_{m_{ie}}$, the coefficient of the county's standardized income exposure, and $\beta_{m_{smk}}$, the coefficient for smokers (versus non-smokers). The coefficient β_{m_k} reproduces life expectancy gaps reported in previous research (Chetty et al., 2016), $\beta_{m_{ie}}$ is set to -0.1 to represent the potential effect of the environment an agent was exposed, and $\beta_{m_{smk}}$ is from empirical models using the US National Health Interview Survey (NHIS) (Jha et al., 2013). As expected, when β_{m_k}, $\beta_{m_{ie}}$, and $\beta_{m_{smk}}$ are equal to zero, agents' life expectancy across different generations is about 78.4 years (see the *Methodological Supplement* for more details), very close to the life expectancy of 78.6 reported by Kochanek et al. (2019) for the US in 2017 using the same mortality rates. It is important to note our baseline model does not include a direct link between county's income mobility exposure and mortality risk. However, to simplify statistical modeling when assessing the conditions under which we can retrieve income mobility effect on mortality, we added a fictitious effect of income mobility exposure on mortality β_{m_f}. Unless otherwise noted, that coefficient is always set to 0.0.

Our model does not include any information about gender, so to obtain a stable population overtime (i.e., 30 generations), we divided birth rates by an adjustment factor to make the population close to stationary (zero growth rate). This "fertility" adjustment factor was derived through calibration. We also applied an income group adjustment to ensure that the size of income groups remained relatively stable and even over time (e.g., the lowest income group has a higher mortality rate, but also a higher fertility rate relative to the highest income group). As expected, the average number of off-springs is about 1.00 (where about 37% of agents are childless), and the population size is relatively uniform over time. 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 allocated randomly in a space grid, they can move to a different county when they are 18 years old or more. To assess the consequences of reinforcing selection processes on health,

we generate income segregation by adapting Schelling's segregation model (Schelling, 2006). As this model shows, a simple rule of satisfaction can generate residential segregation even when agents are tolerant to living 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 is constant (nobody is born or dies), only two groups (colors) are used, and every agent stops moving when satisfied. Instead, in our version of the model agents decide at the 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.[6] With probability $1 - mob_{rand}$, agents assess whether to move or not based a similarity tolerance threshold mob_{thr} (e.g., 10%). When the proportion of people in an 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 stay in their 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. Figure 5.8 in the *Methodological Supplement* displays a agent's decision chart for residential mobility. As shown in the *Methodological Supplement*, this mechanism generates income segregation as values of neighbourhood sorting index (NSI) behave as expected (Figure 5.9).[7] According to Jargowsky and Wheeler (2017), the NSI for the US in 2010 was about 0.396. We adjusted the residential mobility parameters to reproduce those levels of income segregation in MIA.

5.3.3 Income generation and mobility

Agents are given an income category or group k (e.g., quintile) at age 18. After the first generation (G_0), income categories are assigned based on two factors. The first one consists of the intergenerational transmission of resources from parents to child. This process is defined by the probability

[6]For instance, more than 10% the expected county population N_c, where N_c is the population at time t divided by the number of counties.

[7]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 by income across counties, the income variation across counties will be similar to the income variation across agents, and the NSI will be approximately 1. If all counties are perfectly economically integrated (i.e., each county is a microcosm of the entire population), the NSI will be close to 0.

of inheriting agent's parent income group via a transition matrix I_b. We can use probabilities estimated from empirical data. Equation 5.2, for instance, shows the income quintile transition matrix I_b estimated by Chetty et al. (2014) for the US. These probabilities could be employed to assign agents to income groups over generations, so that the income mobility regime in our model is exogenous. Once the income group is assigned, the final income (currency) is sampled from the US Census and American Community Survey microdata (IPUMS) by quintile (Ruggles et al., 2020).[8] Income is defined at age 18 and remains constant throughout the agent's life (i.e., permanent income).

$$I_b = \begin{bmatrix} \mathbf{0.337} & 0.280 & 0.184 & 0.123 & 0.075 \\ 0.242 & \mathbf{0.242} & 0.217 & 0.176 & 0.123 \\ 0.178 & 0.198 & \mathbf{0.221} & 0.220 & 0.183 \\ 0.134 & 0.160 & 0.209 & \mathbf{0.244} & 0.254 \\ 0.109 & 0.119 & 0.170 & 0.236 & \mathbf{0.365} \end{bmatrix} \quad C_k = \begin{bmatrix} 0.45 \\ 0.30 \\ 0.10 \\ 0.10 \\ 0.05 \end{bmatrix} \quad W_k = \begin{bmatrix} 0.4 \\ 0.4 \\ 0.4 \\ 0.4 \\ 0.4 \end{bmatrix} \tag{5.2}$$

It is also possible to change the values of matrix I_b to explore different scenarios. For example, we can examine what would happen if there is no income mobility at all (i.e., the mobility matrix I_b has ones in the diagonal and zeros elsewhere), if the chance of inheriting the agent's parent income group is 60%, or whether there is total (neutral) income mobility (i.e., all probabilities in I_b are set to 0.20).

The second component of the income definition is contingent on where agents lived before age 18. Specifically, the probability of being in a given income category is affected by the average exposure to the residence county's income composition by 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 parents moved.[9] For this, we define two vectors:

[8] We used a sample of 250,000 income values to assign income by quintile. The Gini coefficient of the income distribution sampled from the IPUMS data is 0.48.

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

C_k and W_k. The first one, C_k, is the average income group composition of counties in which agents have lived during their first 18 years of life. W_k, in turn, is a weighting factor for combining the transition matrix I_b and C_k. W_k allows us to control the relative importance of the county composition in the agent's income generation.

For illustrative purposes, we assigned values to the vectors C_k and W_k in Equation 5.2. The first value of C_k is 0.45, while the last value is 0.05. This means that an agent i has spent her first 18 years of life in counties where, on average, 45% of the active population (18 years or more) is part of the first quintile of the US income distribution, while only 5% correspond to the richest quintile. These values might reflect both changes in the county of residence and county's composition. The transition matrix I_b can be updated using C_k and W_k, and computing a weighted average between I_b probability and the average exposure to counties' income composition as shown in Equation 5.3. Thus, the probability i_{11} in I_u would be $i_{11}(1-w_1)+c_1w_1 = 0.337\times(1-0.4)+0.45\times 0.4 = 0.382$. In other words, the probability $i_{11} = 0.337$ in the matrix I_b would increase from 0.337 to 0.382 as agent i has been exposed to counties with a higher proportion of people in the first income quintile, and the contribution of the county's income composition was set to $w = 0.4$. Similarly, the probability i_{55} in I_u would decrease from 0.365 to $i_{55}(1-w_5)+c_5w_5 = 0.365\times(1-0.4)+ 0.05\times 0.4 = 0.239$ as only 5% of people where agent i has lived are in the highest quintile. The vector W_k allows us to adjust the relative importance of the two income generation components. Thus, when $w_k = 0$, I_u would be equal to I_b, while when $w_k = 1$, I_b would be equal to C_k for each income group. We verified these income generation components and explored the income distribution and transition probabilities generated when using different values for I_b, C_k, and W_k (see the *Methodological Supplement* for more details).

$$I_u = \begin{bmatrix} i_{11}(1-w_1)+c_1w_1 & \dots & \dots & \dots & i_{15}(1-w_5)+c_5w_5 \\ i_{21}(1-w_1)+c_1w1 & \dots & \dots & \dots & i_{25}(1-w_5)+c_5w_5 \\ \dots & \dots & \dots & \dots & \dots \\ \dots & \dots & \dots & \dots & \dots \\ i_{51}(1-w_1)+c_1w_1 & \dots & \dots & \dots & i_{55}(1-w_5)+c_5w_5 \end{bmatrix} \tag{5.3}$$

In sum, our model simplifies the income generation process to two factors: (1) parent's income, and (2) 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 that are valued in labor markets. However, all these factors are represented through the average exposure to counties' income composition (C_k). In the same vein, family transmission of skills, cultural capital, behaviors, and access to education and labor market opportunities are represented solely through the association between parent's and child's income (I_b). 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 childhood, and vice versa. However, this compensation process is unlikely to occur in contexts in which income segregation is common.

Our segregation rules do not modify individual income mobility if agents do not change their income based on characteristics of the place where they live. By combining family (I_b) and county *resources* (C_k), we can establish a feedback loop between income and county of residence in such a way that income is assumed to depend on the neighborhood while the neighborhood during adulthood is assumed to depend on individual income.

5.3.4 Smoking

Inspired by the Grossman Model (Grossman, 1972) and the mechanisms discussed in previous sections, we decided to use *smoking* as the key behavior determining mortality risk. According to Grossman (1972) and previous research, individuals with higher discount rates would invest less in their health. Thus, time discounting would be related to health behaviors such as physical activity, substance use, and smoking, representing an important mediating factor linking environmental, social, life-course factors, and health (Barlow et al., 2016; Story et al., 2014). It is important to note that smoking is just one of many possible health behaviors affected by income mobility. It

is chosen because we already have estimates of effects of exposure to income mobility, and also, because it is relatively easy to obtain effects of smoking on chronic conditions or mortality. The latter is even easier to retrieve for other behaviors such as drug addiction, accidents, suicides, but in that, case we have no estimates of effects of income mobility.

We implemented the *smoking behavior* using a logistic model so that agents would decide whether to smoke or not at age 30. In MIA, smoking is a key behavioral mediator through which income mobility operates as we do not include any direct effect of income mobility on mortality risk. To implement this, we resort to information from previous empirical research on the link between early exposure to county's income mobility and smoking during early adulthood (Daza & Palloni, 2021). Smoking is defined at age 30 using:

$$Pr(Y_i) = \frac{e^{\alpha_s + \beta_{s_k}\text{incomeType}_i + \beta_{s_{imob}}\text{incomeMobExp}_i + \beta_{s_{ie}}\text{countyIncomeExp}_i + \beta_{s_{psmk}}\text{parentSmoker}_i}}{1 + e^{\alpha_s + \beta_{s_k}\text{incomeType}_i + \beta_{s_{imob}}\text{incomeMobExp}_i + \beta_{s_{ie}}\text{countyIncome}_i + \beta_{s_{psmk}}\text{parentSmoker}_i}} \tag{5.4}$$

$Pr(Y_i)$, the probability of being an active smoker by age 30 is a function of the agent's income group (k), income mobility exposure by age 18, standardized county's income exposure by age 30, and whether agent's parent smoked. β_{s_k} coefficients were estimated using a subset of the National Health Interview Survey 2019 (NHIS) using a logistic regression of income quintiles on smoking status.[10] The income mobility exposure coefficient $\beta_{s_{imob}}$ is obtained from the effect of early income mobility exposure on smoking reported by Daza and Palloni (2021) using the National Longitudinal Survey of Youth 97 (NLSY97).[11] Agent's parent smoking effect β_{pmsk} is derived from a meta-analysis carried out by Leonardi-Bee et al. (2011): these researchers used 58 studies and estimated the relative odds of smoking for children and adolescents whose parents smoke to be 1.72 (95% CI 1.59 to 1.86), or logit coefficient 0.54. Finally, we set the standardized county's income exposure coefficient $\beta_{s_{ie}}$ to -0.2 to represent the impact of county's characteristics

[10] We defined *current smoking* status 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 ran our model using respondents between ages 30 and 50. 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.

[11] Daza and Palloni (2021)'s coefficient comes from a logistic model, so that it can be directly added to MIA.

on the smoking probability.

The final smoking status of an agent is generated using the probability (p_{s_i}) estimated from Equation 5.4. Agents become smokers if values from a uniform distribution – `uniform(0, 1)` – are less than p_{s_i}. We assume no smoking before age 30 and no smoking cessation. Although those assumptions are unrealistic, they allow us to directly incorporate information from empirical results into our model. Empirical verification of the adequacy of these assumptions via estimation of smoking prevalence is discussed in the *Methodological Supplement*.

5.3.5 Model setup and measurement

At the outset of the simulation, each agent is assigned a set of states and transitions updated over time and to which will be exposed throughout a lifetime. Figure 5.1 displays agent's states and transitions. Most of the events in MIA are stochastic. Duration times (rates) are drawn from exponential distributions, whereas 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*.[12] The initial setup includes 30 counties and 100 agents per county. This generates a total population of around 8,000 *alive* agents. When collecting individual data from our model, we select only three generations (e.g., 20, 25, 30) to avoid excessively large datasets. These generations are created after 800 simulation years.

We track the income generation mechanisms through aggregate measures of income mobility and mortality (i.e., life expectancy). First, we use a *relative mobility* measure to compare the ranking of adults against their peers to the ranking of their parents against their peers: the rank-rank slope suggested by Chetty et al. (2014). These authors propose the following OLS model to estimate relative and absolute income mobility, under the assumption of linearity between parent and children's income ranks:

[12] 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. All relevant model parameters are updated at the time of each event. All files and code to reproduce our analysis are available at https://github.com/sdaza/dissertation/tree/master/ch04.

$$R_{ic} = \alpha_c + \beta c P_{ic} + \varepsilon_{ic} \tag{5.5}$$

R_{ic} represents 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 children. β_c represents the relationship between national parent and kid income ranks. This is not necessarily equivalent to the correlation between ranks, $Corr(P_{ic}, R_{ic})$, as ranks within a small area would not necessarily be uniformly distributed when using national income ranks as Chetty et al. (2014) do. The interpretation of the relative income mobility measure suggested by Chetty et al. (2014) for small areas (i.e., counties) is that it represents 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:

$$\hat{\beta}_c = r \frac{\sqrt{\sum (R_{ic} - \bar{R}_c)^2}}{\sqrt{\sum (P_{ic} - \bar{P}_c)^2}} \tag{5.6}$$

β_c will be higher than the 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 income segregation, the parent's rank might tend to be homogeneous (lower standard deviation), while the kid's rank might vary more than P_{ic} due the income generation probabilities used in our model (e.g., transition matrix I_b). As a robustness check, Chetty et al. (2014) estimated relative mobility based on parent and child ranks using the local income distribution instead of national ranks, and showed their different estimates were highly correlated. Our verification analyses (not shown) suggest, instead, that the difference between the rank-rank slope and the rank-rank correlation can be substantial in scenarios of income segregation. The implications of these measurement differences for assessing the relation between income mobility and health, however, are not explored here, but left for future research.

Figure 5.2 displays some descriptive statistics of rank-rank slopes estimated by Chetty et al.

(2014) in the US at the county and commuting zone level.[13] The distribution of rank-rank slopes across counties looks relatively normal, with an average of 0.26 and a standard deviation of 0.086. County rank-rank slopes are negatively associated with the county's average income ($\rho = -0.33$), and most of that negative association is driven by counties with a high proportion of African-American residents. We also have access to the income quintile transition matrices at the commuting zone level, so we can explore the association between income transition probabilities and rank-rank slope values. Figure 5.2 shows the association between the commuting zone rank-rank slopes and the probability of being in the first income quintile conditional on the parent being in the first quintile, on one hand, and between the commuting zone rank-rank slope and the probability of being in the highest quintile conditional on the parent being in the top quintile, on the other. The association in the first quintile ($\rho = 0.66$) is higher than the association in the highest quintile ($\rho = 0.11$), suggesting an important fraction of the highest rank-rank slopes observed in the data are due to the probability of staying at the bottom not at the top of the income distribution. Again, part of the association between the probability of being in the same income quintile as parents is driven by commuting zones with a high proportion of African-American residents. Overall, these descriptives provide relevant information to calibrate our model.

Aggregate mortality and income mobility information are collected by *birth* cohort (e.g., agents born between years 10 and 50 who started working or died). The size of the cohort is defined by the parameters m_{coht} and mob_{coht} in Table 5.1. Similarly to estimating a moving average, measures are calculated using a moving time window (T_w), so that cohorts have enough number of agents and values are relatively stable overtime. For instance, if the cohort 10-50 is used to estimate county's rank-rank slope and $T_w = 10$, the next cohort would be 20-60, and so on. The county associated with the rank-rank slope is determined by where the agent started working. Even if a working agent moves later to a different county, the income mobility regime remains linked to the county where the agent started working.[14] A similar approach is used when computing the life expectancy

[13]The commuting zones (CZ) identify smaller county clusters within Labor Market Areas (LMA), and are based on journey-to-work data and define clusters of counties with strong commuting ties. For more information, see Tolbert and Sizer (1996).

[14]Chetty et al. (2014) permanently assigns each child to a single county based on the ZIP code from which his

by county, but this time using the ages of death in the county where agents die. In other words, the life expectancy of the county reflects the agent's place of death as mortality data usually do. We compute the average age of death of a birth cohort (e.g., 10-50) when all agents from that cohort have died. Other cross-sectional aggregate measures such as the county's average income, NSI, or Gini coefficient are recorded every T_w years.

We also track agents' exposure to county income composition and income mobility. MIA registers the difference between $t + c$ (current time) and t (time of previous record), Δt_r, and county's income and rank-rank slope at time t, every simulation year or when agents move to a new county. By age 18, for instance, the rank-rank slope exposure is estimated using the formula in Equation 5.7. Average exposure statistics are then used as inputs in the functions outlined above.[15]

$$\frac{\sum_{i=0}^{18} \text{county's income mobility}_i}{18} \tag{5.7}$$

5.3.6 Agents attributes and relationships

To summarize the structure of our model, Figure 5.3 displays the key agents' attributes and relationships in MIA, omitting composition effects (e.g., how agents contribute to the county income average). Factors in green represent children, parents' variables are in blue, and county-level factors in orange. Black arrows are relationships always on, and red arrows can be switched off to explore scenarios.

Before age 18, children 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 income composition of the county where they live (i.e., parent's county). This is represented by the arrow between *parent's income class* and *child's income class*. Children are also exposed to the income class composition in the place where they live (parents' county) or *county's income class exposure*. These are the two key factors

parent filed their tax return in the first year the child was claimed as a dependent. They interpret this as the area where a child grew up. The county where a child grew up does not necessarily correspond to the county where he lives later or where he lives when Chetty et al., 2014 measured his income (at age 30) in 2011–2012. In their core sample, 38% of the children lived in a different county in 2012 relative to where they grew up.

[15]For computational reasons, intensive county's characteristics such as the Gini coefficient and rank-rank slope (income mobility) are computed every ten years (see parameter T_h in Table 5.1).

that define income mobility. Once the income class is assigned based on transition probabilities (I_k), we define the final income by sampling from an observed income distribution for class k (i.e, quintile). Each county's rank-rank slope is computed based on the (national) rank of children who started working in a county. Thus, income mobility of a place becomes fixed once is measured, regardless of where the kids decide to live in the future.

From age 18 on, agents can move to different counties randomly or based on their income class (i.e., segregation represented by the arrow from *kid's income class* to *kid's county of residence*). Kid's income class would increase or reduce mortality risks and the propensity to smoke by age 30. Smoking also affects mortality risks, while mortality and smoking would be influenced by county's resources (the arrow from county's income exposure to mortality and smoking). The impact of individual income mobility works only through changes in income (and not through the stress that may be related to mobility *changes*), whereas the effect estimated by Daza and Palloni (2021) is represented by the arrow from *county's income mobility exposure* to *smoking*.

5.4 Experimental design

5.4.1 Micro-simulation

To examine the population-level consequences for mortality due to changes in the income mobility regime, we estimate the absolute difference in life expectancy using the individual effect of exposure to county's rank-rank slope on smoking across different scenarios. To obtain sensible estimates of the difference in life expectancy, we calibrate our model so that the distribution of rank-rank slopes across counties and the proportion of smokers mimics the empirical data. In the case of smoking, the population prevalence should match the average population in NHIS 2019 when the individual effect of exposure to county's rank-rank slope is active.[16] In addition, we explore the absolute differences in life expectancy across three residential mobility regimes: no

[16]For more details see the *Methodological Supplement*.

residential mobility, random residential mobility, and segregation.[17]

Our first counterfactual scenario is created by setting the rank-rank slope coefficient exposure by age 18 equal to 0 ($\beta_{s_{imob}} = 0$), computing life expectancy at birth, and comparing the results with those obtained when the rank-rank slope coefficient exposure by 18 is equal to the value reported by Daza and Palloni (2021). Daza and Palloni (2021)'s coefficient comes from a logistic regression and represents the proportional change in the odds of smoking when income mobility increases by one standard deviation. Whether that coefficient is 0 or positive, we take into account its uncertainty by sampling from a normal distribution.[18] This exercise allows us to get a sense of the absolute magnitude of the effect estimated by Daza and Palloni (2021) under interventions that, for instance, target smoking behavior directly: without changing income mobility, having an efficient intervention to alter desistance behavior may well affect smoking and reducing the impact of income mobility on mortality. Another option, probably more ambitious, is to promote interventions that affect the income mobility regime itself (e.g., improve educational opportunities).

We then explore a second scenario where we compare the life expectancy when the rank-rank slope is set equal to zero – each cell of the transition matrix I_b is 0.20 – with a regime where the I_b's diagonal is set to $p_k = 0.32$, while the values outside the diagonal are weighted using empirical data ($p_{k_{\text{dist}}} =$`true`).[19] In this case, changes in life expectancy will not be only due to the rank-rank slope exposure by age 18, but also to the individual and aggregate effect of income on smoking and mortality risks. The average county's income under the scenario where the rank-rank slope is set to zero is higher than the regime in which we add some rigidity to the stratification system. Therefore, part of the differences in life expectancy would be just due to changes in income. We can estimate the fraction of life expectancy differences attributable to changes in income and the income mobility regime by simulating only changes in the transition matrix when $\beta_{s_{imob}}$ is zero, and then compare them with the differences when both the transition matrix and the rank-rank slope

[17] To avoid counties without population after several replicates, we did not set a moving decision rate $mob_r = 0$ for the *no residential mobility* scenario. Instead, we used a tiny moving decision rate, $mob_r = 0.001$.

[18] We sampled the coefficient values from a normal distribution with mean equal to 1.395 and standard deviation 0.58, and a mean equal to 0.0 and standard deviation 0.58 when $\beta_{s_{imob}} = 0$.

[19] We used the values from matrix I_b in Equation 5.2. As a result, the contiguous transition probabilities follow an empirical gradient instead of being all $(1.0 - 0.32)/4 = 0.17$.

exposure coefficient $\beta_{s_{imob}}$ change (i.e., decomposition).

Each of the scenarios discussed in this section was run 100 times for a total of 1800 replicates.

5.4.2 Exogenous income mobility and residential mobility

To assess how difficult it is to retrieve the effect of income mobility exposure on mortality, we run statistical models to estimate the association between income mobility and mortality under different residential mobility regimes, income mobility effect sizes, and data collection strategies. The county's transition matrices, in turn, are defined exogenously by sampling from transition matrices estimated by Chetty et al. (2014) at the commuting zone level.

To simplify this exercise, we created a fictitious direct effect between county's income mobility exposure at time t on agent's mortality risk. Thus, we can estimate the effect of income mobility exposure using simple statistical models from the data generated by MIA, and assess the size of the bias associated with different data collection strategies. We add the coefficient β_{m_f} to equation 5.1, and use the values 0.0, 0.3, and 0.5. Those values are then multiplied by the total exposure to the county's income mobility regime every time the mortality function is evaluated (e.g., birth date). If $\beta_{m_f} = 0.5$ and the average county's rank-rank slope exposure goes from 0.0 to 0.30, the hazard ratio would be 1.16, and the mortality risk of those with a higher rank-rank slope should be 1.16 higher than those with a rank-rank slope equal to 0.

We examine three data collection and modeling strategies. First, we analyze individual mortality data and use the average exposure variables specified in MIA, so that each agent contributes with one data record. We then estimate Cox models regressing age of death on total county's income mobility exposure, total county's income exposure, and individual income (natural logarithm). We expect these models to produce the best β_{m_f} estimates using individual data.[20] Second, we analyze records similar to the cross-sectional data used by Venkataramani et al. (2016) to study the association between income mobility and health. In Venkataramani et al. (2016), place of residence is measured at the time of the survey. In our case, we record the county's characteristics

[20] Our models do not include any time-varying covariates.

of the place where agents die, and use a Cox model to regress county's income mobility, county's income, individual income (natural logarithm) on the age of death. Finally, we produce aggregate data to mimic the analyses by Venkataramani et al. (2015) and Daza and Palloni (2018). This time, we estimate a linear model regressing county's income mobility, county's income (natural logarithm), and county's population (natural logarithm) on county's life expectancy.

Again, we ran MIA 100 times for each scenario thus producing a total of 900 replicates. The results of the models were pooled using meta-analysis techniques: we estimated our models for each replicate, collected the estimates and standard errors, and combined them using random-effects models to allow the effects to vary across replicates (Viechtbauer, 2010).[21]

5.4.3 Endogenous income mobility and residential mobility

Our final exercise consists of exploring the association between income mobility and mortality at the individual and aggregate level when no exogenous effect of income mobility on health has been introduced (i.e., no arrow from kid county's income mobility exposure to smoking in Figure 5.3), and the income mobility regime is defined endogenously.

First, we create an association between county's income mobility (rank-rank slope) and income as shown in Figure 5.2. To produce this association, we add heterogeneity to the definition of agents' transition matrix by using different values in vector W_k (Equation 5.2). The vector W_k allows us to make the agents' transition matrix endogenous depending on the income composition of counties where agents have lived by age 18. In particular, we employ values so that low income groups are more susceptible to the county's income composition than better-off groups: $W_k = [0.8, 0.7, 0.3, 0.1, 0.05]$. In other words, the first quintile weight would be $w_1 = 0.80$, so that the income group composition at county c when defining the income transition matrix would weight 0.80. For the highest quintile, instead, the baseline transition probabilities would be much more important than the county's income group composition ($1 - w_5 = 0.95$). This implementation looks plausible as better-off individuals have more resources (economic, social, and cultural)

[21] Using fixed-effects models provide almost identical results. The meta-analysis models we estimate assume that the weights of the coefficients (i.e., standard errors) are known (Viechtbauer, 2010).

to face the disadvantages of the places where they live. We expect, therefore, poorer counties to have a higher rank-rank slope (less income mobility) than richer counties due to both the weight of county's income composition and segregation. The baseline probability for the diagonal of the transition matrix's diagonal is $p_k = 0.30$, while the values outside the diagonal follows an empirical distribution ($p_{k_{\text{dist}}}$ =`true`). Finally, using the three data collection and modeling strategies discussed in the previous section, we check whether or not we are able to retrieve the *no effect* of income mobility and mortality when using individual and aggregate models that adjust for income exposure, county's current income, and individual income. We ran each scenario 100 times, created 300 replicates, and pooled the results through meta-analysis techniques.

5.5 Results

5.5.1 Life expectancy and the effect income mobility exposure on smoking

We first plot the absolute difference in life expectancy due to the individual effect of exposure to county's rank-rank slope on smoking behavior. Figure 5.4 shows the absolute life expectancy differences by residential mobility scenario (no residential mobility, random residential mobility, and segregation) when the individual effect of exposure to county's rank-rank slope on smoking $\beta_{s_{imob}}$ is equal to the value estimated by Daza and Palloni (2021), and the coefficient is set to zero.

The average absolute life expectancy difference associated with the exposure to county's rank-rank slope goes from -0.72 to -0.64 years. Most of the estimated differences are negative (decrease in life expectancy), although we observe more variability when agents can move between counties. While the scenario without residential mobility produces negative values in 99% of the replicates, random mobility and segregation produce negative values in 97% and 95% of replicates, respectively. Figure 5.4 also shows the average county's rank-rank slope (and standard deviation), the NSI, and the proportion of smokers when the income mobility effect on smoking is positive. The rank-rank slope values are close to the average observed in the US (0.26, SE = 0.9 in the US; 0.30, SE = 0.09 in our simulation). As expected, the NSI is higher when agents decide where to live

based on the county's income (0.38) or when there is no residential mobility (0.17), and lower when residential mobility is completely random (0.07). The proportion of smokers in the population reaches about 16% when the income mobility effect is active, very close to the NHIS 2019 estimate (17%).

We created plots similar to Figure 5.4, only this time comparing the first and last income quintile. Previous research using county level data suggests that the association of income mobility with life expectancy generally declined by income quartile (Venkataramani, Daza, & Ezekiel, 2020). Figure 5.5 shows the life expectancy differences for quintiles 1 and 5 by residential mobility scenario. Although the variability in the distributions is higher than what we see in Figure 5.4, the plots show that life expectancy differences in the first quintile are lower than the ones in the last quintile (about 0.8 years). In addition, about 90% of the values in the first quintile are negative (decrease in life expectancy) versus 60% in the highest quintile. These results suggest that the heterogeneity of the effects of income mobility on mortality suggested in previous research could be mostly due to differences in the adoption of healthy behaviors (e.g., smoking rates), and not necessarily to different effects of income mobility on behavior by income group.

We also simulated the consequences of changes in the income transition matrix rather than the individual effect of exposure to county's rank-rank slope. First, we set the rank-rank slope to zero by setting each cell of the transition matrix I_b equal to 0.20. We compare life expectancy from that regime with another in which the diagonal of the transition matrix I_b is set to 0.32. Figure 5.6 shows absolute life expectancy differences by residential mobility scenario. In this case, the average life expectancy difference goes from -0.87 with no residential mobility to -0.82 with segregation. The values of the distributions are mostly negative for all residential mobility scenarios. As we discussed above, the changes we observe in life expectancy are not only due to the rank-rank slope exposure by age 18, but also to the effects of individual and county's income exposure both on smoking and mortality risks. We estimate the fraction attributable to changes in income only by comparing changes in life expectancy by transition matrix with and without the effect of the rank-rank exposure on smoking. Based on that estimation, about 17% of the changes

we observed in life expectancy are due to changes in income only. If we subtract that fraction from the raw estimates in Figure 5.6, we get a life expectancy decrease of about 0.69 years, similar to the estimates in Figure 5.4 (on average 0.67 years). The differences by income quintile (not shown) follow the same pattern described in Figure 5.5: The lowest income quintile has higher decreases in life expectancy than the highest quintile.

5.5.2 Retrieving exogenous income mobility effect on mortality

We now explore how difficult it is to retrieve the effect of the county's income mobility exposure on mortality risk using different data collection strategies when the income mobility regime is exogenous. Table 5.2 displays income mobility effect estimates on mortality across different residential mobility scenarios, β_{m_f} values, data collection and modeling strategies.

The first set of models, *individual mortality & total income mobility exposure*, should reproduce β_{m_f} the coefficients specified in MIA as they include the best measures of total county's income and income mobility (rank-rank slope) exposure at the individual level. When $\beta_{m_f} = 0$, there is no direct or indirect effect of county's income mobility on smoking or mortality, besides the effect of county's and individual income which are controlled for in the statistical models. *Individual mortality & total income mobility exposure* coefficients in all residential mobility scenarios are close to zero and imprecise enough to be distinguished from zero, except for the scenario without residential mobility in which the coefficient has a 95% confidence interval of $[0.03 - 0.09]$. When $\beta_{m_f} = 0.30$ and $\beta_{m_f} = 0.50$, the estimated coefficients across mobility scenarios and replicates are also consistent with the fictitious values used as parameters in MIA. The *individual mortality & total income mobility exposure* models show coefficients close to 0.30 and 0.50, respectively, and are precise enough to be considered different from zero. Thus, our baseline measurement and model specification is able, in most of the cases, to recover MIA's β_{m_f} parameter.

When we replace the exposure variables by county level measures of income and income mobility – *individual mortality & county income mobility* in Table 5.2 – the estimates of the association between income mobility and individual mortality risk show more variability across movement

scenarios. We observe that the best β_{m_f} estimation occurs when there is no residential mobility, with point estimates ranging from 0.10 to 0.36. Except for $\beta_{m_f} = 0$, using county measures tends to bias estimates downward as county measures are only partially correlated with the *actual* exposure because of changes in the county composition and cohort fluctuations. This pattern becomes evident in the case of *random residential mobility* where breaking the link between exposure and county's characteristics at the age of death through a random selection of counties, makes all coefficients practically equal to zero, regardless of β_{m_f}'s values. Intermediate results are observed in the case of segregation: While the coefficients are considerably smaller than the β_{m_f}'s values (i.e., 0.30 and 0.50), they are still positive and precise, ranging from 0.11 and 0.20.

We also explore the association between county's income mobility and life expectancy using aggregate data, mimicking the analysis produced in previous research (Daza & Palloni, 2018; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020). First, the association between county's life expectancy and income mobility (rank-rank slope) is always negative with confidence intervals far from zero. The highest coefficients are observed when there is no residential mobility. For instance, based on our statistical models adjusting for county's income and population size, moving from a county whose rank-rank slope equals to 0 to one in which it takes the value 0.26 reduces the life expectancy by about 0.30 years when $\beta_{m_f} = 0.0$, and 1.09 years when $\beta_{m_f} = 0.5$. The aggregate coefficients under random residential mobility or segregation are considerably smaller, but still negative. While the estimate is -0.67 when $\beta_{m_f} = 0.5$ and there is random residential mobility, it decreases to -1.95 with segregation. In other words, residential movement seems to bias estimates downward as the correlation between individual income mobility exposure and county's income mobility weakens when agents are able to decide in which county to live. Although the coefficients from the *county life expectancy & income mobility*'s models in Table 5.2 when $\beta_{m_f} = 0.0$ are small – an increase in the county's rank-rank slope from 0 to 0.26 would decrease life expectancy by 0.06 years when there is segregation, and 0.31 years when there is no residential movement – they suggest aggregate empirical estimates will show a negative association between income mobility and mortality when no individual effect is present (i.e., ecological fallacy).

5.5.3 Retrieving endogenous income mobility effect on mortality

Finally, we examine how heterogeneity in the definition of the income transition matrix could generate an artificial association between income mobility and mortality, even though there is no direct effect of county's income mobility exposure on smoking or mortality risk. We add heterogeneity in the definition agents' transition matrix by setting different values of the vector W_k (equation 5.2) so that agents define their income transition matrix endogenously based on the income composition of the counties where they live by age 18. Table 5.3 shows the estimates for three different W_k scenarios: (1) all the values in the W_k vector are 0.0, e.g. there is no impact of the county's income composition on the income transition matrix; (2) all the values in W_k are 0.5 to create an income transition matrix equal to the average between the baseline transition probabilities and the income composition of the places where an agent lived before 18 years old; and (3), $W_k = [0.8, 0.7, 0.3, 0.1, 0.05]$ to make the poorest agents more susceptible to county's income composition than the richest ones. Again, we ran three types of models using the same data collection strategies described previously: *individual mortality & total income mobility exposure*, *individual mortality & county income mobility*, and *county's life expectancy & income mobility*. In all these scenarios, the baseline probability of the transition matrix diagonal p_k is equal to 0.30, while the values outside the diagonal follow an empirical distribution ($p_{k_{\text{dist}}} =$`true`).

The baseline scenario simulates a world where the transition matrix for each agent is the same, and there is no influence on the county's income composition ($w_k = 0$). This generates an average county's rank-rank slope of 0.28 (SE=0.08), and NSI of 0.35.[22] When estimating the coefficients of the association between income mobility and mortality, we see that most of the coefficients are negative but very imprecise to be distinguished from zero. The only confidence interval that does not include zero in the first column ($w_k = 0$) is negative and quite small, even though we expect a positive relationship between rank-rank slope and mortality risk. These baseline results are expected as the income transition matrix is homogeneous across agents, and no direct link

[22] See the *Methodological Supplement* for more details on how the rank-rank slope behaves using different transition matrices.

between the county's rank-rank slope and smoking or mortality has been included.

The second scenario sets $w_k = 0.5$ so that the transition matrix now is influenced by the income composition of counties. With segregation, this would create more variability in the rank-rank slope across counties. The average county's rank-rank slope decreases to 0.15 (SE=0.08) and becomes only slightly more variable than the baseline. The NSI, instead, increases to 0.41. Again, the coefficients of the second column of Table 5.3 ($w_k = 0.5$) are very imprecise and not different from zero. Thus, even in the presence of income segregation, a homogeneous influence of county's income composition would not generate a spurious relationship between income mobility and mortality.

In the last scenario, w_k is heterogeneous and related to the income group of agent's parents. The average county's rank-rank slope now is 0.14 (SE=0.13) and the NSI is 0.41. The last column of Table 5.3 shows that the coefficients of the exposure and county's rank-rank slopes are both positive, albeit very small. When using aggregate data, the association between county's income mobility and life expectancy is -1.54, a value not far from the county level estimated when using exogenous transition matrices, $\beta_{m_f} = 0.5$, and under segregation (-1.95, see Table 5.2). Thus, an increase of the county's rank-rank slope from 0.0 to 0.26 would decrease the county's life expectancy by 0.40 years, even after adjusting for county's income.[23]

5.6 Conclusions and discussion

Concerning the paper's first objective, our *microsimulation* results suggest the relative effect of the exposure to county's income mobility (rank-rank slope) during childhood and adolescence on smoking behavior during early adulthood estimated by Daza and Palloni (2021), can generate significant differences in life expectancy at the population level. Assuming that the only mechanism through which income mobility might impact health and mortality is smoking behaviors, growing up in a society with almost complete income mobility instead of in one with income mobility

[23] Adjusting for the county's average age does not change these results.

levels similar to the US reduces life expectancy by 0.67 years[24]. These estimates are robust to different residential mobility scenarios implemented in our model and counterfactual definitions (e.g., change in transition probabilities versus change in the effect – coefficient – of income mobility on smoking). In all cases they are associated with considerable levels of uncertainty. It is important to note that these estimates depend on how we implement our agent-based model (i.e., parameters) and whether the coefficients estimated by Daza and Palloni (2021) are approximately correct. Although Daza and Palloni (2021) suggest the connection between income mobility and health is not as systematic as previous research shows, it is not unreasonable to think that the effect of income mobility on health behavior is as large or larger than the one reported by these authors. That can be the case if we consider, in addition to smoking, other paths connecting income mobility and health and health behavior (e.g., deaths of despair such as suicide, drug overdose, and alcoholic liver disease (Case & Deaton, 2020)). If estimates of the effects of mobility on smoking behaviors suggested by Daza and Palloni (2021) are not upwardly biased, then the estimated impact of income mobility on mortality obtained here should be considered lower bound.

Our microsimulation also shows that heterogeneity of estimated effects of income mobility on mortality reported in previous research (Venkataramani, Daza, & Ezekiel, 2020) can be generated using the observed prevalence of smoking by income quintiles, without varying the effect of income mobility on smoking by income class. In the real world, however, it is likely that both different prevalence of unhealthy behaviors and heterogeneity of effects by income are present, thus reinforcing the differences we observe at the population level in our model.

Regarding our second goal, we examine how difficult it is to retrieve the *effects* of income mobility on mortality under different residential segregation scenarios, data collection, and modeling strategies. When income mobility is defined exogenously by sampling observed income transition matrices (Chetty et al., 2014) instead of using the actual county's exposure variables (i.e., measurement error) we are likely to bias downward the association between income mobility and mortality

[24]Note that to maintain comparability with previous studies, we use life expectancy at birth rather than at adult ages. We are cognizant that the consequences of smoking are only discernible at adult ages so that the years of life lost due to low income mobility we estimate here ought to be compared with those that are expected to be lived in ages, say, 40 or 50.

in cases when we use individual data and downwards *in those when we use county-level data*. These biases appear across all residential mobility scenarios we explore, including the case of constant county's transition matrices. Although the biases are small, the simulation suggests that the use of aggregate data leads to a negative correlation between income mobility and mortality even when no individual effects are present.

Estimation of income mobility effects seems to be worse when we add heterogeneity in the definition of income transition matrices by income group. Even with an optimal study design, e.g., using exposure variables and individual data, we observe some albeit small, bias. Using aggregate data leads to the most misleading estimates as they suggest a strong relationship between income mobility and mortality when no direct link between those variables exists at the individual level. These results suggest we should avoid entirely models in which the exposure is not well defined and measured, even when the mobility regimes we study are invariant across the spatial units that define exposure (e.g., counties). There is no doubt that previous studies using aggregate data have contributed a considerable amount to this research area (Daza & Palloni, 2018; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020). However, our simulation model suggests that the empirical findings highlighted in these studies could be biased. Using individual and longitudinal data would help us measure exposure more precisely and could also help to attenuate the impact of selection and to account for the effects of time-varying confounding. To what extend could these *ideal* studies confirm or not previous aggregate studies is an empirical question.

Furthermore, our results suggest we should consider in a systematic way the impact of heterogeneity. One way to think about heterogeneity is as analogous to unobserved confounding and selection bias when using regression models (Elwert & Winship, 2010). In this particular case, ignoring heterogeneity in the income generation process, could prone to failure when studying the association of income mobility and health by just running regression models with the hope that main-effects-only models can recover the desired quantities.

This is just a first attempt to formalize ideas regarding the connection between income mobility and health. As such, our study has a number of shortcomings that could be addressed in

future research. First, we use a very narrow definition of the mechanisms might trigger differences in health (i.e., contextual income mobility or county's rank-rank slope). However, family income mobility might also impact individuals. For instance, the parent's mobility experience itself (whether they experienced mobility and how significant was the shift) before children turn 18 or when they leave the parental home, could be a more direct (and easy to measure) factor that changes the kid's expectations and adoption of healthy behaviors. Similarly, the average exposure of parents to regimes of mobility depending on the counties they lived in before a child is born might be critical in shaping future expectations and parenting styles that may ultimately affect the kid's adoption of health-impairing behaviors. Unfortunately, we do not have empirical estimates of those effects. However, those mechanisms can be easily added to MIA and used to design empirical studies with individual and longitudinal data. Second, we were able to explore only a limited number of scenarios and possible parameter values. To understand better the behavior of our model, we should carry out more extensive sensitivity analyses with the capability to assess the impact of key parameters on the outputs of our model. Third, we could explore which sources of heterogeneity are critical when trying to estimate the relation of interest, and identify clear analytical tools to retrieve those effects. Finally, MIA could be useful to explore plausible counterfactual and intervention scenarios. Although statistical models can provide insights regarding counterfactual scenarios, a model like MIA offers a more direct framework to incorporate concrete interventions (e.g., smoking treatment, educational reform), and assess both their individual and population-level consequences

Our paper is the first to provide a formal framework to study income mobility, health, and mortality. This allow us not only to assess the population-level consequences of empirical estimates, or under which conditions we can retrieve the *effects* or quantities of interest, but also can help us design new studies and incorporate and explore new mechanisms, in the hopes of more fully accounting for phenomena where individual, contextual, spatial, reciprocal, cumulative effects, and feedback loops come all and simultaneously into play.

5.7 Tables

Table 5.1: MIA Parameters

Parameter	Anylogic Name	Symbol	Baseline value	Description
Mortality				
Mortality rate	`f_baselineMortalityRate`	m_x	See Table 5.4	Age-specific mortality rates per year.
Income type	`p_mortalityCoeffIncType`	β_{m_k}	$[0.1, 0.05, 0.0, -0.05, -0.1]$	Coefficients of income group k on mortality risk.
Smoking status	`p_mortalityCoeffSmoking`	$\beta_{m_{smk}}$	1.030 (SE = 0.05)	Coefficient of the effect of smoking on the mortality risk for all causes of death among men: Hazard Ratio 2.8 (99% CI = 2.4 to 3.1) (Jha et al., 2013, Table 2, pg. 346). The coefficient comes from male models adjusting for age, educational level, alcohol consumption, and body mass index. The standard error was computed using the formula: `(log(3.1) - log(2.4))/(qnorm(0.995)*2)` in the software R.
Income exposure	`p_mortalityCoeffIncExpZ`	$\beta_{m_{ie}}$	-0.1	Coefficient of the effect of standardized county's income exposure on mortality risk.
Income mobility exposure	`p_mortalityFakeCoeffRankSlope`	β_{m_f}	0.0	Fictitious coefficient of the effect of county's income mobility exposure on mortality risks.
Fertility				
Fertility rate	`f_baselineFertilityRate`	f_x	See Table 5.4	Age-specific fertility rates per year.
Fertility adjustment factor	`p_fertilityAdjustment`	f_{adj}	1.69	Age-specific fertility rates per year are divided by f_{adj} to emulate a stationary population (zero growth rate).
Income group fertility adjustment	`p_fertilityCoeffIncType`	$f_{i_{\text{adj}}}$	$[0.3, -0.10, -0.10, -0.10, -0.10]$	Fertility income group adjustment to avoid unbalance of income groups' population.
Residential mobility				
Moving decision rate	`p_movingDecisionRate`	mob_r	0.10	Average number of 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}	0.01	Probability agents move to a random county that has not reached its population limit.

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Parameter	Anylogic Name	Symbol	Baseline value	Description
Moving threshold	`p_movingThreshold`	mob_{thr}	0.22	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}	1.10	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 multiplied by 1.10.
Income				
Income type	`p_incomeType`	k	1-5	Agents' income quintile.
Income	`p_income`	$income_k$	0 – 1,953,700	Agents' income. It is defined by sampling from the IPUMS micro-data family income distribution by quintile (Ruggles et al., 2020).
Parent income type	`p_parentIncomeType`	$parent_k$	1-5	Parent's income quintile.
Parent's income	`p_parentIncome`	$pincome_k$	0 – 1,953,700	Parent's agent income.
County's transition matrix	`v_incomeTransitionMatrix`	I_b	Matrix	Transition matrix used to assign income type to agent i at age 18 and county c. When income mobility is exogenous, the transition matrix comes from a sample of commuting zone transition matrices (Chetty et al., 2014).
Relative importance of county's income exposure	`p_weightVectorCountyIncExp`	W_k	$[0.0, 0.0, 0.0, 0.0, 0.0]$	Weight to combine the transition matrix I_b and vector C_k (see main text for details). When $w_k = 0.5$, the estimate is equivalent to the arithmetic mean.
Endogenous income mobility	`p_endogenousIncomeMob`	endg	`false`	Whether individual transition matrices are defined exogenously (by sampling from observed transition matrices) or endogenously by the parameters and dynamic of the model.
Baseline probability in the diagonal of the transition matrix	`p_baselineSameIncomeProb`	p_k	0.20	When the individual transition matrices are endogenous, the diagonal of the transition matrix is equal to p_k.
Empirical distribution of out-of-diagonal transition probabilities	`p_empiricalDistributionTransMob`	$p_{k_{\text{dist}}}$	`false`	When the individual transition matrices are endogenous, the out-of-diagonal values follow an empirical distribution (e.g., equation 5.2), not the default value $(1-p_k)/(k-1)$.
Smoking				

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Parameter	Anylogic Name	Symbol	Baseline value	Description
Income type	`p_smokingCoeffIncType`	β_{s_k}	$[-0.91, -1.25, -1.69, -2.10, -2.86]$	Coefficients of income group on smoking. These coefficients where estimated using the National Health Interview Survey 2019 (NHIS). When running micro-simulations the values used were $[-1.27, -1.65, -2.13, -2.51, -3.29]$.
Income mobility	`p_smokingCoeffRankSlope`	$\beta_{s_{imob}}$	1.395 (SE = 0.58)	Defined by dividing the z-score coefficient reported in Daza and Palloni (2021) (Table 1, adjusted models) by the standard deviation of relative income mobility (rank-rank slope) across counties (SD = 0.086): `0.12 / 0.086 = 1.395`.
Income exposure	`p_smokingCoeffIncExpZ`	$\beta_{s_{ie}}$	-0.2	Coefficient of the effect of standardized county's income exposure on smoking status.
Parent smoking status	`p_smokingCoeffSmkParent`	$\beta_{s_{psmk}}$	0.54 (SE = 0.04)	Agent's parent smoking coefficient comes from Leonardi-Bee et al., 2011's meta-analysis: Odds ratio = 1.72 (95% CI 1.59 to 1.86). The standard error was computed using: `(log(1.86) - log(1.59))/(qnorm(0.975)*2)`.
Other				
Population per county	`p_peoplePerCounty`	pop	100	Initial number of agents per county
Number of counties	`p_numberCounties`	cty	30	Total number of counties.
Last complete generation	`p_lastGeneration`	G	30	Last generation before stopping the simulation.
Mortality cohort size	`p_mortalityCohortSize`	m_{coht}	40	Number of years used to define the cohort in which life expectancy is computed by county.
Mobility cohort size	`p_mobilityCohortSize`	mob_{coht}	60	Number of years used to define the cohort in which income mobility indicators are computed.
Recurrent time of cohort measurements	`p_measurementCohortWindow`	T_w	10	Years between mortality and income mobility measurements.
Recurrent time of heavy computations	`p_recurrentTimeHeavyComp`	T_h	10	Years between computationally heavy measurements (.e.g, Gini coefficient).

SD = Standard deviation; SE = Standard error.

Table 5.2: Estimates income mobility (IM) effect on mortality

	$\beta_{m_f} = 0.0$	$\beta_{m_f} = 0.3$	$\beta_{m_f} = 0.5$
No residential mobility			
Individual mortality & Total IM exposure (Cox)	0.06^* $[0.03; 0.09]$	0.30^* $[0.28; 0.33]$	0.51^* $[0.48; 0.54]$
Individual mortality & County IM (Cox)	0.10^* $[0.07; 0.14]$	0.24^* $[0.21; 0.28]$	0.36^* $[0.32; 0.39]$
County LE & IM (GLM)	-1.19^* $[-1.35; -1.03]$	-2.96^* $[-3.14; -2.78]$	-4.19^* $[-4.39; -3.99]$
Random residential mobility			
Individual mortality & Total IM exposure (Cox)	0.03 $[-0.04; 0.10]$	0.32^* $[0.25; 0.39]$	0.53^* $[0.47; 0.60]$
Individual mortality & County IM (Cox)	-0.01 $[-0.04; 0.02]$	0.01 $[-0.03; 0.04]$	0.02 $[-0.01; 0.05]$
County LE & IM (GLM)	-0.26^* $[-0.38; -0.15]$	-0.42^* $[-0.56; -0.28]$	-0.67^* $[-0.80; -0.54]$
Segregation			
Individual mortality & Total IM exposure (Cox)	0.02 $[-0.01; 0.06]$	0.30^* $[0.26; 0.33]$	0.49^* $[0.46; 0.53]$
Individual mortality & County IM (Cox)	0.03 $[-0.01; 0.06]$	0.11^* $[0.08; 0.14]$	0.20^* $[0.17; 0.23]$
County LE & IM (GLM)	-0.23^* $[-0.42; -0.04]$	-1.34^* $[-1.54; -1.14]$	-1.95^* $[-2.15; -1.75]$
Replicates per scenario (9)	100	100	100

* Null hypothesis value outside the 95% confidence interval. Each coefficient comes from random-effects model pooling replicate estimates. The total number of replicates is 900, 100 for each β_{m_f} value and residential mobility scenario.
GLM = generalized linear model. LE = Life expectancy.

Table 5.3: Mortality on income mobility (IM) with $\beta_{m_f} = 0.0$ by w_k values

	$w_k = 0.0$	$w_k = 0.5$	w_k
Individual mortality & Total IM exposure (Cox)	-0.06^* $[-0.11; -0.01]$	0.05 $[-0.01; 0.10]$	0.15^* $[0.11; 0.19]$
Individual Mortality & County IM (Cox)	-0.01 $[-0.05; 0.03]$	0.01 $[-0.04; 0.06]$	0.06^* $[0.02; 0.09]$
County LE & IM (GLM)	-0.17 $[-0.35; 0.01]$	0.13 $[-0.03; 0.30]$	-1.54^* $[-1.70; -1.38]$
Replicates per scenario (3)	100	100	100

* Null hypothesis value outside the 95% confidence interval. Each coefficient comes from a random-effects model pooling replicate estimates. The total number of replicates is 300, 100 for each w_k scenario.
The W_k vector for the last scenario is $[0.8, 0.7, 0.3, 0.1, 0.05]$.
GLM = generalized linear model. LE = Life expectancy.

5.8 Figures

Figure 5.1: Person State Chart

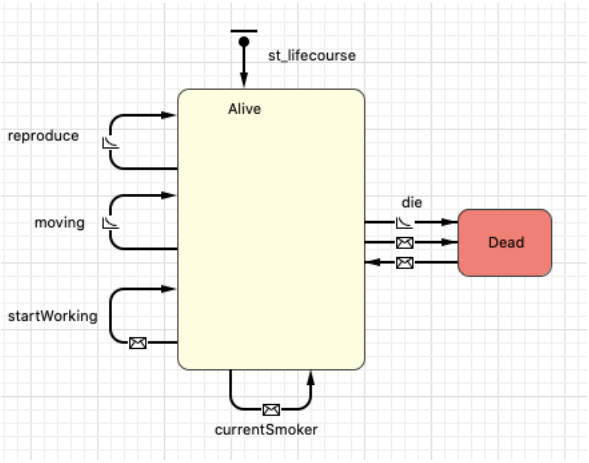


Figure 5.2: Chetty et al., 2014's rank-rank slope descriptives
CZ = Commuting zones

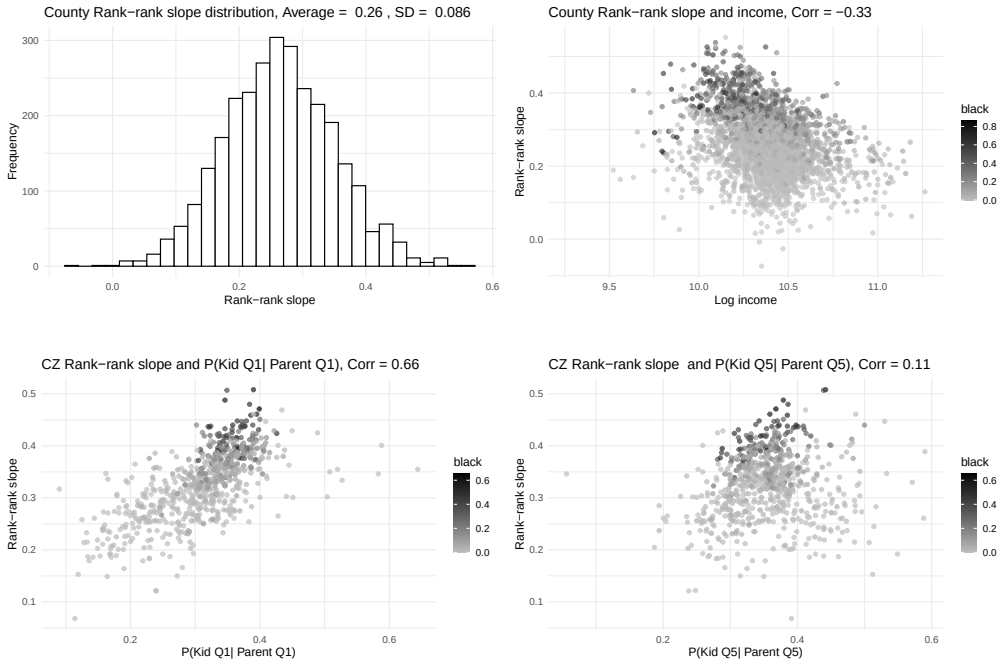


Figure 5.3: Agent's attributes and relationships

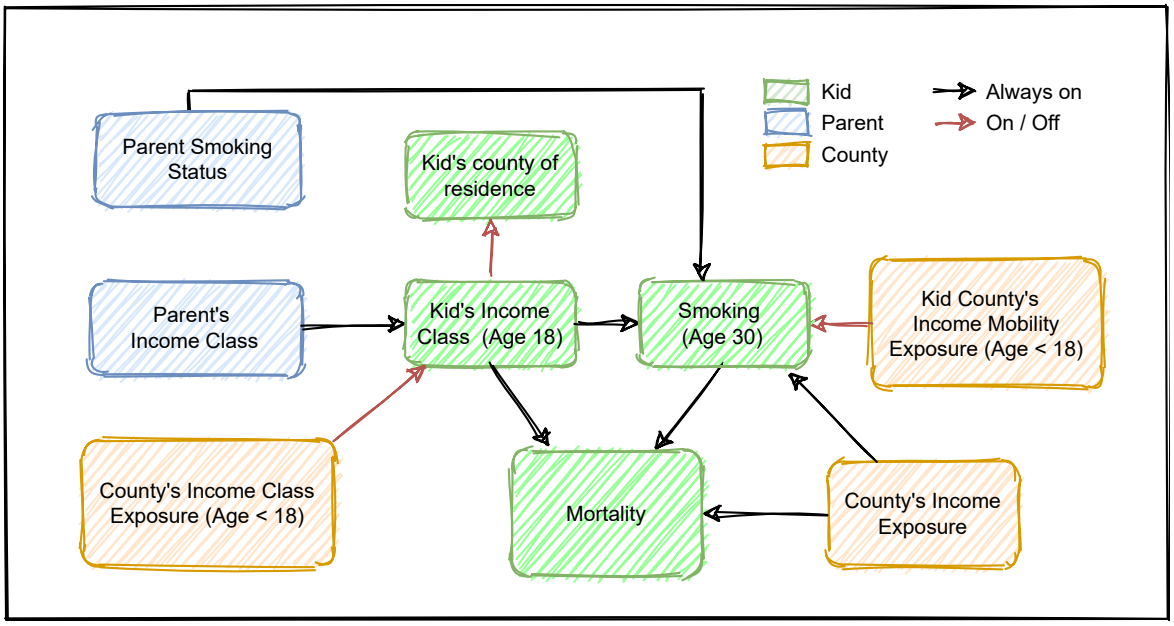


Figure 5.4: Micro-simulated life expectancy (LE) differences for the rank-rank slope effect on smoking

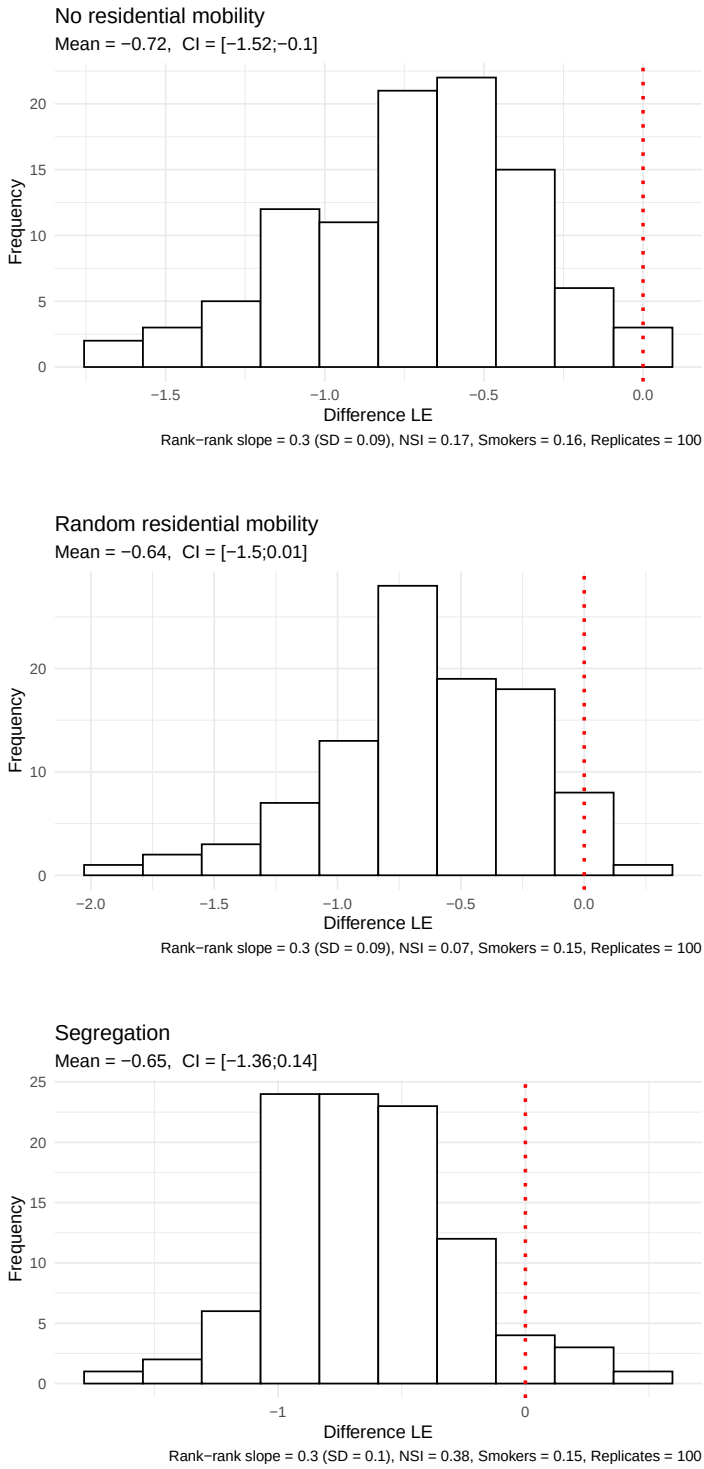


Figure 5.5: Micro-simulated life expectancy (LE) differences for the rank-rank slope effect on smoking by income Q1 and Q5

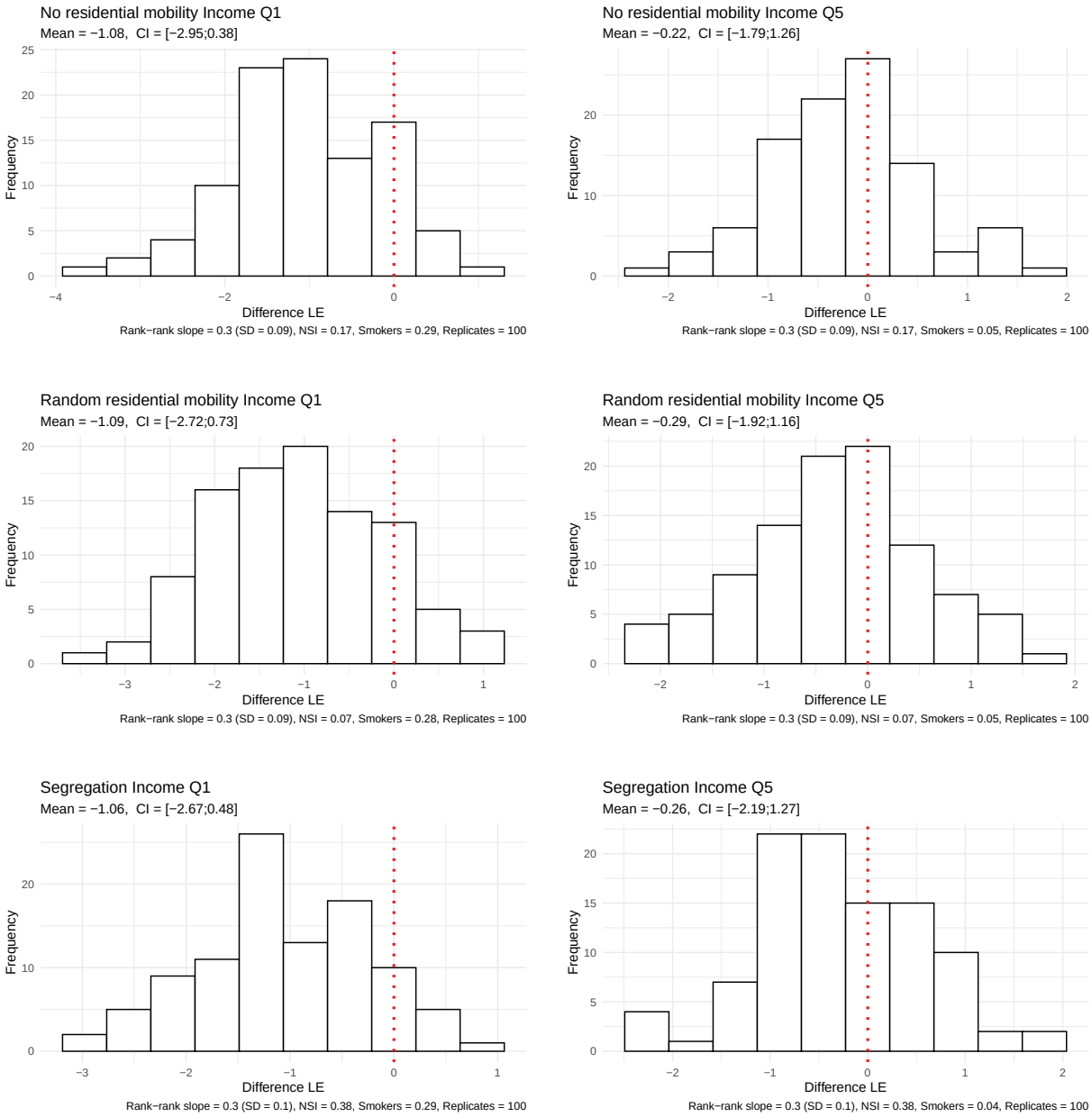
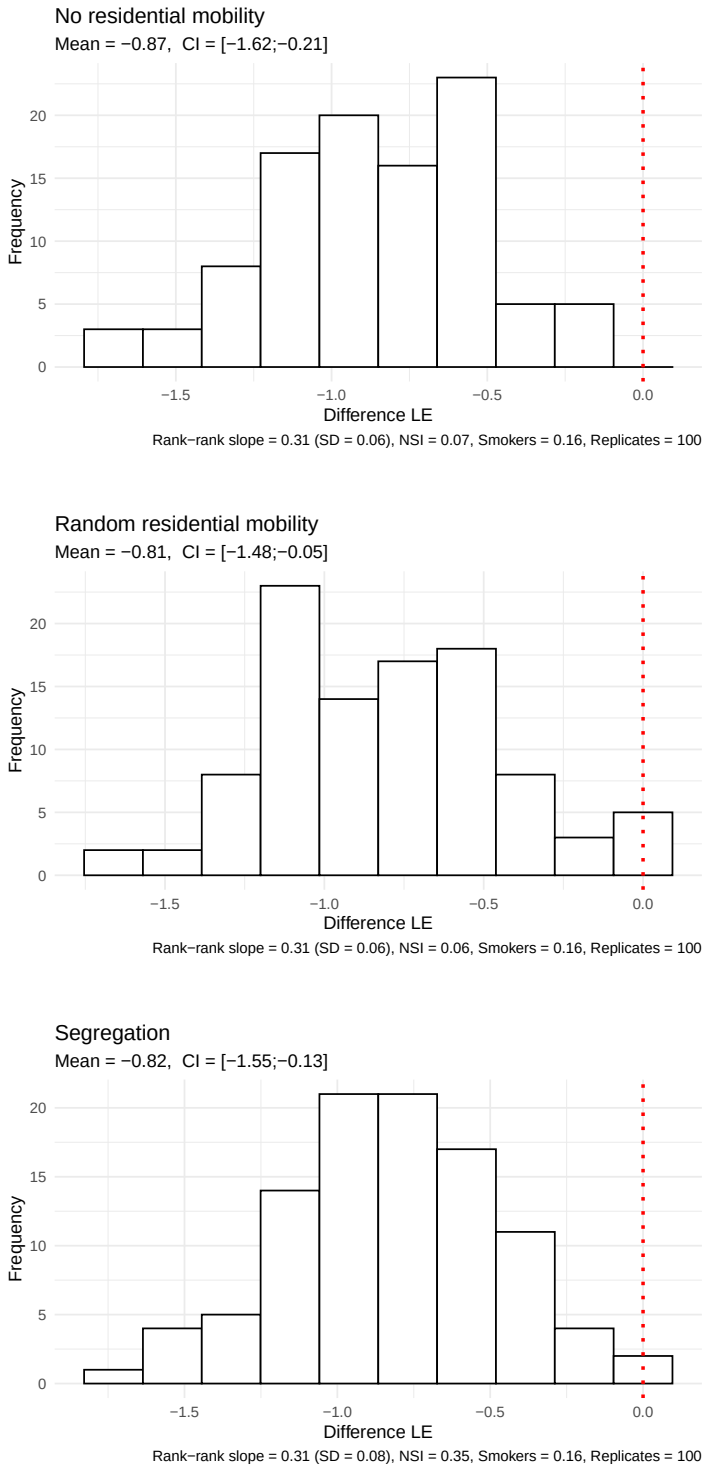


Figure 5.6: Micro-simulated life expectancy (LE) differences for the rank-rank slope effect on smoking using transition matrices as counterfactual



Methodological Supplement

The code to reproduce our models, analyses, and plots is available at https://github.com/sdaza/dissertation/tree/master/ch04. In this supplement, we show and discuss the verification and calibration of MIA's modules to ensure our model implementation is correct. For this purpose, MIA's general setup is: (a) 30 counties with an initial population of 100 agents, (b) five income groups (quintiles), (c) a population limit per county of 10% the expected population at time t, (d) a decision moving rate of $mob_r = 0.10$, (e) 30 replicates per scenario, and (f) 30 as the the last complete generation before finishing the simulation.

Demographic dynamics

To examine MIA's demographic behavior, we collected individual information for generations 20, 25, and 30, over 30 replicates. Figure 5.7 summarizes the key demographic processes implemented in MIA using US mortality and fertility rates as baselines (Table 5.4). We adjusted fertility rates to create a relatively stable population and keep the size of income groups relatively even (e.g, the lowest income group has a higher mortality rate, but also a higher fertility rate relative to the highest income group). As expected, the average number of offsprings per agent is practically 1. The distribution of age of death follows the shape expected given the age-specific mortality rates in the US, with an upper cut-off at 110 years (all agents must die by age 110). There are picks of mortality at the beginning of each 5-year age interval. This is expected as the timing is sampled from an exponential distribution when an agent enters an age group. Simulated life expectancy for complete cohorts concentrates around 76.3 years, close to 78.6, the life expectancy estimated by Kochanek et al. (2019) for the US (2017). The life expectancy values are not the same as the ones estimated by Kochanek et al. (2019) because the baseline mortality rate is not adjusted by income and the exposure to the county's conditions or income-specific effects that modify the mortality risk in groups of agents.[25] The population sizes are relatively stable and uniform overtime, although

[25] When all income effects on mortality and smoking are removed, the estimated life expectancy is 78.4, practically the same as the life expectancy estimation reported by Kochanek et al. (2019).

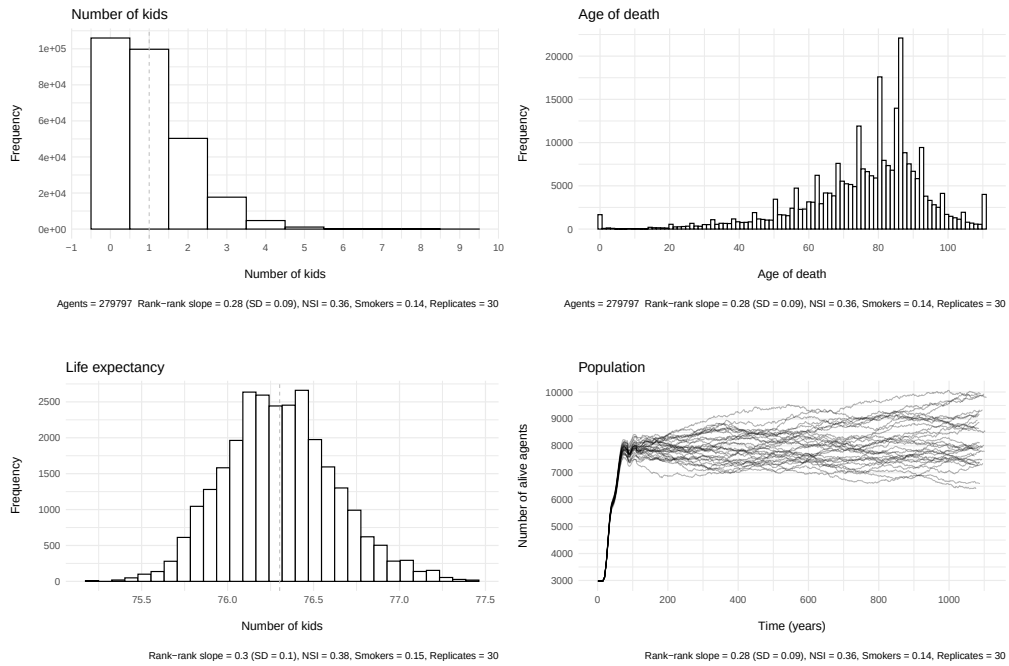
the total number of agents fluctuates considerably by replicate. The differences in life expectancy by income group are in the same order of magnitude as the differences reported by Chetty et al. (2016): around seven years between the highest and lowest quintile when income-specific effects on smoking and mortality are active.

Table 5.4: 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).

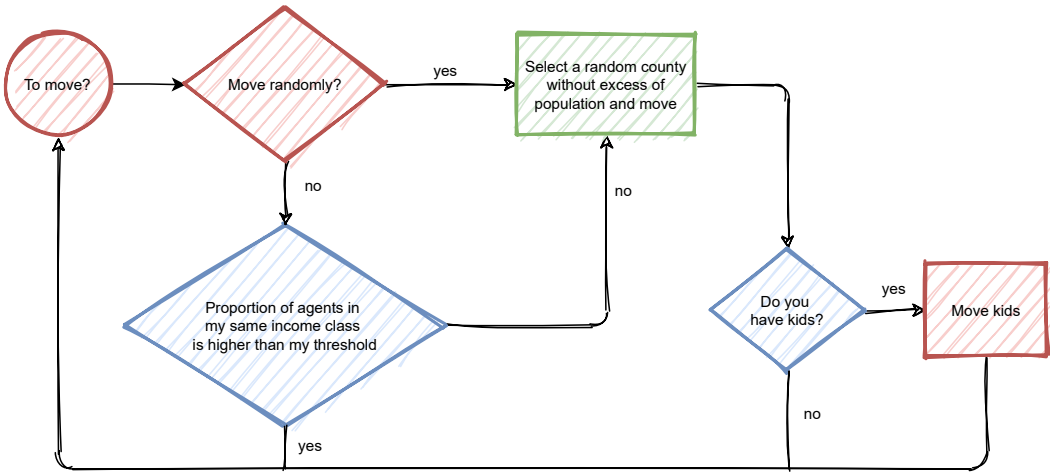
Figure 5.7: Population Dynamics (30 replicates)



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 decide to move to new county either randomly or based on the proportion of people with the same income group living in a county and similarity tolerance threshold mob_{thr}. Suppose the proportion of people in the income group k is lower than the tolerance threshold mob_{thr}. In that case, agents would move to a random county, excluding those that have reached their population limit so that to avoid an extreme concentration of agents in only some counties. Figure 5.8 displays the decision chart associated with the residential mobility mechanism.

Figure 5.8: Agent's decision chart for residential mobility



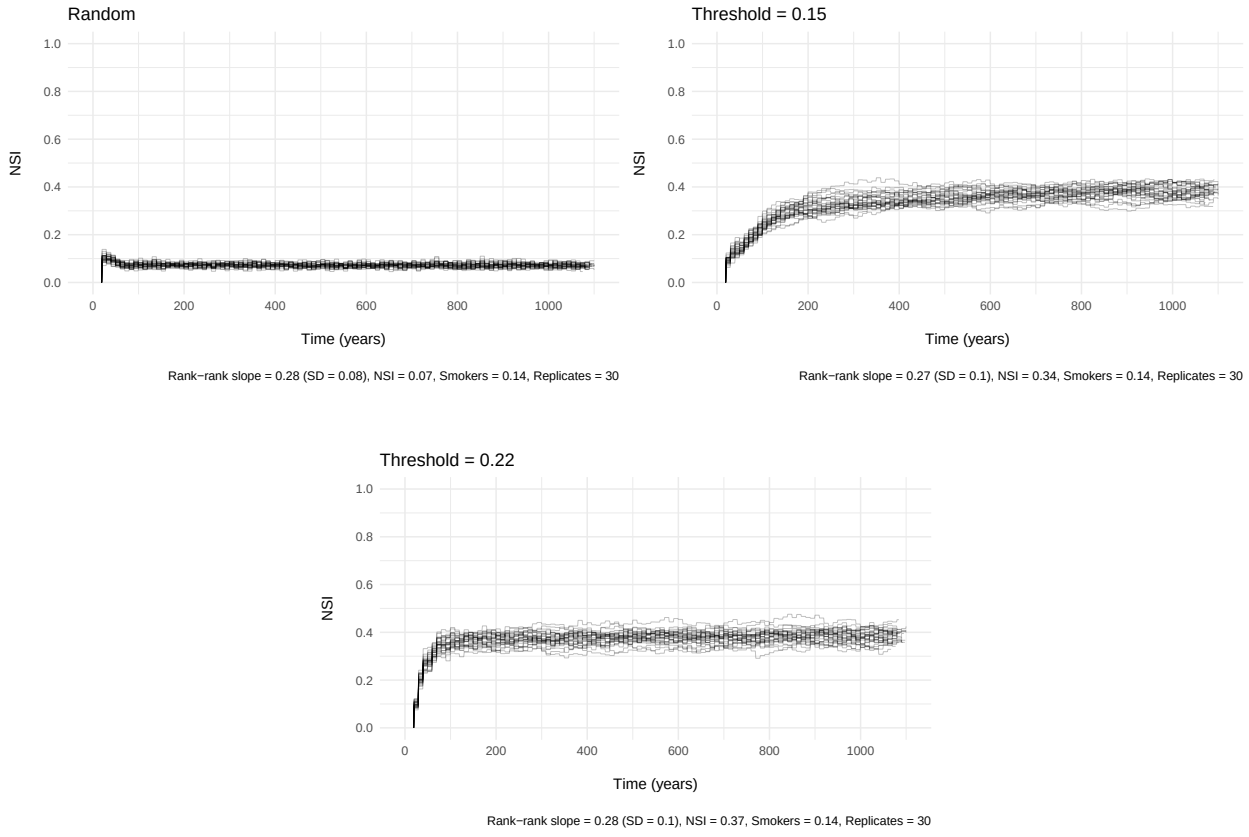
We use the neighborhood sorting index or NSI to measure income segregation (Jargowsky & Kim, 2005). The NSI compares the income variation across all neighborhoods (or counties) in a metro area with the income variation across all households in that metro area. If agents are segregated across counties 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 propose of our model, that limitation is not problematic.

To examine the changes in the levels of income segregation, we use a decision to move rate (mob_r) of 0.10 per year. When movement is completely random, that rate generates about 7.6 moves on average over agents' life course. Once the segregation mechanism is in action, the number of moves is reduced about half as agents do not always have incentives to leave their

county of residence. We explored three scenarios: (1) all agents move randomly, (2) only 1% of agents move randomly with tolerance threshold 0.15, and (3) we increased the tolerance threshold to 0.22. Figure 5.9 shows the dynamics of the NSI over 30 simulated generations and about 1100 simulation years per replicate. The NSI goes from 0.07 when the movement is completely random, to 0.36 when the moving threshold is 0.22.

Figure 5.9: Neighborhood sorting index (NSI) by year (30 replicates)



These differences depend on how the segregation model was implemented (income transition matrices used, number of income groups and counties). Despite random fluctuations, the NSI trajectories are relatively stable over time. As expected, the highest number of moves is observed when the movement is completely random (7.6 moves on average during agents' life course), while the fewest moves (0.7) when mob_{thr} is the lowest (0.15), and 2.3 moves when $mob_{thr} = 0.22$. The average of moves during childhood (before age 18) goes from 0.25 to 1.8. According to Jargowsky

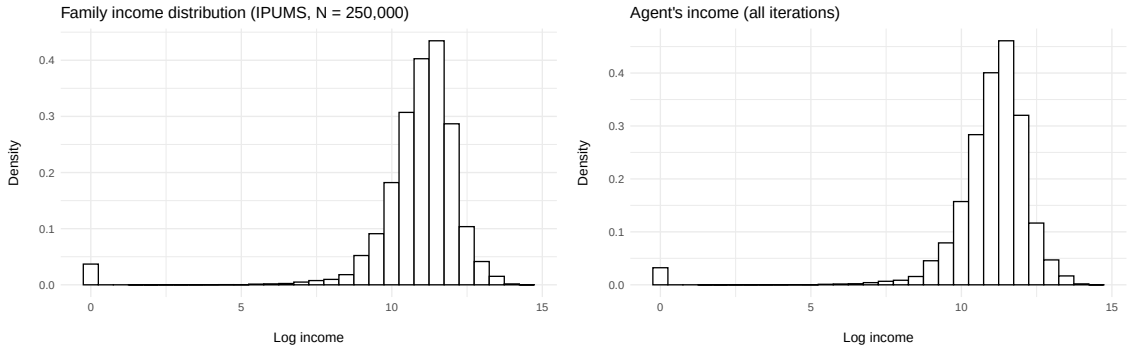
and Wheeler (2017), the economic segregation in the US in 2010 was 0.396. To reproduce those levels of income segregation, we use a tolerance threshold mob_{thr} that generates NSI levels close to 0.40.

Income mobility

MIA's income generation and mobility mechanisms consist of two components: (1) the link between the parent's and child's income group, and (2) the impact of the exposure to the county's resources on child's income group. We verified the implementation of both mechanisms by examining the distribution of income, income mobility, and segregation. We defined three scenarios or iterations using the baseline parameters (see Table 5.1 in the paper): (1) the base probability of being in the same income group of the parent is 0.30, the probability for the rest of the income groups is $(1-0.3)/4 = 0.175$, and $w_k = 0$, that is, there is no effect of the county's income group composition on the child's income group definition; (2) the same as (1), but this time the transition probabilities change based on the exposure of county income group composition using ($w_k = 0.5$); (3) the transition probabilities come from a sample of commuting zones probabilities estimated by Chetty et al. (2014), and they are assigned at the county level using $w_k = 0$. As expected, MIA generates an income distribution that mimics the IPUMS family income distribution (sample size = 250,000, see Figure 5.10). Once an agent has been assigned to an income class (1 to 5), we drew a weighted-sample from the IPUMS income distribution for that income quintile.

The transition matrices shown below correspond to the average transition probabilities across agents and replicates for each of the three scenarios we explored. The first matrix I_1 displays the expected probabilities and they are pretty close to the expected values: 0.30 for the diagonal and 0.175 elsewhere. The overall rank-rank slope in this iteration is 0.13 (all agents), almost the same as the average of county's rank-rank slopes (0.12, SD = 0.08), while the NSI is 0.35 (see Table 5.5).

Figure 5.10: Individual income distribution (30 replicates)



$$I_1 = \begin{bmatrix} 0.299 & 0.175 & 0.176 & 0.175 & 0.175 \\ 0.176 & 0.300 & 0.175 & 0.175 & 0.175 \\ 0.175 & 0.175 & 0.300 & 0.175 & 0.175 \\ 0.175 & 0.175 & 0.175 & 0.301 & 0.174 \\ 0.175 & 0.175 & 0.175 & 0.175 & 0.301 \end{bmatrix} \qquad I_2 = \begin{bmatrix} 0.331 & 0.165 & 0.163 & 0.168 & 0.173 \\ 0.161 & 0.333 & 0.166 & 0.168 & 0.172 \\ 0.159 & 0.163 & 0.342 & 0.170 & 0.166 \\ 0.160 & 0.163 & 0.168 & 0.343 & 0.166 \\ 0.160 & 0.166 & 0.161 & 0.162 & 0.351 \end{bmatrix}$$

$$I_3 = \begin{bmatrix} 0.322 & 0.263 & 0.190 & 0.134 & 0.091 \\ 0.222 & 0.234 & 0.216 & 0.192 & 0.137 \\ 0.151 & 0.186 & 0.215 & 0.235 & 0.213 \\ 0.112 & 0.149 & 0.199 & 0.259 & 0.281 \\ 0.096 & 0.112 & 0.170 & 0.247 & 0.376 \end{bmatrix}$$

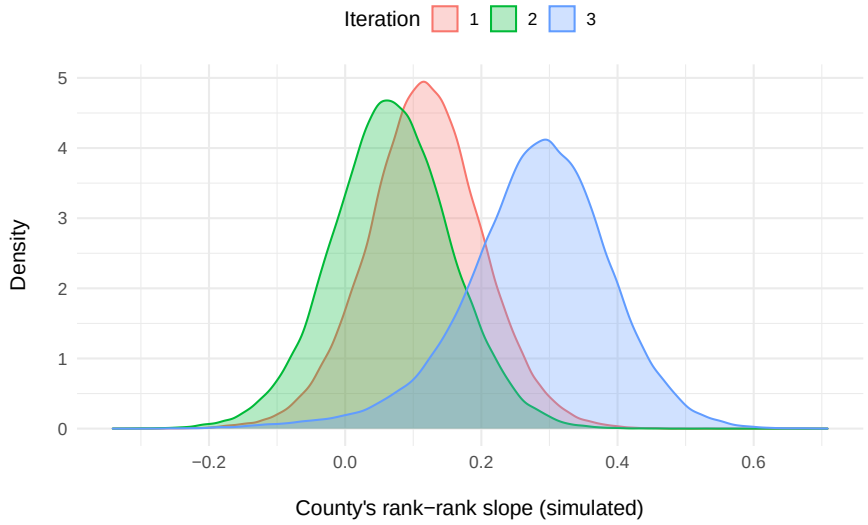
The second scenario adds the effect of average exposure to income composition on the transition probabilities using $w_k = 0.5$. The probabilities of being in the same income group of their parents (diagonal) increase about 4 percentage points, while county's resources generate more variability in the probabilities outside the diagonal, although differences are not bigger than 1 percentage point. Interestingly, the NSI increases as the probability of being in the same income group of the previous generation is higher. In other words, the NSI might change independently of the parameters of residential mobility and only due to changes in the transition matrix. The overall rank-rank slope increases, while the average of county's rank-rank slopes is closer to 0 with

a slightly higher standard deviation (0.06, SD = 0.09). The second scenario generates a higher number of counties with a small or negative rank-rank slope (see Figure 5.11) as counties become relatively more homogeneous (higher NSI and lower variability of income within counties).

Table 5.5: Summary statistics income generation verification by scenario (30 replicates)

Scenario	NSI	Gini	Rank-rank slope	County's rank-rank Slope	County's rank-rank Slope SD	Avg. Population
1	0.35	0.47	0.13	0.12	0.08	8079.50
2	0.40	0.47	0.16	0.06	0.09	8097.60
3	0.38	0.46	0.31	0.28	0.11	7994.60

Figure 5.11: County's rank-rank slope distribution (simulated)



Finally, the transition probabilities are defined exogenously by sampling the commuting zone's transition matrices estimated by Chetty et al. (2014). The transition matrix I_3 is similar to the overall transition matrix estimated by Chetty et al. (2014) (see Equation 5.2 in the paper). The NSI is 0.38, and the overall rank-rank slope is slightly higher (0.31) than the the average of county's rank-rank slopes (0.28, SE = 0.11). As the observed rank-rank slope distribution in Figure 5.2 (in the paper), most of the county's rank-rank slopes are positive (Figure 5.11). The NSI is similar to the second scenario, but mostly due to an increase in the probability of the transition matrix diagonal for the lowest and the highest quintile rather than a higher overall county homogeneity. Indeed, the standard deviation of the variability of the income distribution within counties is higher than what we observe in iterations one and two.

Smoking

MIA defines the smoking status of agents at age 30 using Equation 5.4 in the paper. To assess the absolute magnitude of the effect of income mobility on life expectancy, we calibrated the smoking prevalence by income quintile so that the proportion of smokers matches the proportions observed in NHIS 2019 when the income mobility coefficient $\beta_{s_{imob}}$ is higher than zero. Table 5.6 displays

the smoking prevalence by income quintile. When $\beta_{s_{imob}}$ is equal to the effect estimated by Daza and Palloni (2021) (MIA treatment), the proportion of smokers increases by 5 percentage points with respect to the counterfactual where $\beta_{s_{imob}} = 0$. The distribution of the column *MIA treatment* is pretty close to the empirical distribution estimated using NHIS 2019. The effect of smoking on mortality also generates life expectancy differences that are about 10 years on average (10.9). As expected, those differences are practically the same across income groups.

Table 5.6: Proportion smoking by income quintile

Income group	NHIS 2019	MIA Counterfactual	MIA Treatment
1	0.29	0.21	0.28
2	0.22	0.15	0.22
3	0.16	0.09	0.13
4	0.11	0.07	0.10
5	0.05	0.03	0.04
Total	0.17	0.10	0.15

NHIS 2019 = 10,338 respondents ages 30-50. MIA counterfactual = 8407 agents (30 replicates), MIA treatment = 8226 agents (30 replicates).

Measurement

We verified rank-rank slope, NSI, Gini coefficient, life expectancy were measured correctly by exporting data generated by MIA, and recomputing those statistics using *raw* data outside Anylogic. The measurement of the county's rank-rank slopes and life expectancies was implemented using moving cohorts (i.e., similar to moving averages), so that we have enough data records to estimate these values every t number of years. While Figure 5.12 displays how MIA record information at the agent level, Figure 5.13 shows the size's distribution of the cohorts used to estimate both rank-rank slopes, and life expectancy.

Figure 5.12: Agent's measurement diagram

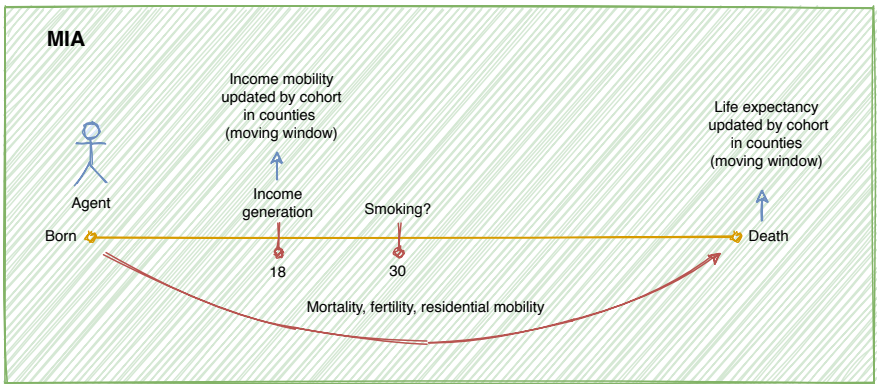
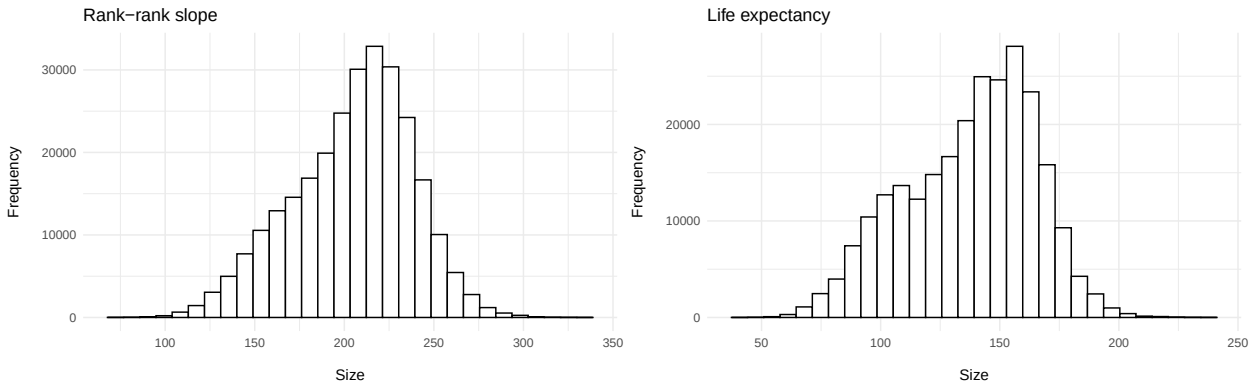


Figure 5.13: Cohort size to estimate county's rank-rank slope and life expectancy (30 replicates)



Final remarks

In this dissertation, I use different data sources and modeling approaches to examine the strength and magnitude of the association between place's income mobility and health and to start formalizing some of the mechanisms that generate it.

My results are somewhat mixed. While I am able to confirm some of the associations reported in previous studies, (Venkataramani et al., 2016; Venkataramani et al., 2015; Venkataramani, Daza, & Ezekiel, 2020), especially when using the aggregate data, the most robust of the analysis – employing individual and longitudinal data – shows that the association between income mobility county's exposure during childhood and adolescence with health indicators and behavior is not systematic. My *stylized* agent-based model also shows that estimates are sensitive to different measurement schemes, even when the stratification regime remains the same over time. Still, the most systematic findings I found are related to smoking, and the results from the *microsimulation* using as outcome life expectancy at birth reveal that the independent impact of the stratification regime on smoking might have the potential to explain part of the longevity gaps observed in the US. These effects might be larger if we consider other behaviors associated with the so-called *deaths of despair* (Case & Deaton, 2020), such as drug and alcohol use.

Future research on this subject has many challenges, but also opportunities. Given the theory and mechanisms proposed in the literature, research should focus mostly on individual-level analysis using data that capture, as best as possible, exposure to place's income mobility. This is not trivial at all, as research on the effects of early conditions on adulthood outcomes has shown (Haas & Bishop, 2010). Data availability at the level of small geographic units is also a limitation. All

analyses discussed in this dissertation come from Chetty et al., 2014's income mobility estimates from data sources that are pretty difficult to access (e.g., tax and social security records). It seems more feasible to estimate intergenerational mobility using longitudinal data and explore how those *individual* measures relate to health and mortality, under the assumption that they would represent better the individual *perception* of the income mobility regime or prospects of upward mobility.

Additional analysis would also be needed to assess the consequences of measurement error of Chetty et al., 2014's income mobility measures. As Mogstad et al. (2020) and some of our analyses suggest, measurement error might be driving some of the associations between social mobility and health. Previous studies can incorporate measurement error into their models and examine if their results hold. Our simulations also show different income mobility measures (e.g., rank-rank correlation vs. rank-rank slope) might have consequences on the results, especially when analyzing small geographic areas and in a context of segregation. We need to thoroughly explore these issues using our agent-based models to provide concrete and clear recommendations to researchers.

Finally, generative models like the one I propose can help formalize old or new mechanisms to create an association between income mobility and health (e.g., intergenerational transmission, network effects, etc). Besides exploring the relative importance of the potential mechanisms, a generative model might also help design new studies, data collection efforts, and guide analysis.

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