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

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Abstract

We test the conjecture that intergenerational social mobility is tightly linked to adult mortality even after accounting for income levels, income inequality, and indicators of social capital, health behaviors and levels of access to health services. We use a large dataset from The Health Inequality Project and estimate simple multivariate models with U.S. counties and commuting zones as units of analysis. We find that the estimated effects of indicators of social mobility are strong and in the expected direction, robust to model specification, and translate into differences in life expectancy at age 40 as large 2.0-4.8 years among males and 0.1-2.0 among females. These are equivalent to 5.1-12.5 and 0.2-4.7 percent of the U.S. average life expectancy at age 40 among males and females respectively. On average, the magnitude of these effects is 1.5 to 2.5 times as large as those of income inequality and about 40 (males) and 25 (females) percent of the magnitude of a change from the lowest to the highest quartile of the U.S. income distribution.
1 The relation between income inequality and adult mortality

1.1 Empirics

There is a large body of research on the relation between income inequality and health and mortality (Pickett and Wilkinson 2015; Subramanian and Kawachi 2004; Wagstaff and vanDoorslaer 2000; Wilkinson and Pickett 2006, 2010; Kawachi et al. 1997; Wilkinson 1992). Empirical evidence from country data as well as from small areas (even individuals) suggests the existence of an association between levels of aggregate inequality and aggregate indicators of health and mortality (Daly and Wilson 2013). In the most recent contribution primarily aimed at sizing the effects of income on mortality, Chetty and colleagues (Chetty et al. 2016) confirm the existence of an empirical association, albeit modest, that follows the expected pattern - higher inequality is associated with higher mortality - among sub-populations at the bottom of the income distribution. Furthermore, the relation is stronger among those at the top of the income distribution. Thus, as conjectured by some scholars, higher levels of inequalities appear to be damaging for all (Subramanian and Kawachi 2006; Pickett and Wilkinson 2009; Wilkinson and Pickett 2010, 2009).

1.2 Mechanisms

Aside from population composition effects induced by the relative size of the population in the lowest income ranks or other such artifacts (Lynch et al. 2004) the relation between income inequality and health and mortality has been attributed to two mechanisms operating separately or jointly. The first mechanism involves contextual conditions shared by unequal societies that damage the health of all individuals, including those occupying top positions. Societies with highly unequal income distributions also have lower levels of human capital investments, rank lower in the magnitude of public expenditures and welfare (Kawachi and Kennedy 1997; Wilkinson 1992), experience higher crime rates and levels of insecurity (Peterson and Krivo 2012), poorer environmental quality (Leigh and Smeeding 2009) and, more generally, are characterized by divisive and corrosive social relations.

Although previous findings and argumentation point to the existence of negative health and mortality effects for the entire population exposed to a given regime of inequality, Chetty’s data suggest a stronger effect for those at the top of the income distribution.

In what follows we will use the term effects when referring to the magnitude and sign of standardized or unstandardized path coefficients measuring the strength of the relation between two variables. Thus, unless explicitly noted, when we refer to effects of $x$ on $y$ we do not presume the existence of a proven causal relation whereby a change in $x$ induces a direct or indirect change in $y$. 
Wilkinson and Pickett 2009 and weak social cohesion (Kawachi and Kennedy 1997; Lynch et al. 2004; Berkman and T.Glass 2000; Sampson 2013) all of which damage the fabric of social relations and have deleterious effects on the health for all individuals alike (Wilkinson 1992; Pickett and Wilkinson 2009).

A second set of mechanisms implicates biological and psychosocial processes and is the focus of intense attention. It has elicited empirical support from disparate disciplinary corners, including neurosciences, psychiatry, and even primate studies (Marmot and Sapolsky 2014; Sapolsky 2005; Marmot 2004). These mechanisms operate through a number of pathways that generate relative deprivation and sustained increases in levels of individual stress among those in lower income ranks. When these impacts are enduring and remain unchecked by coping outlets, they can cause significant physiological and mental damage (Marmot 2004; Meaney 2001; McEwen 1998; Seeman and Lewis 1997; Cacioppo et al. 2002).

These two distinct mechanisms are somewhat independent though they could reinforce (or offset) each other. Thus, in theory at least, if one could exogenously alter conditions that regulate the creation and persistence of social cohesiveness and the quality of personal relations in highly unequal societies, the individual health damage associated with chronic stress would be attenuated. As a consequence, the health and mortality risks of individuals in the most vulnerable social positions would resemble more closely those of individuals in similar positions living in societies with a more equitable income distribution.

We argue below that the existence of a flexible and well-oiled regime of social mobility could operate in ways similar to an exogenous interventions, reducing the exposure to health risks or improving resilience to manage conditions detrimental to health in communities and societies with unequal income distributions.

2 Why should there be a relation between social mobility on mortality?

Just as social inequalities manifest themselves in individual ill-health conditions and augmented mortality risks, so could social mobility unlock mechanisms that damage individuals’ health status. Two pathways could cement an association between indicators of social mobility and mortality.
One is a result of an artifact and the other is a genuinely causal one.

2.1 Artifact

The first pathway is a product of an artifact that occurs as a result of the relation between income inequality and income/social mobility. This is the inverse relation, referred to as the “Great Gatsby Curve”, that singles out an intriguing empirical regularity, namely, that countries with higher income inequality score lower in an indicator of social mobility (Corak 2013; Winship 2015; Krueger 2012). Recent empirical findings show that the relation is not just observable across countries but also across smaller units of analysis, such as commuting zones in the U.S. (Chetty et al. 2014). To the extent, then, that income inequality is associated with mortality levels, one would expect that indicators of income mobility should also be associated with adult mortality if and when the influences of income inequality are not properly purged out. Indeed, if the relation between these two dimensions of income stratification is very tight it could be difficult, if not impossible, to disentangle the impacts that pertain to the domain of income inequality and those attributable to income mobility.

It could be argued, however, that the presumed relation between income inequality and income mobility is neither causal nor empirically robust. In fact, estimates of the association between income mobility and income inequality have been questioned and demonstrated to be somewhat vulnerable. The workhorse indicator of income mobility is the intergenerational elasticity of income (Solon 1992; Lee and Solon 2009) or the regression coefficient of offspring log income on parental log income. This indicator - on which most accounts of the great Gatsby curve relies upon - is unstable partly because the log-log relation is non-linear and sensitive to the magnitude of inequality imparted by income concentration at the top of the income distribution, and partly because elasticity estimates vary with the strategy researchers use to handle observations with no or very small incomes.

An alternative measure, the rank-rank specification (Dahl and DeLeire 2008), is immune to these two problems and is used in recent investigations on intergenerational income mobility (Chetty et al. 2014; Winship 2015). This research reveals, for example, that while U.S. income inequality has been

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To avoid cluttering we use the term “inequality” as shorthand for income inequality and “social mobility” or “mobility” as an abbreviation for income mobility. We are cognizant that both inequality and social inequality can be measured using indicators other than income.
steadily rising for over twenty years, the rank-rank income mobility measure shows no significant changes over time, at least for Americans cohorts born between 1970 and 1990 (Lee and Solon 2009; Kopczuk et al. 2010; Chetty et al. 2014, 2016). Similarly, the association observed across American labor markets is weak and vanishes after proper controls are added (Chetty et al. 2014). Estimates from very simple models we propose below further confirm findings of a weak relation between income inequality and income mobility across counties and commuting zones in the U.S.

2.2 Plausibility of a causal relation between social mobility and health and mortality

The fact that the relation between income inequality and income mobility is not perfect and highly variable, open the gates to the possibility of empirically identifying mechanisms whereby income mobility, instead of or in addition to income inequality, influences mortality. Are there plausible and verifiable pathways through which societies with higher intergenerational mobility may host social and economic environments where individual health status is superior and mortality risks reduced relative to societies with lower intergenerational mobility, independently of effects of income and income inequality?

A causal relationship between income mobility and health and mortality can be expressed in the following terms: individuals and groups who occupy the most vulnerable and exposed social positions within unequal communities are comparatively better off when they confront higher income mobility prospects than when they do not. Just as individuals who command lower incomes in communities with more equitable income distribution may experience better health conditions than individuals with similar incomes in societies with higher income inequality, so too could individuals and groups in lower ranked income positions living in societies with higher income mobility enjoy better health than counterparts in societies with more rigid stratification systems.

Admittedly, highly mobile communities may also experience a share of downward mobility and, therefore, expose a sub-population to hypothetical deleterious effects. However, in a steady state what is relevant is not the volume or even the existence of the actual downward flow but rather individuals’ current behaviors and experiences designed to protect against the anticipated threat of a descent of income ranking. The potential health losses in terms of stress induced by heightened vigilance among those at the top are reduced or offset by the benefits inherent in current high
To simplify assume that societies can be classified in one of the four cells in Table 1. Each cell in the table may be characterized by a health and mortality profile. Further, in view of the moderate relation between inequality and social mobility, we take for granted that the frequency of observations is non-zero in all cells, that is, that communities simultaneously characterized by unequal income distribution and flexible mobility regimes or, alternatively, by generous income distributions and high levels of income rigidity, are observable.

Our conjecture is that indicators of mortality and health will be more beneficial in communities with less inequality (B and D) than in those with high inequality (A and C). We also expect that, at a given level of inequality, better health and mortality conditions will be experienced by members of communities with higher mobility, A versus C, on one hand, and B versus D, on the other. As we argue below, the existence of an efficient income mobility-health (mortality) mechanism must produce two empirical regularities: (a) a graduated relation between mortality and income mobility indicators so the highest (benign) effect is felt among those in the lowest income rankings and the lowest is felt by those situated at the top of the income distribution; (b) there should be significant interaction effects so that differences in income mobility ought to be more consequential under highly unequal income distributions than in more equitable communities. We test these two predictions below.

2.3 Income mobility, individual behaviors, and health and mortality

What are feasible routes through which a contextual property such as aggregate income mobility could impact individual health and mortality? A massive literature on health and mortality disparities provides clues about sources of strong linkages between social mobility and health and mortality. It is well-known 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). Although we have not successfully identified a single factor that could account for these disparities, there is agreement that early conditions and upbringing of individuals matter (Palloni et al. 2009; Case and C.Paxson 2002). Early conditions may not be the smoking gun we are seeking but it is a candidate explanation that researchers should not ignore or deemphasize.

In what sense do early conditions matter? There are separate bodies of research, involving quite
disparate disciplines, each identifying two sets of linkages. One is rooted in developmental biology and affine fields. The most significant contributions in this area come from the so-called Developmental Origins of Adult Health and Disease (DOHaD) (Gluckman and Hanson 2006), a body of work which adopts, expands, and enriches the idea of fetal programming initially put forward by Barker (1998). In a nutshell, the idea is that insults, deprivation and adversity experienced in utero and early life may induce physiological and psychological damage that remain latent and manifest themselves as delayed ill-health and higher mortality risks at adult ages.

The second line of research focuses on conditions during early stages of socialization and upbringing, a period during which individuals experience sensitive or critical windows for the acquisition of cognitive and non-cognitive abilities that are the foundation of skills acquired later in life (Knudsen et al. 2006; Shonkoff et al. 2009; Heckman 2007; Cunha and Heckman 2009). Some of these early traits are also implicated in the formation of outlooks and attitudes that influence adult behaviors including health behaviors.

Both lines of research offer empirical support for the idea that some of the health and mortality disparities observed during adulthood could be traced to early conditions. Furthermore, if they do matter for health and mortality disparities they ought to also matter as explanations for the relations between income inequality and adult mortality. Thus, some of the health differentials between men in low and high ranking positions initially attributable to chronic stress associated with subordinate positions (Marmot 2004; Sapolsky 2005) may be rooted in antecedent health conditions sculpted early in life (Case and Paxson 2011). This means that at least part of the impact of aggregate income inequality that materializes in worse health status among those occupying lower strata have roots in early childhood and conditions experienced therein.

By the same token, health behaviors critically associated with modern chronic illnesses, such as smoking, alcohol consumption, substance abuse, choice of diet and physical activity, are in part determined by capabilities sculpted early in life (Grossman 2000). Early adoption of healthy behaviors has large health payoffs in adulthood because these behaviors are closely related and reinforce each other, because the physiological and psychological damage they produce is accumulated over time, and because these are strongly non-reversible. Early adoption of healthy behaviors is facilitated by socialization that emphasizes strong 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 additional healthy years lived are associated with increasing returns. We know from empirical research that negative affect, chronic stress, subordination, and poverty in general lead to increases in time discounting (Haushofer and Fehr 2014). Higher time preference favors or reduces resistance to the adoption of unhealthy behaviors that may yield immediate rewards and discourage those that have a more distant and elusive pay off (Schlam et al. 2013; Eigsti et al. 2006).

Communities with low-income mobility distort opportunities and incentives, heighten income-related disparities of differential distribution of individual traits, undermine and undervalue public institutions that sculpt characteristics and skills valued in the labor market, and craft support for non-meritocratic forms of reward allocation. These properties influence the way parents socialize children and favor (discourage) the adoption of positive outlooks and the value of skill acquisition. Lack of mobility and an inflexible stratification system foster individual hopelessness, mistrust, lack of confidence in the system, 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.

Three caveats are needed. First, some of the contextual conditions generated by income inequality are themselves the direct cause of income rigidity. For example, the strength of nepotistic relations is surely stronger in communities with high-income inequality. But entrenched nepotism also inhibits income mobility in addition to distorting incentives and biasing rewards thus inducing mistrust in the system.

Second, not all highly unequal societies are created equal. In some cases, inequality arises from growth of the share of income going to the very top one percent of the income distribution. This is consistent with the U.S. experience over the last 20 years. But, in other cases, inequality is caused by an increase in the share of the population in the lower income ranks. These are two different types of inequality regimes, each with its own repertoire of possibly different effects on health and mortality. More importantly, these regimes may allow contrasting income mobility which, by themselves, can exert different health and mortality effects. Unless one is able to formally define and empirically identify these various configurations, we cannot aspire to formulate precise predictions, models or estimation.

Finally, none of this would matter much in societies that are not under the grip of modern chronic illnesses, all highly dependent on individual choices, behaviors and attitudes. Neither income in-
equality nor income mobility could be attributed causal priority in pre-germ theory epidemiological regimes dominated by infectious and parasitic diseases.

3 Estimation

3.1 Description of dataset and measure of mobility

We use the dataset created by Chetty and colleagues (Chetty et al. 2016). This dataset contains information on income from tax records for the period 1999 and 2014. A total of 1.4 billion tax records were used and linked to Social Security Administration records. Estimates of race and ethnicity adjusted mortality rates between ages 40 and 76 were expanded to include older ages using parameters from a Gompertz function fitted to rates for ages younger than 76. The information on age-specific mortality rates was converted into an estimate of life expectancy at age 40. This indicator summarizes mortality experiences at ages older than 40 for cohorts born between 1923 and 1959.

The dataset also contains a number of income mobility indicators derived from measures of the association between incomes of children born between 1971 and 1993 and their parents’ income. In this paper, we use only two measures. The first is relative mobility or the rank-rank slope defined as the correlation between a child’s income rank in her birth cohort income distribution and parents’ income rank in parents’ income distribution. The second indicator is the absolute upward mobility score or “the mean rank (in the national income distribution) of children whose parents are at the 25th percentile of the national parent income distribution” (Chetty et al. 2014, p. 7). At least at the national level, both the relative and absolute measure of mobility provide similar information.

For our purposes at least, these data contain an important limitation. All the measures of mobility characterize cohorts that are younger than the cohorts whose mortality experiences is embedded in estimates of life expectancy at age 40. One could think that we are availing ourselves of measures of offspring income mobility experiences but assess their parent’s mortality experiences. And yet our objective is to test the conjecture that the exposure to health and mortality risks during early and late adulthood are related to social mobility prospects during life stages preceding...

(Data for cohorts born between 1971 and 1979 come from different sources than those for the cohorts born between 1980 and 1993. In addition, for the most recent cohort (born after 1986) the measure of mobility uses probabilities of attending college rather than their income. For a full description of the measures see Chetty et al. 2014, 2016.)
the completion of educational attainment. Thus, there is a generational dislocation between the mortality risks we consider and the income mobility experiences we measure. The analysis that follows can only lead to approximately correct inferences if income mobility in the small areas we study is not a fleeting feature but rather reflects entrenched local conditions that make the social mobility experience of one generation an enduring feature shared by subsequent generations.

There are two pieces of evidence to support this contention but neither offers a safe and clean escape from the problem. First, there is mounting evidence that national and local U.S. mobility trends have been steady for longer than a generation. This suggests that there must be a more than modest correlation between the income mobility regimes that parents and offspring experience during relevant life cycle stages.

Second, analysis of the mobility data by commuting zones and counties reveals the existence of strong correlations between income mobility regimes and indicators of small area characteristics that are not transient. Thus, there are strong correlations with race composition, levels of segregation, income levels and inequalities, school quality, family structure and, finally, a number of indicators of social capital (Chetty et al. 2016). These empirical results suggest permanence, not fleetingness, of mobility regimes and even suggest policy interventions that could only be issued if the indicators of social mobility are related to strategic structural, enduring properties of local communities: “The main lesson of our analysis is that intergenerational mobility is a local problem, one that could potentially be tackled using place-based politics” (Chetty et al. 2014, p. 42).

These disclaimers support the idea that current income mobility regimes are a good indicator of past mobility regimes and, in particular, that studying the relation between older cohort’s mortality through the prism of younger cohorts’ mobility may not produce completely erroneous inferences. However, to prevent reading too much into the results we discuss below we also compute bounds of uncertainty of correlations under alternative assumptions about the association of multigenerational income mobility regimes.

Income mobility across generations may follow a simple process \( IM(t) = \alpha \cdot IM(t-k) + \varepsilon \) where \( IM(t) \) and \( IM(t-k) \) stand for income mobility in generation \( k \) and \( t-k \) respectively and \( \varepsilon \sim N(0, \sigma) \). In the supplemental material we use this expression to compute bounds of uncertainty for our estimates of effects of social mobility on life expectancy. The sources of uncertainty are (a) the strength of the association between income mobility across generations \( \alpha \), and (b) the error variance \( \sigma^2 \).
3.2 Estimation of effects

It is well known that income is associated with mortality rates at all ages and with summary measures of longevity such as residual life expectancy (Cutler and Lleras-Muney 2006). Estimates from the local area dataset used here (Chetty et al. 2016) suggest that differences in life expectancy at age 40 between those in the first and fourth quartile of the income distribution are approximately 8 years for males and 5 years for females, representing 21 and 12 percent of the male and female life expectancy in the U.S., respectively. These gaps have increased slightly between 2000 and 2015 and have substantial geographic variation (Chetty et al. 2016).

3.2.1 Additive models

We begin by assessing the relation between income inequality measured by Gini index (GI) and relative and absolute income mobility (IM). Figure 1 displays the plots of the relative and absolute income mobility indicator and the Gini index. As expected, the relation is moderately strong ($r = -0.67$ and $r = 0.48$) and local places with low levels of income mobility tend to have higher aggregate inequality, irrespective of the nature of IM.

The upside of the empirical association between the two dimensions of income stratification provides some support for the idea that income mobility measured by indicators pertaining to a younger generation is not a transient feature of communities. In fact, it is standard in the literature to interpret income inequality at a particular time as a reflection of structural, lasting properties of a community. The downside of the association is that it complicates the task of separating the effects of each on health and mortality.

Figure 2 includes scatter plots of the relation between life expectancy and income inequality (GI), on one hand, and life expectancy (LE) and relative income mobility (IM), on the other, across commuting zones. There are three salient features in these plots. First, the association between GI and LE among males ($r = -0.47$) confirms results from other research that increased

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6We use GI computed after excluding the top 1 percent of the income distribution. The numerical range for relative IM is (-1,1) and the higher its value, the lower is income mobility. The numerical range for the absolute IM is (0-100) and the higher its value the higher is income mobility.

7Hereafter we only discuss estimates based on commuting zones (CZ) as units of analysis instead of counties. This is because CZ are the analytic units on which mobility indexes were computed (Chetty et al. 2014). Furthermore, we will show results using the indicator of relative mobility only. In the supplemental material, we present results using counties as units of observations and absolute mobility as indicator of income mobility. None of our inferences are sensitive to these choices.
inequality has harmful effects on mortality. Second, the association between IM and LE among males \((r = -0.50)\) is slightly stronger than the one with GI and also in the expected direction. Third, both associations are stronger among males than among females \((r = -0.34 \text{ and } -0.31)\).

Even though Figures 1 and 2 reveal associations at the aggregate level, none of them helps to sort out to what extent the association between IM and LE is attributable to background GI. We show below that most of the effects of IM that are detectable in Figure 2 persist even when one accounts for effects of GI.

The patterns of relations also change significantly when we examine them by income quartiles. Figure 3 and 4 display the associations between GI, IM and LE within four income quartiles. The relationships between GI and LE in Figure 3 are moderate and range from \(-0.17\) to \(-0.44\) among males and from \(0.01\) to \(-0.32\) among females, are weakest at both extremes of the income distribution and, as in the aggregate case examined before, they are weaker among females than males. Figure 4 display associations between IM and LE. The relations are somewhat stronger than those involving GI for males, all are properly signed and, as conjectured before, appear to be graduated by income quartile. Thus, the associations for males range from \(-0.22\) to \(-0.49\), the highest value is at the bottom of the income distribution and the lowest at the top. Among females, the associations are weaker and range from \(-0.16\) to \(-0.22\) by income quartiles.

So far one could argue that income mobility is, at the very least as important for LE as is income inequality. Thus, it is rather surprising that it has not received any attention as a potentially important mortality determinant.\(^8\) But there is more to this story. As we show below the effects of income mobility are quite durable, even after accounting for income inequality whereas the latter’s behave erratically, flip signs or vanish.

Tables 3 and 4 display estimated effects of GI, IM in five alternative models.\(^9\) The first two columns contain estimates of the relation between LE and the z-scores of GI (first column) and IM (second column). The second model (third column) includes both indicators simultaneously whereas the third (fourth column) adds an interaction term between GI and IM. The fourth model

\(^8\)We hasten to add that there is a vast literature on the effects of individual experiences of social mobility on individual health and mortality. Instead, our conjecture is about effects of social contexts with variable income mobility on health and mortality experienced in the community.

\(^9\)We address conditional dependence between counties and commuting zones by state using generalized estimating equations (GEEs) with an “exchangeable” or “compound symmetry” correlation structure. Although GEE estimates using other correlation structures may alter the results, GEE is only slightly responsive to the choice of correlation structure (Liang et al. 1992). We present robust and clustered standard errors.
adds a suite of community variables as controls (see Table 2 for descriptives). The estimated effects of IM in the third column of Table 3 (males) are statistically significant for the first two income quartiles \((p < .001)\), always properly signed and, importantly and as predicted, become weaker as one climbs in the income distribution. Instead, the estimates of GI are positive and significant in the first income quartile - inequality seems beneficial at the bottom of the income distribution - and flip sign but become insignificant in higher income quartiles. That is, among males income inequality is better for those who are worst off and neutral for the rest of the population.

The patterns for females in Table 4 are similar. First, as happens among males the estimated effects of IM are significant in the first two income quartiles, all are properly signed, and drift to zero in the upper part of the income distribution. Second, the effects of inequality are positive and significant in the first quartile, flip sign and maintain significance in the other quartiles. That is, among females income inequality is better for life expectancy for those who are worse off but worst for those who are better off.

3.2.2 Models with interactions

The results for males at least are consistent with the first conjecture formulated before about graduated effects of IM on LE. What about the second conjecture, namely, that effects of IM should be stronger when income inequality is sharper? The estimates in the fourth column (model with the term GI x IM) of Tables 3 and 4 do not confirm this. Among males the direction of the relation is opposite to expected as effects of IM decrease as GI increases, although the levels of significance are marginal. In contrast, among females the estimates have the right sign for all income quartiles and their magnitude slightly exceeds those for males.

3.2.3 Models with controls

The last column of Tables 3 and 4 displays estimates in models that are like model 3 (fourth column) but with a suite of control variables added to it. These include indicators of access to health care, environmental factors, labor market variables, health behaviors (including smoking) (see Table 2). We estimate these models not as a confirmatory device but rather to set low bounds for the estimates of interest. It could be argued that some of the control variables included in this last model are indicators of mediators through which either IM or GI influence LE, in which
case introducing the control sequesters part of or all the impact that belongs to IM (or GI). Thus, for example, suppose that weak income mobility induces a lack of individual vigilance regarding exposure to health risks and increases smoking uptake. If so, a control for smoking (proportion of smokers in the CZ) in the model will obscure the relation since it factors out part of the effect we are interested in. Thus, the lower bounds of estimates in the fifth column are useful if one insists that the relation between IM and LE is spurious and all controls added are proxies for the left out variables that generate the spuriousness. The results in the fourth column of the tables suggest that at least among males the effects of income mobility become statistically insignificant but remain properly signed, whereas those for females also drift away except in the first quartile of the income distribution where they remain significant and properly signed.

The results above follow a pattern that admits the interpretation that income mobility not only has effects of its own but is an additional, but so far ignored, mechanism through which income inequality influences health and mortality. Consequently, the effects of income inequality that remain after controlling for income mobility - that is, not many - reflect the impact of other, better known and better studied mediators. None of this detracts from the importance of income mobility (and its own mediators).\textsuperscript{10}

\subsection*{3.2.4 The magnitude of effects}

The evidence discussed above at least shows that income mobility may matter and helps to make the case for income mobility (and social mobility in general), much as the case has been amply made for income inequality. After all, income mobility and income inequality are two different, albeit related, dimensions of a social stratification system.

But, how much does social mobility matter? It turns to matter more than trivially. First the magnitude of the coefficients in Tables 3 and 4 are equivalent to changes in life expectancy given a change of one standard deviation of the IM (or GI). Thus, for example, males in communities with IM located one standard deviation below the mean lose on average .60 years of life expectancy (Table 3, GI + IM, second row). This is equivalent to 3 percent of the prevailing LE among low-income males. A modest change but also twice as large (and in the right direction) than the change

\textsuperscript{10}All models we estimated using different specifications. Among them is a specification with state fixed effects which, unsurprisingly, yields weaker, but still significant, effects of income mobility.
Second, arguably income effects on LE are large: on average, the difference between the first and last quartile of the income distribution are of the order of 8 years for males and 5 for females. To compute quantities analogous to these but with more detail we proceed as follows: we simulate the change in life expectancy expected under the third model when there is a shift from the value of the first quartile of IM to the fourth quartile of IM within each income quartile. The average change and associated range per income quartile are plotted in Figure 5a. The maximum differences in LE among males are in the first quartile and ranges between 0.3 and 1.2 years, that is, never larger than 13 percent of the total effect of income. Among females, the impact is lower. Contrast these patterns with Figure 6a, a plot with quantities analogous to those in Figure 5a but using GI instead of IM: the maximum differences here are at most half the size of those associated with changes in IM.

Third, to place upper bounds on the impact of IM we compute quantities analogous to those plotted in Figure 5a but using shifts between the largest and smallest values of IM observed within each income quartile: by how much would LE increase if an average CZ with the lowest values of IM makes a swift transition and becomes a CZ with the highest value of observed mobility in that part of the income distribution? As Figure 5b suggests the largest range for the bottom of the income distribution implies increases in LE between 2.0 and 3.9 years whereas the increases among the richest income quartile range between 0 and 2 years of life. These are nontrivial quantities, at least for the poorest members of the population exposed to the lowest income mobility. If the target is to increase their life expectancy to equal that of members of the richest income quartile the improvement would have to be of the order of 8 years. As much as 62 percent of this change could be attained without income changes at all but through a shift in the income mobility regime from the lowest to the highest observed in the poorest income quartile. Instead, as revealed by Figure 6b, the gains implied by shifts in GI are at their largest (and when in the proper direction), half the magnitude of those associated with IM.

Finally, how much is there to gain in computing zones that are the extremes of LE? Figure 7 displays averages and ranges of change in LE in the ten CZ with the highest and the lowest LE. In CZ that already have low mortality conditions, the gains fluctuate between 0.5 and 2 extra years of life. These represent on average about 1.3 percent of levels of LE already attained. In CZ with
the worst mortality conditions the gains are somewhat larger, between 0.5 and 2.5 or, on average about 2 percent of their present values.

4 Implications

The first lesson we draw from the empirical evidence above is that if population health scientists and policy makers are concerned and intrigued by the effects that increased income inequality may have on health and mortality in the U.S. and elsewhere, they should be equally concerned and intrigued by the effects of waning or static income mobility. Indeed, when compared with income effects, those of income mobility surpass the magnitude of effects associated with income inequality, at least in the sample of small areas we used here. It is true that the gross impact of income is significantly larger than those associated with income mobility. Still, these are nontrivial and certainly larger than attributed to other mortality determinants that command more attention than income mobility.

This lesson is valuable even if, as suggested before, income mobility is a mediator between income inequality and health and mortality, one among many others. If so, and because of the strength of its influence demonstrated before, income mobility should be considered explicitly and jointly with other mediators in the analysis, modeling and estimation of the impact of income inequality on health and mortality.

Clearly, there is a need for more precise models to issue predictions other than the ones we tested here, to account for patterns uncovered before and for others that remain to be discovered. Why, for example, should women mortality be less sensitive to income mobility regimes than males but more sensitive to income inequality instead? Are the patterns revealed above the same for different age groups? Does mortality among younger individuals, aged between 20 and 40 express more acutely the effects of lack of social mobility than mortality at older ages?

Until we know more about the nature of mechanisms involved it is difficult to make precise policy recommendations to better adult life expectancy. The computations we carried out before are only illustrative and designed to identify what may be important and what may not. The counterfactual gains estimated and plotted in Figures 5a, 5b, and 7 can only be used as crude guides because they are based on very stylized models without support from insights about mechanisms. They simply
highlight the potential role of mobility vis-à-vis other determinants. We cannot shift counties across mobility boundaries anymore than we can alter their mortality levels by allocating its population to the top of the income distribution.

It is from highlights produced by empirical estimates and counterfactuals that we draw a second lesson with policy implications. If the models do represent, however crudely, relations in the real world, and if empirical estimates and counterfactuals do not lead us astray, one could argue for the need to target some or all the possible mechanisms that may produce linkages between life expectancy and social mobility, independently of whether or not income mobility is, as many others are, a mediator between income inequality and mortality. These mechanisms involve early upbringing and socialization, crafting of skills that enable individuals to associate returns to extra years of healthy living, and the adoption of attitudes and behaviors that build individual environments with lower exposure and more resiliency. Growing up in a community with a rigid stratification system (with more or less inequality) may discourage those situated in less advantageous positions, erode future outlooks, reinforce mistrust, encourage high rates of discount of the future, and facilitate the adoption of behaviors that provide immediate rewards, some of which are highly noxious, difficult to abandon, and bearers of effects that take a long time to manifest. The policy interventions are then straightforward and no different that those advocated by economists to increase human capital, and they all imply early interventions. If early cognitive and noncognitive skills matter for adult success, they also matter for adult health and not only because adult success breeds good health but because they are the same that need to be sculpted to limit adherence to high-risk behaviors. While one cannot shift the mobility regime of a county overnight, anymore that one can shift its income levels, it is possible to design and implement educational programs that shield individuals and families from the negative outlook that more or less rigid mobility regimes produce.

Lastly, there are also implications for the persistence of health and social inequalities: if current inequality and rigidity in the stratification system induces, via complicated pathways, lower levels of health and higher mortality risks, couldn’t these, then, in turn, breed additional inequality and rigidity? Mortality is just the proverbial tip of the iceberg. Where there are mortality disparities there surely are health status disparities. And these have effects on individuals adult lives that confine and bound the contexts within which younger children are brought up and socialized. There is then a flickering possibility that the effects of social mobility are self-perpetuating and that past
declining trends of social mobility will bring additional declines via health and mortality pathways \textit{ceteris paribus}. 
References


5 Tables and Figures

Table 1: Simplified Theoretical Scenarios

<table>
<thead>
<tr>
<th>Mobility</th>
<th>Inequality</th>
<th>High</th>
<th>Low</th>
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<td>Low</td>
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Table 2: Descriptives Commuting Zone Level

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Figure 1: Inequality and Mobility, Commuting Zones U.S.

(a) Absolute Mobility

(b) Relative Mobility

N = 595; Correlation = −0.67

N = 595; Correlation = 0.48
Figure 2: Life Expectancy at 40 and Inequality / Mobility Measures by Gender at Commuting Zones U.S. (N = 595)

(a) Men Gini Index

(b) Women Gini Index

(c) Men Relative Mobility

(d) Women Relative Mobility

Correlation = −0.47

Correlation = −0.34

Correlation = −0.5

Correlation = −0.31
Figure 3: Life Expectancy at 40 and Inequality by Gender and Income Quartile at Commuting Zones U.S. (N = 595)

(a) Men Q1

(b) Women Q1

(c) Men Q2

(d) Women Q2

(e) Men Q3

(f) Women Q3

(g) Men Q4

(h) Women Q4

Adjusted Life Expectancy at Age 40

Gini Index Within Bottom 99%

Correlation = −0.17

Correlation = 0.01

Correlation = −0.43

Correlation = −0.28

Correlation = −0.44

Correlation = −0.32

Correlation = −0.25

Correlation = −0.26
Figure 4: Life Expectancy at 40 and Relative Mobility by Gender and Income Quartile at Commuting Zones U.S. (N = 595)
Table 3: Predicting Life Expectancy (40) Men by Income Quartile (Commuting Zones)

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</table>

***p < 0.001, **p < 0.01, *p < 0.05. GEE models using an exchangeable correlation structure by state. Robust standard errors. GI = Gini Index, IM = Income Mobility, C = Controls.
Table 4: Predicting Life Expectancy (40) Women by Income Quartile (Commuting Zones)

<table>
<thead>
<tr>
<th>Income Quartile</th>
<th>GI</th>
<th>IM</th>
<th>GI + IM</th>
<th>GI x IM</th>
<th>GI + IM + C</th>
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<td>0.21***</td>
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Num. obs. 586 586 586 586 586
Num. clust. 49 49 49 49 49

***p < 0.001, **p < 0.01, *p < 0.05. GEE models using an exchangeable correlation structure by state. Robust standard errors. GI = Gini Index, IM = Income Mobility, C = Controls.
Figure 5: Gains in LE Due to Changes in IM, Commuting Zones U.S. Median, Percentile 2.5 and 97.5 of Predicted Values

(a) P(25) vs P(75)

(b) Min vs Max
Figure 6: Gains in LE Due to Changes in GI, Commuting Zones U.S. 
Median, 2.5th and 97.5th Percentile of Predicted Values

(a) P(25) vs P(75)

(b) Min vs Max

Difference between p75 (0.34) and p25 (0.26) observed GI

Difference between highest (0.45) and lowest (0.17) observed GI
Figure 7: Gains in LE due to Changes in IM, 10 Commuting Zones U.S. Median, 2.5th and 97.5th Percentile of Predicted Values

(a) High LE Q1 Men

- CO, Glenwood Springs, IM = 18, LE = 79.83
- SD, Mitchell, IM = 29, LE = 80.26
- CO, Cortez, IM = 22, LE = 80.21
- WY, Cody, IM = 20, LE = 80.06
- CO, Steamboat Springs, IM = 18, LE = 80.02

(b) High LE Q1 Women

- MN, Marshall, IM = 25, LE = 84.27
- CO, Glenwood Springs, IM = 18, LE = 86.31
- SD, Mitchell, IM = 29, LE = 86.06
- ND, Bismarck, IM = 24, LE = 85.08
- ND, Jamestown, IM = 24, LE = 84.68

(c) Low LE Q1 Men

- KS, Leavenworth, IM = 33, LE = 74.08
- IN, Terre Haute, IM = 36, LE = 74.01
- NV, Elko, IM = 26, LE = 73.92
- IN, Vincennes, IM = 39, LE = 73.88
- IN, Muncie, IM = 38, LE = 73.69

(d) Low LE Q1 Women

- TX, Littlefield, IM = 39, LE = 77.85
- OK, Guymon, IM = 26, LE = 79.55
- KS, Liberal, IM = 30, LE = 79.5
- IN, Vincennes, IM = 39, LE = 79.45
- IL, Springfield, IM = 38, LE = 79.44
- TX, Midland, IM = 28, LE = 79.22
- NM, Hobbs, IM = 30, LE = 79.21
- TX, Wichita Falls, IM = 33, LE = 79.18
- IN, Terre Haute, IM = 36, LE = 79.02
- TX, Pampa, IM = 28, LE = 78.44
- TX, Littlefield, IM = 39, LE = 77.85