THREE ESSAYS ON WEALTH AND INCOME INEQUALITY AND POPULATION HEALTH IN GLOBAL AND DOMESTIC CONTEXTS

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ABSTRACT

Paul Henry Brodish: Three Essays on Wealth and Income Inequality and Population Health in Global and Domestic Contexts
(Under the direction of Benjamin Mason Meier)

Essay 1 investigates the contextual effect of community-level wealth inequality on HIV serostatus using DHS data pooled from six sub-Saharan African countries. Multilevel logistic regressions relate the binary dependent variable HIV positive serostatus and two weighted aggregate predictors generated from the DHS Wealth Index. A 1-point increase in the cluster-level Gini coefficient and cluster-level wealth ratio is associated with a 2.35 and 1.3 times increased likelihood of being HIV positive, respectively, controlling for individual-level demographic predictors, with larger effects in males. The association is partially mediated by more extramarital partners.

Essay 2 uses multiple cohorts of the National Longitudinal Mortality Study (NLMS) to quantify the absolute income effect on mortality in the United States. Multivariate logistic regressions assess the impact on mortality rate ratios of two hypothetical interventions: lifting everyone living on an equivalized household income at or below the U.S. poverty line in 2000 to the income category just above, and shifting everyone's income by 10–40% to the mean household income, equivalent to reducing the Gini coefficient by the same percentage. The absolute income effect is in the range of a three to four percent reduction in mortality for a 10% reduction in the Gini coefficient. Larger mortality reductions result from larger reductions in the Gini, but with diminishing returns. Inequalities in estimated mortality rates are reduced by a

larger percentage than overall estimated mortality rates under the same counterfactual redistributions.

Essay 3 uses multiple NLMS cohorts and multilevel Cox proportional hazards regressions to estimate the contextual effect of state-level income inequality on premature mortality in the United States. It uses six different measures of state income inequality, controls for inflation-adjusted, equivalized family income, and adjusts for eight individual-level socioeconomic and demographic variables, and for state-level percentage black and percentage in poverty. The contextual effect varies markedly by inequality measure, gender, and regression method. Effect sizes are generally in the range of a one to five percent increase in the likelihood of premature death for a one standard deviation increase in income inequality. The contextual effect may cause a sizeable number of premature deaths, especially among males.

To my mother, Mary Helen Stone Brodish, who embodied the virtues of humility, compassion, empathy, caring, and a keen intellect, with which she quietly influenced countless students and acquaintances alike. She treated everyone, no matter their station in life, with the equal respect that they deserved. She knew nature as a salve for the soul. She lives on in me.

"God is the ultimate combined concepts of goodness, kindness, mercy, and love. It is our purpose in life to aspire to that goodness, kindness, mercy and love in our daily deeds and interactions with others. That is what makes us God-fearing. That is what can make us humane."
– Mary Helen Stone Brodish

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We all stand on the shoulder of giants. Mine are Ichiro Kawachi, who suggested Essay 2, Tony Blakely and Nick Wilson, authors of the New Zealand cohort study, Joseph Stiglitz, who originated the 99%:1% dichotomy which became the rallying slogan for the Occupy Wall Street strategy, Sir Michael Marmot, Sir Anthony Atkinson, Angus Deaton, Stephen Bezruchka, Dennis Raphael, and my committee members, all working vigorously to make a positive difference in the world. While well versed in econometrics and the calculus of predicting the behavior of boundedly rational self-interested actors, when they grill their steaks at night and drink their clean tap water, the gnawing feeling in their stomachs is more than hunger. It must include the dis-ease that comes from knowing that millions of children will go to bed not having eaten anything that evening by virtue of where they were born, many exposed to agents of disease but unexposed to determinants or pre-requisites of health, even in the richest country on earth.

PREFACE

Wealth and income inequality have received increasing attention in recent decades. Since the early 1980's they have steadily increased to extraordinary levels in many societies, particularly among liberal democratic countries such as the United Kingdom and the United States. These increases have important implications for public health, operationalized herein as HIV prevalence (essay one) and premature mortality (essays two and three), in both developing and developed countries. Among the explanations linking income distribution to population health is the absolute income effect: given that health is a function of income (of course, the reverse is also true), there are diminishing marginal returns of income to health [$hi = f(y_i)$, $f' > 0$, $f'' < 0$]. The concave shape of the relationship between income and health predicts that, ceteris paribus, more unequal societies have worse average health. Secondly, the contextual effect of income inequality theorizes an additional negative effect on population health from living in a society with high levels of income inequality [$hi = f(y_i, \text{Gini})$]. For example, when incomes of the top 1% pull away from the rest, they cause a variety of "pollution effects" on the quality of life of the bottom 99% (Kawachi & Subramanian, 2014; Subramanian & Kawachi, 2006; Wagstaff & van Doorslaer, 2000).

Paper one employs the contextual effect to investigate whether neighborhood wealth inequality predicts HIV prevalence in six sub-Saharan African countries with HIV prevalence rates exceeding five percent. This essay follows a line of investigation of the neglected structural drivers of the ongoing AIDS pandemic in sub-Saharan Africa. It was particularly influenced by

the work of Hunsmann, Fox, Parkhurst, Shelton, Shandera and others who have been investigating and tracing the structural drivers of HIV infection and specifically the relationship between wealth distribution and HIV prevalence (Fox, 2010, 2012; Hunsmann, 2009, 2012; Parkhurst, 2010; Shandera, 2007; Shelton, Cassell, & Adetunji, 2005). It was also influenced by my work in the field of program evaluation, in which I have observed how obstacles to addressing structural drivers extend into our approach to and treatment of global health problems. For example, proximate causes of disease and health services remedies tend to have favored status over more distal causes and structural remedies, the former being seen as more measureable, actionable, and more in line with our own approaches to population health.

Essay two was suggested as a dissertation topic by Ichiro Kawachi, M.D., Ph.D., Professor and Chair of the Department of Social and Behavioral Sciences at the Harvard School of Public Health and a leading scholar in health inequities. He indicated that the absolute income effect has not yet been calculated for the United States, and he is "waiting for someone to take it on" (Kawachi, 2014). He is also Co-Editor in Chief, with S.V. Subramanian, Ph.D., of Social Science and Medicine, the target journal. The National Longitudinal Mortality Study (NLMS) is the best available U.S. dataset to answer the specific research question, but the full dataset is not available for public use. A Census Bureau committee chaired by Norman Johnson, Ph.D., Statistics, Yale University, PI of the NLMS, approved the project and assigned a Census Bureau statistician, Jahn K. Hakes, Ph.D., Economics, Duke University, to work closely with me to generate analyses, which I then used in further calculations to generate the final table outputs. Additionally, I used a Public Use Microdata Sample (PUMS) of the full NLMS file (with limited observations and variables compared to the full dataset) to test code and models, which were run on the full dataset. Finally, essay three complements essay two by attempting to identify and

measure for the United States, as accurately as possible and over a five- to ten-year time period to fully account for lag effects on mortality, the contextual effect of state-level income inequality on premature mortality over and above the absolute income effect. It uses a variety of income inequality indices and a novel approach of proportional hazards multilevel modelling to attempt to discern the presence and magnitude of this effect.

The focus on the distribution (of wealth, income, and mortality) in society and concern for equity takes inspiration from the words of Sir Anthony Atkinson, a preeminent scholar on inequality:

> "I was taught, in Cambridge, England, and Cambridge, Massachusetts, to ask, 'Who gains and who loses?' from an economic change or policy. This is a question often missing from today's media discussion and policy debate. Many economic models assume identical representative agents carry out sophisticated decision-making, where distributional issues are suppressed, leaving no space to consider the justice of the resulting outcome. For me, there should be room for such discussion. There is not just one Economics" (Atkinson, 2015) p. 5.

It is important that the third "E" of public policy (Effectiveness, Efficiency, and Equity) get its fair hearing.

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AIC	Akaike information criterion
AIDS	Acquired immunodeficiency syndrome
ASEC	Annual Social and Economic Supplement
CI	Confidence interval
CPS	Current Population Survey
CSDH	Commission on the Social Determinants of Health
DC	District of Columbia
DHS	Demographic and Health Surveys
EITC	Earned income tax credit
ELISA	Enzyme-linked immunosorbent assay
GDP	Gross domestic product
GNI	Gross national income
HR	Hazard ratio
HIV	Human immunodeficiency virus
IMF	International Monetary Fund
Ln	Natural logarithm
MEASURE	Monitoring and Evaluation to Assess and Use Results
MSA	Metropolitan statistical area
NDI	National Death Index
NLMS	National Longitudinal Mortality Study
OECD	Organization for Economic Cooperation and Development
OR	Odds ratio

PAR	Population attributable risk
PPP	Purchasing power parity
PUMS	Public Use Microdata Sample
RR	Relative risk
SD	Standard deviation
SDOH	Social determinants of health
SEA	Statistical enumeration area
SES	Socioeconomic status
SSA	sub-Saharan Africa
STD	Sexually transmitted disease
UNAIDS	Joint United Nations Programme on HIV and AIDS
UNU-WIDER	United Nations University World Institute for Development Economics Research
WHO	World Health Organization

ESSAY 1: AN ASSOCIATION BETWEEN NEIGHBOURHOOD WEALTH INEQUALITY AND HIV PREVALENCE IN SUB-SAHARAN AFRICA[1]

Introduction

The prevailing explanation for extraordinarily high HIV prevalence rates in parts of sub-Saharan Africa (SSA) employs a behavioral paradigm and emphasizes the high rate of concurrent sexual partnerships, although there are strongly opposing viewpoints in the literature regarding the role of the latter (Epstein, 2010; Epstein & Morris, 2011; Lurie & Rosenthal, 2010a, 2010b; Mah & Halperin, 2010a; Mah & Halperin, 2010b; Mah & Shelton, 2011; Morris, 2010; Sawers & Stillwaggon, 2010). Donor countries and international aid agencies have expended enormous effort to try to alter individual sexual behaviors, and only relatively recently has sexual concurrency *per se* been seriously addressed. Throughout the long history of the regional pandemic both donor and recipient countries have largely neglected the contexts and structural drivers of individual sexual behaviors—some have suggested, for political reasons (Hunsmann, 2009). As Paul Farmer notes in *Partner to the Poor*, "the failure to contemplate social and economic aspects of epidemics stunts our understanding of them," making it much more difficult to contain and defeat them (Farmer, 2010; Rosen, 2012).

Although behaviorally-focused prevention appears to have produced recent reductions in HIV incidence rates in the region (Joint United Nations Programme on HIV/AIDS, 2010), it is unclear which interventions have been most effective, nor to what extent. There is clearly a need

[1] This essay previously appeared as an article in the Journal of Biosocial Science. The original citation is as follows: Brodish P.H. "An Association between Neighbourhood Wealth Inequality and HIV Prevalence in sub-Saharan Africa," *Journal of Biosocial Science* 47, no. 3 (May 2015): 311-328.

to better understand the nature and role of network factors such as long-term sexual concurrency, which has been inadequately captured and underreported in sexual behavior surveys, and is at least partially structural in nature because it involves deeply entrenched social and cultural norms (Epstein & Morris, 2011). The heavy toll of the ongoing HIV pandemic in SSA has prompted renewed attention to the social and economic upstream contextual or structural factors, sometimes termed "the causes of the causes" of disease, which may facilitate viral transmission and undermine intervention effectiveness (Commission on Social Determinants of Health, 2008; Gupta, Parkhurst, Ogden, Aggleton, & Mahal, 2008). In a recent supplement to the *Journal of the International AIDS Society* devoted entirely to structural drivers of HIV transmission, Seeley et *al.* (2012) note elimination of HIV will require "a comprehensive HIV response, that includes meaningful responses to the social, political, economic and environmental factors that affect HIV risk and vulnerability" (Seeley et *al.*, 2012).

Also, a prevailing view emphasizes the role of poverty in the spread of HIV, despite numerous studies demonstrating an inverse relationship between HIV serostatus and poverty status in SSA, which is opposite to the case in the developed world and contrary to common expectations about disease susceptibility and poverty status (Gillespie, Kadiyala, & Greener, 2007; Mishra et *al.*, 2007; Parkhurst, 2010; Shelton et *al.*, 2005). Commenting in *the Lancet*, Shelton et *al.* (2005) suggested that both wealth and economic disadvantage may play pivotal roles in HIV transmission through sexual concurrency networks, with wealth being "associated with the mobility, time, and resources to maintain concurrent partnerships" and where women "might improve their economic situation by having more than one concurrent partner" (Shelton et *al.*, 2005) p. 1058. Several investigators have attempted to help resolve the ongoing controversy about the relative importance of poverty, inequality, and sexual concurrency in

explaining the severity of the SSA pandemic. For example, a review by Shandera (2007) identified several viral, host, transmission, and societal factors that might explain the higher rates of infection in the region (Shandera, 2007). A country-level empirical study by Nattrass (2009) identified a number of social factors associated with HIV prevalence rates, finding little effect of poverty but large and significant effects of the predominant religious affiliation of the country (Nattrass, 2009). Within SSA countries, HIV prevalence rates are generally higher in urban compared to rural areas, but there is also much regional variation, with some poorer, rural areas, such as the Nyanza region of Kenya, having very high prevalence rates. Nattrass et *al.* (2012) provides an excellent review of the recent literature on the complex interrelationships among poverty, sexual behavior, and HIV in SSA and the methodological challenges inherent in studies attempting to shed light on them. The authors use a panel dataset on young men in Cape Town, South Africa to overcome problems of endogeneity and blunt indicator measurements of sexual behavior, finding important differences by sex (Nattrass, Maughan-Brown, Seekings, & Whiteside, 2012).

A review by Fox (2010) identified a positive association between HIV prevalence at the country level and the Gini coefficient (a standard measure of economic inequality) among SSA countries (Fox, 2010). These findings suggested a potential association between HIV prevalence and rapid economic development affecting primarily the urban regions of poor developing countries and reflected in rising wealth inequalities, such that it is not poverty or wealth *per se*, but the level of inequality in a region that predicts HIV prevalence. However, cross-country aggregate-level comparisons are prone to problems such as ecologic fallacy or aggregation bias, and to omitted variable bias from the inability to control for many potentially important explanatory factors. Also, if absolute income (or wealth) affects health and there are diminishing

returns to health, then a relationship between health and income is produced at the aggregate level in the absence of a direct effect of economic inequality—the so-called absolute income effect (Gravelle, Wildman, & Sutton, 2002; Kawachi, 2011).

In contrast, the income inequality hypothesis argues that income inequality is an indicator of "social distance" and that greater distance causally leads to greater psychosocial stress and poorer health outcomes (Wilkinson & Pickett, 2009; Wilkinson, 2005; Wilkinson & Pickett, 2006). In the field of economics, this concept implies that "utility" from consumption depends on comparison of one's own income and consumption to that of others, a concept that has gained recent empirical support in behavioral economics (Fliessbach et *al.*, 2007; Luttmer, 2005). Yet a third "society-wide effects" hypothesis argues that the effects of inequality are related to social capital, trust and social cohesion, with increasing inequality causing reduced cohesion and increased crime and violence (Leigh, Jencks, & Smeeding, 2009). Social heterogeneity, or a social context of varying and potentially competing population preferences and needs, has been linked to the under provision of public goods (Banerjee & Somanathan, 2007).

Using Demographic and Health Surveys (DHS) data from 170 regions across 16 countries, Fox (2012) extended her earlier work by employing multilevel modeling techniques to control for regional-level absolute wealth and a number of individual-level HIV risk factors and establishing an independent association between regional-level wealth inequality and HIV prevalence (Fox, 2012). It has also been noted that the geographic level of the community studied might affect the results of an evaluation of the association between wealth inequality and health outcomes, with support in the literature of a general pattern that the smaller the community, the less likely it is that you will observe the association (Wilkinson & Pickett, 2006). Nearly all studies of HIV/AIDS and wealth inequality have been cross-country or regional

comparisons. However, one recent study examined two regions (districts and DHS sampling clusters) simultaneously within one country, Malawi, using a multilevel framework (Durevall & Lindskog, 2012). Specifically, the authors evaluated the effect of district-level consumption inequality and cluster-level (neighborhood) wealth inequality on risk of HIV infection in Malawi women aged 15-24, finding a strong positive association between risk of HIV infection and inequality at both geographic levels, but no association for individual poverty.

The current study builds on these prior efforts by empirically investigating the relationship between wealth inequality at the statistical enumeration area (SEA) or cluster level within multiple countries in southeastern SSA using the most recent DHS data on HIV prevalence and several socio-economic and demographic factors. The advantages of this study are that all data within a given country are from the same survey; the number of data points is much larger than previous country-level studies; it examines the inequality-HIV association at a lower level of aggregation (i.e., at the cluster level, compared to the regional or district level) where it has been harder to detect; and two different measures of SEA wealth inequality are utilized as an internal validation of the key independent variable.

Conceptual model

This paper uses ecological systems theory applied to health (or the social ecological model of health). It views individual health status as determined by a broad array of factors operating at multiple levels, often termed macro-, exo-, meso-, and micro-, which describe influences as intercultural, community, organizational, and interpersonal or individual, and has been adopted by World Health Organization's Commission on the Social Determinants of Health. While this conceptual model applies to general health status, it is utilized here to examine potential influences on specific disease susceptibility. In the developing world the major

threats to population health are infectious disease vulnerability and transmission. HIV is the leading cause of adult mortality in southern SSA and has been responsible for reversing a long-term trend of decreasing mortality rates there. Adult mortality (or, conversely, life expectancy) is a key indicator of population health and directly reflects the general health status of the population.

As a more direct mechanism of action, researchers theorize that rapid economic development is associated with rising wealth inequality and reduced social cohesion, leading to the breakdown of traditional family structures. For instance, new opportunities in urban regions may prompt economic migration by male or female household members. They, and those left behind in rural regions, may then take on informal, long-term partners, leading to higher prevalence of HIV in more unequal settings (Fox, 2012). Durevall et *al.* (2012) note several specific potential links between structural inequality and risk behaviors, particularly transactional sex providing young women and their families the means to remain above subsistence or to improve their economic status (Durevall & Lindskog, 2012). This paper investigates whether HIV prevalence rates are in part determined by such wealth inequities, which reflect differences in social position and levels of social cohesion within a given geographic region (in this case the DHS SEA or cluster), controlling for individual/household wealth and other key individual-level variables.

Methods

Data and sample

A pooled analysis was conducted using DHS household survey data collected since 2006 from six SSA countries with HIV prevalence rates exceeding five percent and HIV biomarker data and data on all covariates. The UNAIDS program classifies a national prevalence rate

higher than 1% as a generalized epidemic (Joint United Nations Program on HIV/AIDS, 2011). Data are downloadable from the MEASURE DHS website at http://www.measuredhs.com/data/available-datasets.cfm. The six countries were: Kenya, Lesotho, Malawi, Swaziland, Zambia, and Zimbabwe. The countries are located in southeastern SSA and have among the highest HIV prevalence rates on the African continent (Table 1.1).

DHS surveys are nationally representative population-based surveys with large sample sizes (usually between 5,000 and 30,000 households). In all households, women age 15-49 are eligible to participate; in many surveys men age 15-54(59) from a sub-sample are also eligible to participate. There are three core questionnaires in DHS surveys: A Household Questionnaire, a Women's Questionnaire, and a Male questionnaire. HIV biomarker data complements self-reported household survey information by providing an objective profile of a HIV status in the population. The sample is usually based on a stratified two-stage cluster design. The first stage is the SEA (or cluster), generally drawn from Census files. In the second stage, within each SEA, a sample of households is drawn selected from an updated list of households. The sample is generally representative at the national level, residence (urban-rural), and regional (departments, states) levels. This paper evaluates regional or community-level factors (i.e., characteristics of the SEA) that may affect HIV prevalence. Admittedly, the SEA is an arbitrary geographic boundary used only for the purposes of the survey, but it is nevertheless based on Census data and can be aggregated proportionally using the DHS sampling weights so that it should remain representative of the populations under study.

This study analyzed the binary dependent variable HIV positive serostatus. The DHS provides anonymous, voluntary testing using blood spots collected on filter paper from a finger prick. An initial ELISA test is performed in the laboratory, with retesting of all positive tests and

5-10 percent of the negative tests with a second ELISA. For those with discordant results on the two ELISA tests, a new ELISA or a Western Blot is performed (Measure DHS, 2012).

Two key independent variables were created aggregated at the SEA level: 1) the Gini coefficient, representing household wealth inequality, which was constructed using a transformation of the DHS wealth index score and a Stata user-provided program called FastGini for calculating a weighted Gini-coefficient; 2) a second inequality index using the categories of the DHS categorical wealth index variable: the ratio of the mean wealth of households in the top 20% wealth quintile to that of those in the bottom 20% quintile. I controlled for several key household- or individual-level characteristics, including household wealth quintile (using the DHS-provided household wealth index), frequency of multiple sexual partnerships during the past year and number of lifetime sexual partners, self-reported sexually transmitted infection in the past year, condom use at last intercourse, and several demographic variables associated with HIV serostatus, though it should be recognized that the sexual behavior "controls" were recognized as potential mediators of the association.

Because they were constructed using principal components analysis, the wealth index scores in the DHS included negative values. Therefore, they had to be transformed in order to make all the values greater than zero in order to calculate a Gini coefficient. Although the most common method for doing so is additive transformation (adding the lowest negative value to make all scores positive), this method has been shown to have distortionary effects on the underlying distribution (Sahn & Stifel, 2003). Therefore, I used an alternative and more reliable exponential transformation of the wealth index scores (Fox, 2012; Wai-Poi, Spilerman, & Torche, 2008).

Empirical model

The final empirical model regressed the dependent variable individual HIV serostatus on the two key community-level independent variables (mean cluster-level Gini coefficient and wealth ratio) separately, and included 12 individual-level control variables: number of sexual partners (other than husband/wife) in the past year (dummy-coded as 0, 1, 2, and 3 or more), lifetime number of sexual partners (coded as 1, 2, 3-5, 6-10, and >10), condom use at last intercourse, self-reported STD in the past year, wealth status, male sex, urban residence, age (in years), education level, employed (currently working, having worked in past year, or on leave in the past 7 days), married or living together, and religious affiliation (Catholic, Protestant/other Christian, Muslim, No/other religion).

Multicollinearity was evaluated by examining the correlation matrix for potential control variables included in the final model. I initially evaluated two sexual risk behavior control variables which measured the number of sexual partnerships in the past year. These two variables were the number of sexual partners 1) other than the spouse and 2) including the spouse, during the past year. Because these two variables were correlated above 0.75, I chose a single measure—number of sexual partnerships (other than the spouse) during the past year—as the one providing the best measure of this construct. I also included a measure of number of lifetime sexual partners.

Analysis

Because the data were nested within clusters and within countries, the assumption of standard logistic regression that respondents are independent within each cluster and that there is equal variance among clusters did not hold. Therefore, a multilevel regression framework was

needed to account for the hierarchical structure of the data. I used a final multilevel logistic regression model of the form:

$$\text{logit}\,[\Pr(HIV_{ijk} = 1|X_{ijk}, \zeta_{jk}, \zeta_k)] = X'_{ijk}\beta + \zeta_{jk} + \zeta_k\ ,$$

where $\zeta_k|X_{ijk} \sim N\ (0, \psi)$ at level 3, and $\zeta_{jk}|X_{ijk}, \zeta_k \sim N\ (0, \omega)$ at level 2. This model assumed random variation in the intercepts across clusters and countries (random intercepts model) but constant slopes for the beta coefficients.

The three levels consisted of 43,091 respondents clustered within 2,641 SEAs across six countries. The analysis proceeded in four steps. First, I ran a null or base model including only the dependent variable HIV prevalence to establish the degree of variance at each of the two higher levels in order to validate use of a multilevel framework. Next, I added the level-1 demographic control variables to the model in order to assess the improvement in model fit and presence of significant effects for individual-level predictors of HIV serostatus. Finally, in each of two separate models, I added the key independent variables wealth Gini coefficient and wealth ratio to test for significance of these two predictors, controlling for the individual-level demographic variables. Finally, I repeated these first three steps using a model that included individual-level measures of sexual risk behaviors serving as potential mediators of the association, in order to look for attenuation of the effects of the inequality measures. Also, because concurrent sexual partnerships are one mechanism linking HIV infection to increased economic inequality, in separate ordered logit models I assessed the pathway of extramarital relationships as the dependent variable to attempt to better understand the potential mechanisms by which inequality might increase HIV prevalence.

Results

There were significant differences by low and high values (above and below the median) for both key independent (predictor) variables considered in the model and for most demographic and sexual behavior variables. Table 1.2 indicates these differences, reporting means and standard errors for continuous variables and counts and percentages for categorical variables. Overall, the mean HIV prevalence rate was 17.3%. The mean percent of households in the lowest wealth quintile was 17.2%; the percent of respondents reporting multiple sexual partnerships in the past year was about 29%; the percent reporting condom use at last sex was 22.5%; and the percent reporting an STD in the past year was only 3.8%. Results were remarkably similar for the two measures of wealth inequality (Gini coefficient and wealth ratio), and most comparisons between low and high groups within these two separate inequality measures were statistically significant and largely in the anticipated direction. In these bivariate analyses, higher cluster-level Gini coefficients and wealth ratios were associated with higher HIV prevalence rates and generally with higher rates of risky sexual behaviors.

The final multilevel regression models are shown in Table 1.3 (for the Gini coefficient) and Table 1.4 (for the wealth ratio), both for women and men combined and separately for women and men. The coefficients and their patterns in the two tables are remarkably similar. Base models (data not shown) including only the dependent variable HIV prevalence rate showed significant variation by country as anticipated from Table 1.1. In Model 1 of both Table 1.3 and Table 1.4, including the key independent variables cluster-level wealth Gini coefficient and wealth ratio, respectively, and individual-level demographic control variables, both inequality measures were associated with an increased likelihood of being HIV positive. Male sex was protective, while urban residence, age, and being employed were associated with a

slightly increased likelihood of being HIV positive. Compared to unmarried persons (the referent group), those who were married or living together were at higher risk and those not living together were at much higher risk of being HIV positive. Including the cluster-level wealth Gini coefficient and wealth ratio in Model 1 significantly improved the model fit (significant log likelihood ratio test) relative to base models. Both the cluster-level wealth Gini coefficient (OR = 2.35, $p < 0.05$) and the wealth ratio (OR = 1.32, $p < 0.01$) were associated with a significant increase in the likelihood of being HIV positive. The marginal effect of the Gini coefficient was that a 1 point increase in the Gini coefficient of an SEA cluster was associated with a 2.35 times increased likelihood of being HIV positive, controlling for all other variables in the model. Similarly, a 1 point increase in the wealth ratio was associated with a 1.3 times increased likelihood of being HIV positive, controlling for the other variables in the model.

Adding in the sexual behavior variables in Model 2 of Tables 1.3 and 1.4 attenuated the effects of both measures of wealth inequality. There was a dose-dependent increase in the odds of being HIV positive with more lifetime sexual partners such that reporting 11 or more partners increased the likelihood of being HIV positive over five-fold. Condom use at last intercourse and an STD in the past year increased this likelihood by two and almost two and-a-half times, respectively. (Note that because this analysis is correlational, endogeneity or reverse causality probably explains the former association, i.e., condom use is likely to be more frequent among those who know they are HIV positive and/or who engage in higher-risk sex.)

Looking at Tables 1.3 and 1.4, Models 3 and 4 for women only, some interesting findings are evident. In both tables the odds ratios decrease slightly but remain significant. Comparing Model 1 to Model 3 in both tables, the odds ratio for married/living together (compared to the referent group unmarried) reverses and becomes less than 1 for women, indicating a protective effect for women

who are married to or living with their partner, controlling for age (which becomes non-significant in Table 1.3). More education among women appears to be slightly protective (decreased odds of HIV infection), whereas it appears to increase risk in the combined men-women models. Also, the odds of HIV infection associated with not living together reduces (from 4.3 to 2.9 times) compared to that for men and women combined. Comparing the full models incorporating the risk behaviors (Model 2 compared to Model 4), again age becomes non-significant and the odds ratio for married/living together again becomes greater than 1, suggesting that risk behaviors are mediating some of this effect and removing any protection associated with cohabitation with sexual partners for women.

Looking at Tables 1.3 and 1.4, Models 5 and 6 for men only, odds ratios for the Gini coefficient and wealth ratio are larger for men. Age is a significant predictor in both reduced and full models, and education appears to increase, rather than decrease risk, although it becomes non-significant in the full models (Model 6 in both tables). It is apparent that the risks associated with cohabitation and not living together (compared to the unmarried referent group) in the combined men-women model is driven by males. The coefficients increase from Model 5 to Model 6 for married/living together for males, suggesting that some other factors increasing risk are not being fully picked up by the risk behaviors.

Results from the ordered logit models predicting numbers of extramarital partners in the past year (Table 1.5) indicated that the likelihood of having more extramarital partners was higher in clusters with more wealth inequality, although this relationship was significant only for the model including the wealth ratio predictor variable (OR = 1.27, $p < 0.001$). Not surprisingly, marriage/cohabitation appeared to be highly protective in all ordered logit models (OR = 0.01, $p < 0.001$), while being a younger male appeared to increase risk by over 4-fold. Looking at these models by sex indicated several interesting differences. The significant result for wealth ratio

held for women (OR = 1.54, $p < 0.001$) but not for men (OR = 1.15); age was not a significant factor in the models for women but was significant and protective in the models for men; urban residence was a significant risk factor for women but not for men; among women, primary education appeared to be a significant risk factor, while a secondary or higher education was no longer significant (compared to models for women and men combined). The risk associated with secondary or higher education appeared to work in opposite directions for men compared to women, reducing risk for women and increasing it for men. Religion was not a significant factor among women but was among men. Among men, being of Protestant or other Christian faith (compared to Catholic) significantly reduced risk, while being of Muslim faith increased it.

Discussion

The relationships between HIV prevalence and the control variables were all in the anticipated direction based on previous studies and expectations about HIV risk and demographic and sexual variables operating at the individual level. Both the cluster-level Gini coefficient for household wealth and the wealth ratio were significant predictors of HIV serostatus, controlling for all other variables in the models, including household wealth and several known behavioral and demographic predictors of positive serostatus. This is the second known study to produce empirical evidence of these effects using multiple countries and regions in SSA, and the second to demonstrate this effect at the DHS cluster level by utilizing its inherent population-based survey sampling strategy. Although a large literature suggests that economic inequality increases the risk for a variety of diseases after controlling for absolute levels of wealth or income (Subramanian & Kawachi, 2004; Wilkinson & Pickett, 2009; Wilkinson, 2005), very few have demonstrated it in the context of infectious disease in developing countries. Similarly to these two prior studies (Durevall & Lindskog, 2012; Fox, 2012), there is a persistent association

between regional/district- and/or neighborhood/cluster-level wealth inequality and HIV serostatus after controlling for household-level wealth. Also consistent with these two prior studies, results from models with extramarital partners as the dependent variable suggest that the mechanism is at least in part mediated by an increase in risky sexual behavior.

Consistent with the one prior study of this association at the DHS cluster level in Malawi (Durevall & Lindskog, 2012) but contrary to findings from national-level studies, household wealth was not significantly associated with HIV positive serostatus. This result could be explained by more recent evidence pointing to a complex, dynamic association between wealth and HIV serostatus in SSA. Parkhurst (2010) found that, at the country level, as per capita GDP increased, the confirmed trend for the prevalence of HIV infection to increase with increasing wealth quintile dissipated. He identified a threshold of approximately U.S.$ 2,000 above which this tracking with wealth becomes inconsistent. Half of the countries in the current sample exceeded this GDP threshold. Furthermore, Parkhurst's analysis of trend data from two Tanzania DHS studies suggested that HIV has become more prevalent in poorer individuals as development has progressed (Parkhurst, 2010). Similarly, Fox (2012) found that in poorer regions/countries, individuals with more wealth were more likely to be infected, whereas in wealthier regions/countries, individuals with less wealth were more likely to be infected with HIV. She noted that these results, combined with those of prior supportive studies, suggest a changing social gradient with increasing wealth and development in which, due to increasing knowledge, prestige, and power, wealthier segments of the population are better able to adapt to new health threats, socially reproducing health inequalities via social stratification mechanisms (Fox, 2012; Phelan, Link, & Tehranifar, 2010).

The null results for the major religious affiliations could be explained in that most national studies have looked at national-level religious affiliation, and most African Muslim nations are supra-Saharan, where HIV prevalence rates are much lower. Muslims comprised a very small segment of this pooled sample (less than 5%) so the power to detect a significant effect was reduced. However, the direction of the odds ratios suggested a slight protective effect for Muslim religion. The significant effect for "none/other" religious affiliation, which is attenuated with the addition of the sexual behavior variables to the models, is consistent with the possibility of increased risky-taking behaviors in this minority group in association with lower social status or differential treatment based on religion.

Disaggregation by sex indicated that the relationships between community wealth inequality and HIV positive status were stronger for males. However, in the ordered logit models predicting risk behavior (number of extramarital partners in the past year), the wealth ratio was a significant predictor only in models for women. Primary education was a risk, and age was not a risk in models for women (these two reversed in the models for males). Although causality cannot be inferred from these results, taken as a whole, they could be interpreted as suggesting a situation in which neighborhood wealth inequalities, particularly in urbanized areas, are associated with, and possibly promoting, increased extramarital sexual relationships by women of childbearing age, who are able to trade sex for money or resources provided by men in households with greater wealth, thus escalating HIV risk within sexual networks. The positive relationship for women with a primary education could reflect increased contact in urbanized areas, through the educational system, with men seeking extramarital partnerships, with women increasingly engaging in such relationships in contexts of high wealth inequalities but less so as they achieve higher levels of education and a trajectory leading to greater autonomy and

financial independence. These results point to the importance of disaggregating by sex in such analyses and exploring potential mechanisms of action/causal pathways through modeling behavioral mediators. They are also consistent with the Nattrass et al, 2012 panel study, in which household assets were negatively correlated with HIV status for women but not for men, and in which HIV-positive women were much less likely to have made the transition from (primary) school to tertiary education, a transition which would place them on a trajectory of lowered risk (Nattrass et *al.*, 2012).

It is important to note several study limitations. One methodological limitation is the assumption of constant slopes on the beta coefficients for the two key independent variables. However, adding random slopes to the models would have introduced greater complexity and was not necessary for the purposes of the analysis, and the limited number of countries (six) at level 3 precluded adding this feature at that level. Another likely limitation is violation of basic ordinary least squares logistic regression assumptions through inadequate model specification and the presence of omitted variables, which would bias the beta estimate for the key independent variables in the direction of a type-1 error. Clearly the final regression model did not capture all variables affecting HIV prevalence at multiple levels and may conflate to some degree mediation and confounding, but it did capture important available individual-level predictors, and by adding potential behavioral mediators last, I was able to assess the extent to which some variables might mediate the observed effects of the independent variables. Endogeneity in the independent variables or omission of a "left-out common cause" of both wealth inequality and HIV prevalence in which an omitted variable is associated with both cluster-level wealth inequality and with HIV prevalence, or other forms of omitted variable bias, are also a potential sources of bias in this analysis. Also, the DHS Wealth Index used to construct

these independent variables has limitations which may lead to erroneous conclusions regarding the direct effect of wealth on HIV status. Because different assets are used in each country to construct the index (although a basic set of assets, such as type of flooring, water supply, sanitation facilities, appliances, transportation, etc. are included in every survey), it is not directly comparable across countries. Also, the index is not the best proxy for consumption expenditure, the SES measure preferred by economists (Howe, Hargreaves, Gabrysch, & Huttly, 2009). It also tends to negatively weight assets from traditional forms of subsistence production and over-weight assets obtained in the modern cash economy, and thus tends to capture involvement in the modern, cash-oriented economy, which is also highly correlated with both urbanization and education level (Bingenheimer, 2007). This property may help explain its consistent positive association with HIV status among poorer developing African countries. However, the DHS index is considered a reasonable measure of economic well-being, it is the measure that was available in the datasets, and its major purpose was to construct within-cluster relative measures of economic inequality and to control for absolute measures of individual wealth status rather than to compare wealth status across countries.

Also, because these data are cross-sectional, we can only observe the relationship between wealth inequality and HIV prevalence at a single point in time. Reverse causality, in which HIV infection affects household wealth and cluster-level wealth distribution, is undoubtedly present. Although the sexual behavior variables were somewhat weak measures of HIV risk behavior (with floor effects and apparent underreporting), a dose-response relationship for lifetime sexual partners suggested that variance associated with this risk factor was captured. Future studies should try to identify more valid measures of behavioral risk and to assess these factors as potential individual-level mediators in a multi-level modeling framework. Also, these

results point to some of the difficulties in doing empirical work in the field of social/structural determinants of health. It is often difficult to relate macro-level social factors to individual health status due to unavailability of accurate measures of both micro- and macro-level factors, and to the complexity of methodologies needed to adequately control for factors operating at multiple levels. Nevertheless, neglect of these higher-level factors moderating individual behaviors risks ascribing too much predictive power to micro-level factors and may lead to missed opportunities to modify social environments and create structural changes which induce more health-supportive behaviors.

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TABLES

Table 1.1. Country sample, sub-Saharan Africa, DHS 2006-2011

Country	HIV prevalence (CI)[a]	Tested sample	Year of survey	Refusal rate (%)	GNI per capita (2010)[b]	GDP growth rate (2010)[c]	Gini ratio (most recent year)[d]
Kenya	6.4 (5.4– 7.4)	6,906	2008/9	10.8	1640	5.6	41.9
Malawi	10.7 (9.8–11.7)	13,927	2010	6.5	860	7.1	39.0
Zambia	14.2 (13.1–15.4)	10,873	2007	20.0	1380	7.6	50.8
Zimbabwe	15.3 (14.4–16.2)	13,897	2010/11	14.1	—	9.0	56.6
Lesotho	23.0 (21.5–24.5)	6,924	2009	4.7	1970	5.6	58.1
Swaziland	25.9 (24.5–27.5)	8,187	2006/07	12.8	5600	2.0	50.4

[a] Countries ordered from lowest to highest prevalence. Figures weighted for probability of selection into the sample.
[b] GNI per capita, PPP (purchasing power parity, current international $), 2010. Zimbabwe's data missing due to the ongoing political and economic crisis; data from World Bank, World Development Indicators Database.
[c] Annual GDP growth rate for 2010. Data from World Bank, World Development Indicators Database.
[d] Gini data from UNU-WIDER World Income Inequality Database, Version 2.0c, May 2008.

Table 1.2. Summary statistics for variables included in final model, by low and high Gini coefficient and wealth ratio

		Gini coefficient		Wealth ratio	
	Total	**Low**	**High**	**Low**	**High**
n	43,032[a]	21,520	21,512	21,522	21,509
Dependent variable					
HIV positive, n (%)	7,444 (17.3)	3,395 (15.8)	4,048 (18.8)***	3,347 (15.6)	4,097 (19.0)***
Independent variables					
Gini coefficient, mean (SE)	0.42 (0.004)	0.28 (0.003)	0.56 (0.003)***	0.34 (0.006)	0.50 (0.004)***
Wealth ratio, mean (SE)	1.49 (0.008)	1.35 (0.009)	1.62 (0.012)***	1.23 (0.003)	1.74 (0.011)***
Demographic variables					
Age in years, mean (SE)	30.8 (0.06)	31.1 (0.09)	30.5 (0.08)***	31.1 (0.09)	30.5 (0.08)***
Male, *n* (%)	20,403 (47.4)	10,337 (48.0)	10,066 (46.8)*	10,421 (48.4)	9,982 (46.4)***
Urban residence	12,273 (28.5)	7,808 (36.3)	4,465 (20.8)***	8,540 (40.0)	3,733 (17.4)***
Working	30,916 (71.8)	15,893 (73.8)	15,023 (69.8)***	15,954 (74.1)	14,962 (69.6)***
Education					
None	3,348 (7.8)	1,993 (9.3)	1,355 (6.3)***	1,591 (7.4)	1,757 (8.2)
Primary	20,418 (47.4)	10,105 (47.0)	10,313 (47.9)	9,611 (44.6)	10,807 (50.2)***
Secondary+	16,650 (38.7)	7,699 (35.8)	8,951 (41.6)***	8,651 (40.2)	7,998 (37.2)**
Wealth quintile					
Lowest	7,380 (17.2)	4,819 (22.4)	2,561 (11.9)***	3,504 (16.3)	3,876 (18.0)
Second	7,940 (18.4)	3,953 (18.4)	3,988 (18.5)	3,739 (17.4)	4,201 (19.5)*
Middle	8,386 (19.5)	2,892 (13.4)	5,494 (25.5)***	3,826 (17.8)	4,560 (21.2)***
Fourth	9,284 (21.6)	3,429 (15.9)	5,855 (27.2)***	4,352 (20.2)	4,932 (22.9)*
Highest	10,041 (23.3)	6,428 (29.9)	3,613 (16.8)***	6,101 (28.4)	3,940 (18.3)***
Marital status					
Unmarried	8,363 (19.4)	3,935 (18.3)	4,428 (20.6)**	3,624 (16.8)	4,739 (22.0)***
Married/living together	31,888 (74.1)	16,260 (75.6)	15,628 (72.6)***	16,504 (76.7)	15,384 (71.5)***
Not living together	2,780 (6.5)	1,325 (6.2)	1,455 (6.8)*	1,394 (6.5)	1,386 (6.4)
Religion					
Roman Catholic	8,369 (19.4)	4,452 (20.7)	3,917 (18.2)**	4,149 (19.3)	4,220 (19.6)
Protestant/other Christian	27,131 (63.0)	13,961 (64.9)	13,170 (61.2)***	14,578 (67.7)	12,553 (58.4)***
Muslim	1,793 (4.2)	947 (4.4)	846 (3.9)	818 (3.8)	975 (4.5)
None/other	5,739 (13.3)	2,160 (10.0)	3,579 (16.6)***	1,978 (9.2)	3,762 (17.5)***
Sexual behavior variables					
No. extramarital sex partners past year					
0	30,684 (71.3)	15,661 (72.8)	15,023 (69.8)**	15,979 (74.2)	14,705 (68.4)***
1	10,463 (24.3)	5,007 (23.3)	5,456 (25.4)**	4,722 (21.9)	5,741 (26.7)***
2	1,531 (3.6)	669 (3.1)	862 (4.0)***	645 (3.0)	886 (4.1)***
3+	354 (0.8)	183 (0.9)	171 (0.8)	177 (0.8)	177 (0.8)
No. lifetime sex partners					
1	13,653 (31.7)	6,809 (31.6)	6,844 (31.8)	6,918 (32.1)	6,735 (31.3)
2	9,946 (23.1)	4,960 (23.0)	4,986 (23.2)	4,888 (22.7)	5,058 (23.5)
3 - 5	12,339 (28.7)	6,083 (28.3)	6,256 (29.1)	6,018 (28.0)	6,321 (29.4)*
6 - 10	3,864 (9.0)	2,027 (9.4)	1,837 (8.5)*	2,028 (9.4)	1,836 (8.5)*
11+	3,229 (7.5)	1,641 (7.6)	1,588 (7.4)	1,670 (7.8)	1,559 (7.2)
Condom use last sex	9,697 (22.5)	4,477 (20.8)	5,220 (24.3)***	4,315 (20.0)	5,382 (25.0)***
STD past year	1,624 (3.8)	738 (3.4)	887 (4.1)**	717 (3.3)	907 (4.2)***

[a] Estimated population size adjusted for survey sampling frame.

*p < 0.05 **p < 0.01 ***p < 0.001.

Table 1.3. Parameter estimates[a] (odds ratios) for three-level models predicting HIV positive status, Gini coefficient

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Fixed effects						
Community (cluster) characteristic						
Gini coefficient	2.35*	2.04*	2.19*	1.88*	2.49*	2.27*
Individual demographic characteristics						
Age (years)	1.03***	1.02***	1.02	1.01	1.04***	1.03***
Sex (male)	0.69***	0.39***	—	—	—	—
Residence (urban)	1.85***	1.64***	1.91***	1.67***	1.60***	1.50**
Working	1.19**	1.06	1.18**	1.05	1.13	1.06
Education						
None (Ref.)						
Primary	1.25**	1.22*	1.32*	1.32*	1.15	1.09
Secondary+	1.31**	1.25*	1.28*	1.27	1.28*	1.17
Marital status						
Unmarried (Ref.)						
Married/living together	1.36*	2.16***	0.82**	1.25*	2.42***	3.71***
Not living together	4.31***	3.73***	2.90***	2.36***	6.57***	6.19***
Wealth quintile						
Lowest (Ref.)						
Second	1.07	1.07	1.06	1.05	1.11	1.10
Middle	1.08	1.08	1.10	1.08	1.08	1.09
Fourth	1.12	1.12	1.19	1.21	1.12	1.07
Highest	1.01	1.01	1.03	1.03	1.12	1.06
Religion						
Roman Catholic (Ref.)						
Protestant/other Christian	1.02	1.02	1.03	1.01	1.02	1.04
Muslim	0.93	0.89	1.02	0.95	0.95	0.90
None/other	1.22***	1.13	1.28*	1.22	1.22***	1.12
Individual sexual behaviors						
No. extramarital partners past year						
0 (Ref.)						
1	—	1.15	—	1.00	—	1.14
2	—	0.84	—	0.75	—	1.06
3+	—	0.81	—	1.33	—	0.96
No. lifetime sex partners						
1 (Ref.)						
2	—	2.39***	—	2.53***	—	2.24***
3 – 5	—	3.64***	—	3.95***	—	3.31***
6 – 10	—	4.47***	—	5.11***	—	4.00***
11+	—	5.38***	—	4.49***	—	4.92***
Condom use last intercourse	—	2.06***	—	2.21***	—	2.09***
Any STD past year	—	2.41***	—	2.46***	—	2.57***
Random effects						
Cluster-level (level 2) variance	0.50	0.45	0.38	0.37	0.55	0.53
Country-level (level 3) variance[b]	2.04***	1.84***	0.56***	0.28***	0.51***	0.41***
Log likelihood	-17545.25	-16636.01	-9990.919	-9344.24	-7607.446	-7297.367
AIC	35128.51	33328.03	20017.84	18742.48	15250.89	14648.73
Log likelihood Ratio Test (X^2)[c]	34.69***	27.21***	24.18***	14.44***	22.24***	17.64***

[a] Parameters for predictors (fixed effects) are reported as odds ratio; for random effects, the parameter is the variance.

[b] Significance of random effects evaluated by comparing model with a similar one in which random effects have been constrained to be zero.

[c] Compared with model excluding the independent variable.

*p<0.05 **p<0.01 ***p<0.001

Table 1.4. Parameter estimates[a] (odds ratios) for three-level models predicting HIV positive status, wealth ratio

	Men and women		Women only		Men only	
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Fixed effects						
Community (cluster) characteristic						
Wealth ratio	1.32**	1.30***	1.25**	1.24**	1.36**	1.35***
Individual demographic characteristics						
Age (years)	1.03***	1.02***	1.02***	1.01	1.04***	1.03***
Sex (male)	0.69***	0.39***	—	—	—	—
Residence (urban)	1.83***	1.63***	1.88***	1.66***	1.59***	1.49***
Working	1.19**	1.07	1.18**	1.06	1.13	1.06
Education						
None (Ref.)						
Primary	1.26**	1.23**	1.33*	1.33*	1.17	1.11
Secondary+	1.32**	1.26*	1.29*	1.28*	1.30**	1.19
Marital status						
Unmarried (Ref.)						
Married/living together	1.36*	2.15***	0.82**	1.24*	2.42***	3.72***
Not living together	4.32***	3.74***	2.90***	2.36***	6.59***	6.22***
Wealth quintile						
Lowest (Ref.)						
Second	1.10	1.10	1.10	1.09	1.17	1.15
Middle	1.15	1.14	1.18	1.15	1.19	1.19
Fourth	1.18	1.18	1.28	1.28	1.22	1.16
Highest	1.04	1.04	1.07	1.06	1.17	1.10
Religion						
Roman Catholic (Ref.)						
Protestant/other Christian	1.03	1.03	1.04	1.01	1.02	1.04
Muslim	0.93	0.89	1.02	0.95	0.94	0.89
None/other	1.22***	1.13*	1.28*	1.22	1.23***	1.13
Individual sexual behaviors						
No. extramarital partners past year						
0 (Ref.)						
1	—	1.14	—	1.00	—	1.14
2	—	0.84	—	0.74	—	1.07
3+	—	0.81	—	1.29	—	0.96
No. lifetime sex partners						
1 (Ref.)						
2	—	2.39***	—	2.53***	—	2.25***
3 – 5	—	3.66***	—	3.97***	—	3.33***
6 – 10	—	4.49***	—	5.13***	—	4.02***
11+	—	5.40***	—	4.51***	—	4.96***
Condom use last intercourse	—	2.06***	—	2.22***	—	2.11***
Any STD past year	—	2.41***	—	2.47***	—	2.56***
Random effects						
Cluster-level (level 2) variance	0.51	0.45	0.39	0.37	0.56	0.53
Country-level (level 3) variance[b]	1.93***	2.13***	0.77***	0.33***	0.50***	0.41***
Log likelihood	-17553.54	-16640.29	-9998.201	-9346.904	-7612.116	-7299.98
AIC	35145.08	33336.57	20032.4	18747.81	15260.23	14653.96
Log likelihood Ratio Test (X^2)[c]	18.12***	18.67***	9.62**	9.12**	12.90***	12.41***

[a] Parameters for predictors (fixed effects) are reported as odds ratio; for random effects, the parameter is the variance.

[b] Significance of random effects evaluated by comparing model with a similar one in which random effects have been constrained to be zero.

[c] Compared with model excluding the independent variable.

*p<0.05 **p<0.01 ***p<0.001.

Table 1.5. Parameter estimates[a] (odds ratios) for three-level ordered logit models predicting number of extramarital partners in the past year

	Men and women		Women only		Men only	
Explanatory variables	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**
Fixed effects						
Community (cluster) characteristics						
Gini coefficient	1.41	—	1.29	—	1.48	—
Wealth ratio	—	1.27***	—	1.54***	—	1.15
Individual demographic characteristics						
Age (years)	0.99**	0.99**	1.01	1.01	0.97***	0.97***
Sex (male)	4.41***	4.41***	—	—	—	—
Residence (urban)	1.27***	1.29***	1.68**	1.80**	1.12	1.12
Working	1.44***	1.45***	1.49**	1.51***	1.39***	1.39***
Education						
None (Ref.)						
Primary	1.06	1.07	1.39***	1.37***	0.95	0.96
Secondary+	1.14*	1.14*	1.08	1.06	1.11	1.12
Marital status						
Unmarried (Ref.)						
Married/living together	0.01***	0.01***	0.01***	0.01***	0.01***	0.01***
Not living together	0.25***	0.25***	0.08***	0.08***	0.41***	0.41***
Wealth quintile						
Lowest (Ref.)						
Second	0.93	0.95	1.22	1.25	0.85	0.87
Middle	1.00	1.04	1.28	1.33	0.93	0.98
Fourth	0.99	1.02	1.23	1.27	0.92	0.96
Highest	1.07	1.09	1.10	1.12	1.10	1.12
Religion						
Roman Catholic (Ref.)						
Protest./other Christian	0.91**	0.91**	1.00	1.01	0.87***	0.87***
Muslim	1.17	1.16	1.15	1.15	1.15**	1.14**
None/other	1.10	1.10	1.11	1.11	1.10	1.11
Random effects						
Cluster-level (level 2) variance	0.24	0.24	0.50	0.49	0.32	0.32
Country-level (level 3) variance[b]	0.30***	0.33***	0.61***	0.57***	0.26***	0.32***
Log likelihood	-15059.09	-15054.39	-4139.389	-4131.821	-10607.86	-10608.94
AIC	30160.19	30150.79	8318.779	8303.643	21255.73	21257.88

[a] Parameters for predictors (fixed effects) are reported as odds ratio; for random effects, the parameter is the variance.

[b] Significance of random effects evaluated by comparing model with a similar one in which random effects have been constrained to be zero.

*p<0.05 **p<0.01 ***p<0.001

ESSAY 2: QUANTIFYING THE INDIVIDUAL-LEVEL ASSOCIATION BETWEEN INCOME AND MORTALITY RISK IN THE UNITED STATES USING THE NATIONAL LONGITUDINAL MORTALITY STUDY

Occurrent, quod genus egestatis gravissimum est, in divitiis inopes.[2]

Introduction

Evidence to date, albeit from a relatively limited number of experimental or quasi-experimental studies, supports the conclusion that income poverty has an adverse causal impact on health. In a recent review of the evidence from social epidemiology, Glymour and colleagues discuss the difficulties of establishing a causal relationship due to reverse causation and to factors jointly determining income and health (Glymour, Avendano, & Kawachi, 2014). There is a tendency for social epidemiologists to view the relationship as causal from income to health: more income means greater ability to purchase good housing, food, clothing, and other health-generating goods. But those employing a health capital theoretical orientation stemming from human capital theory or the Grossman model (Grossman, 1972) tend to see the causal relationship going from health to income, or at least as more bi-directional. Particularly useful in understanding relationships between employment and health, the Grossman model views individuals as both consumers and producers of health: health is both a consumption good providing direct utility, and an investment good increasing productivity, reducing time in

[2] "To be poor in a wealthy society is the worst kind of poverty." Lucius Annaeus Seneca. *Ad Luclilum epistulae morales*: Epistle LXXIV (c. 4 BC-AD 65). The Roman stoic philosopher Seneca the Younger died a "tripartite death" once Nero turned against him: vein opening, hemlock draft and then asphyxiation in a hot bath. In *De Ira* he writes: "You ask what is the path to freedom? Any vein in your body." This contrasts with Sen's conception of freedom.

sickness, and increasing income. The individual derives utility from his/her health stock, consumption of other goods, and leisure time.

Experimental and quasi-experimental studies addressing the income health relationship have fallen into two main categories. First, randomized experiments assign one group of individuals or households to some form of income transfer, compared to a control group not assigned a transfer. Second, natural experiments assign income exogenously or "as-if-random" due to such "shocks" as a change in entitlement laws, lottery winnings, or stock market windfalls. Overall, the results of this literature suggest that policy-induced change via conditional cash transfers that require human capital investments have significant positive health effects on mothers and children, and that income shocks in the first five years of life, compared to other points in the life course, have the largest effects on health (Glymour et *al.*, 2014). Long-term or permanent increases in income appear to yield positive health benefits, based on evaluations of intermediate outcomes such as child developmental milestones and school enrollment, but there is a need for evidence on long-term, sustained health improvements. Additionally, total costs of programmatic interventions must be considered, because in the final analysis, if income improves health, then the cost of potential health interventions of any description must be weighed against the alternative of simply giving program beneficiaries the money directly, as with social cash transfers (Glymour et *al.*, 2014).

Less well appreciated than the income-health relationship, many chronic diseases that manifest clinically in adulthood have antecedents traceable to impoverished circumstances during early childhood. Further, these effects produce continuing, accumulating, and lasting detriments to health (Case, Lubotsky, & Paxson, 2002; Gluckman, Hanson, Cooper, & Thornburg, 2008; Hertzman, 1999; Shonkoff, 2010; Shonkoff, Boyce, & McEwen, 2009;

Shonkoff & Levitt, 2010) and are targetable through early interventions (Mercy & Saul, 2009). In particular, Shonkoff and colleagues have developed a *biodevelopmental framework* for understanding the origins of disparities in learning, behavior, and health based on evolutionary biology and recent scientific advances in understanding of gene-environment interactions. He cites a 2007 *Lancet* series on child development in low-income countries, which estimates that 200 million children worldwide are not reaching their cognitive potential due to deep poverty and its associated toxic stress (Grantham-McGregor et *al.*, 2007; Shonkoff, 2010). In the United States, with a childhood poverty rate of nearly 20 percent, there is ample scope for moving public policy forward to close "the gap between what we know and what we do to promote the healthy development of young children" (Shonkoff, 2010).

The above framework is necessarily highly interdisciplinary. Raphael reviews the large number of disciplinary perspectives involved in understanding the association between poverty in childhood and adverse health outcomes in adulthood, and in doing something about it (Raphael, 2011). These disciplinary perspectives include political science, political economy, sociology, and institutions (e.g., the coevolution and collective action literature), but also societal discourses/explanations/rationalizations to justify tolerating such high levels of childhood poverty and its adverse health effects into adulthood. Other disciplines or theoretical perspectives from which to view the study of the adverse health effects of early adverse experiences, and the structural determinants of health more broadly, include economic theory; normative theory—social justice, equity, egalitarianism, human rights; developmental psychology/psychopathology and neuroscience; and social epidemiology. Further, in a discussion of mechanisms to promote health equity, the close kinship among the most prominent disciplines or fields of scholarship engaged in this work—social epidemiology, social medicine, and health and human rights—

should be recognized and promoted. It should also be mentioned that the development economist Amartya Sen was instrumental in setting up the World Health Organization Commission on Social Determinants of Health (WHO CSDH) and was one of its members. There is a close affinity between his ethical arguments for viewing social justice as the expansion of freedoms and social epidemiological research on the effects on health of having control, autonomy, and full participation in social relationships (Sen, 2000, 2008; Venkatapuram, Bell, & Marmot, 2010).

Three general theoretical frameworks provide orientation to how social conditions influence health. First, the materialistic/structuralist framework emphasizes the material conditions under which people live and is common to all three frameworks. These conditions include the availability of sufficient material resources in early childhood to assure adequate educational opportunities, food and housing, etc., largely determined by employment security and the quality of working conditions and wages. Second, the neo-materialistic framework additionally examines societal factors that determine the quality of the distribution of the social/structural determinants of health (SDOH), i.e., how a society decides to *distribute* resources among citizens. Finally, the psychosocial comparison framework, in addition to incorporating the materialistic/structuralist framework, examines position in the social hierarchy and includes mechanisms at two levels. At the individual level, perception/experience of one's status/class position in unequal societies causes stress and poor health through harmful effects on neuroendocrine, autonomic, metabolic, and immune systems (Brunner & Marmot, 2006). Also, comparisons to those of a higher social class can lead to overconsumption/taking on debt, overworking to increase income, and harmful coping behaviors such as overeating and using alcohol and tobacco (Kawachi & Kennedy, 2002). At the communal level, widening and

strengthening of hierarchy weakens social cohesion, which is a determinant of health (Kawachi & Kennedy, 1997). These psychosocial comparison mechanisms are secondary to how societies distribute material resources and provide security to citizens (the materialist and neo-materialist mechanisms).

The tendency is to individualize these issues: e.g., governments or other authorities may view early life as being primarily about parental behaviors toward their children and then try to promote better parenting or particular school-based programs, rather than providing more resources to families. For every SDOH an individualized manifestation of each is available, but there is little evidence for the efficacy of such approaches in improving the health status of those most vulnerable to illness in the absence of efforts to modify their adverse living conditions (Cooper, Cooper, McGinley, Fan, & Rosenthal, 2012; Raphael, 2011). There is also the tendency to use health care services to address these issues—to equate health with health care. Liberal democratic countries like Canada and the United States are among the biggest spenders on health care services. Yet, based on many international comparative measures and particularly on key poverty and inequality indicators, they have sparse and wholly inadequate post-market institutions supporting population health (e.g., the social safety net, educational institutions, health institutions), most recently evident after the Great Recession of 2008 (The Stanford Center on Poverty and Inequality, 2014). It makes little sense to spend vast sums of money treating people's illnesses in the health care system to then send them back into the conditions that generated those illnesses. In doing so, it suggests that the solutions to the problems are coming from within the problems themselves—e.g., more health care spending and services to meet demands on the system, rather than social services spending to decrease demand on the system (Cooper et *al.*, 2012). More health care spending has not delivered on the goal of improved

population health, nor can it—various recent estimates put its relative contribution to population health status at between 10% and 25% (Marmot & Allen, 2014; Marmot & Bell, 2011). Despite spending nearly half of the world's health care bill (Bezruchka, 2010), over thirty countries have better health than the U.S. population by many common population health indicators (Bezruchka, 2014).

In addition to having enough income in the absolute sense to acquire the most basic prerequisites of health, the distribution of income in a society has implications for population health, not only for those at the low end of the distribution, but for all members of society except perhaps the extreme top. This paper addresses one mechanism by which income distribution affects population health: the direct relationship between individual (or household) income distribution and population health, also known as the *absolute income effect*. This effect rests on two well-founded assumptions. First, at least part of the relationship from income to health (in this case life expectancy) is causal (Case et *al.*, 2002; Costello, Compton, Keeler, & Angold, 2003). Second, the shape of the relationship is concave, i.e., there are diminishing marginal returns of more income to health (Backlund, Sorlie, & Johnson, 1996; Deaton, 2002b; Subramanian & Kawachi, 2004). That is, among households with very low incomes, each additional dollar has a greater effect on health than it does among those households with sufficient income to meet basic needs of nutrition, shelter, and clothing, etc., or than it does among households with much more than sufficient income. Diminishing marginal returns of income to health has an important implication for income distribution and population health, first observed by Rodgers (Rodgers, 1979).

Assume y1 is the initial average life expectancy in Figure 1. If we tax a rich person (x4) and transfer the money (without deadweight loss) to the poor person (x1), then the spread of the

income distribution narrows from x1 to x4, to x2 to x3, and the mean income (x) remains constant at x. We have effected a mean-preserving income transfer, and the post-tax life expectancy has increased from y1 to y2. This effect suggests that, *ceteris paribus*, among countries with similar levels of development, those with a narrower distribution of income will have higher average life expectancy because any health loss to a rich household is more than offset by an increase in health to a poor household. Rodgers simply restated the principle of philanthropy toward health issues: when billionaires donate to the world's poor, they do not diminish their own health and may even improve it through the "warm glow" of doing a good deed (Dunn, Aknin, & Norton, 2014). Meanwhile, with roughly one third of people on the planet subsisting on less than $2 per day, the gift can mean the difference between life and death. For example, an insecticide-treated bed net to prevent malaria costs $3 to $5 (Kawachi & Subramanian, 2014).

Rodger's theory and its tremendous impact in the academic literature were revisited in a second article and a series of commentaries in the *International Journal of Epidemiology* (Deaton, 2002a; Lynch & Smith, 2002; Porta, Borrell, & Copete, 2002; Rodgers, 2002; Wilkinson, 2002). The commentaries on whole did not seem to question the theoretical case or basis for Rodgers' claims, but rather the lack of adequate data (at the time) to make firm conclusions, and the mechanisms or pathways through which the effects are realized. As Deaton explains, there is little question that infant mortality and child mortality in developing countries would be improved by decreasing income inequality, and the evidence is supportive in developed countries as well, using sources such as the Luxembourg Income Survey. The data issues have tended to dwarf the theoretical or conceptual issues (Deaton, 2003). Further, there has tended to be an overemphasis on income inequality compared to other forms of inequality—income

inequality is important, but others may be more so. He also called on a fuller recognition of the mutual dependency between health and the ability to earn an income. If the only channel through which income affects health is directly through material resources, then better health and better insurance systems against disability and income loss due to disability would lessen the impact of income inequality on health, effectively recruiting the reverse pathway as a policy mechanism to reduce inequalities in income and thus improve health (Deaton, 2002a, 2003). Similarly, Lynch and Smith note that social inequality is multidimensional and not limited to income inequality, and it is expressed as inequalities in education, occupation, housing, access to services, and discrimination by ethnicity, gender, age, etc. The interconnections among these dimensions are "intricate and difficult to disentangle." Also, health is " a multidimensional construct that is not captured by death statistics" (Lynch & Smith, 2002). They note that if individual income itself is the only mechanism through which inequality affects health, this result is still important and implies a major role in policy for redistribution, because we have good evidence that redistribution works, particularly in countries such as the United Kingdom and United States.

Assuming the absolute income effect exists, the question becomes, "what is the magnitude of the effect?" Two investigators attempted to answer this question for New Zealand (Blakely & Wilson, 2006). They simulated the impact on mortality risk of shifting equivalized (for the number of household members) household income between 10% and 40% toward the mean household income of a cohort comprising all respondents to the New Zealand 1996 census (n = 1.3 million), following respondents forward for three years. This exercise was equivalent to reducing the Gini coefficient by the same percentage, as the authors demonstrated in the appendix to their article. They found that shifting household income by 10% toward the mean income resulted in a 4% reduction in overall mortality, adjusting for confounders including age,

marital status, education, car access, and neighborhood socioeconomic deprivation score. This percentage translated into 1,100 annual deaths averted, exceeding those due to motor vehicle accidents, but may have been an overestimate because 1) the authors assumed the full benefit of the transfer is captured, and 2) did not account for the "leaky bucket" phenomenon, whereby some of the money transferred must be lost during the transfer (Kawachi & Subramanian, 2014).

This study attempts to quantify the absolute income effect of income on mortality and inequalities in mortality in the United States among persons aged 25 to 59, using three cohorts followed for three years from the NLMS. It also investigates the sensitivity of this effect to assumptions regarding the causal effect of income on mortality. The intent is to help gauge the likely impact on mortality risk of potential policy interventions to reduce income inequality in the United States. Knowing the magnitude of the expected impact on mortality of narrowing income differentials can help to verify whether the reductions in mortality and in inequalities in mortality are large enough, relative to the costs (including the opportunity and transaction costs), to warrant public policy intervention. To our knowledge, the absolute income effect has not been quantified for the United States. Further, few prior studies have followed individuals forward for several years using nationally representative cohorts in order to adequately capture the effects on mortality (Blakely, Kennedy, Glass, & Kawachi, 2000; Zheng, 2012). Further, the study period corresponds to one during which national-level income inequality has achieved its highest recorded level in American history as we enter what has been termed the Second Gilded Age.

Conceptual Model

The conceptual approach utilized in this paper includes Link and Phelan's fundamental cause theory (Link & Phelan, 1995; Phelan, Link, & Tehranifar, 2010), which draws attention to the preeminence of underlying social and economic factors and to the distribution of resources

needed to improve health, and is incorporated into the conceptual framework adopted by the WHO CSDH shown in Figure 2. The CSDH framework begins with analysis of the socioeconomic and political contexts, including governance, policy (macroeconomic, social, health), and cultural and societal norms and values, which shape the social position or class of individuals within society. These social positions are manifested in levels of education, occupation, and income, and are related to gender, ethnicity and race. These societal-level processes then determine individuals' exposures to health-damaging or health-promoting conditions in their daily lives: the intermediary health determinants, including material circumstances, behavioral and biological factors, and psychosocial factors that make people more or less vulnerable or resilient to disease (Marmot & Allen, 2014). At each point in the system, there are complex feedbacks, which can have positive or negative effects. These models should be considered dynamic in nature, allowing multiple intervention points or levers by which the distribution of health in the population might become more equitable, and overall population health improved (Glymour et *al.*, 2014).

Fundamental cause theory claims that high-SES persons have varied resources "such as money, knowledge, prestige, power, and beneficial social connections that protect health no matter what mechanisms are relevant at any given time" (Glymour et *al.*, 2014; Link & Phelan, 1995; Phelan, Link, Diez-Roux, Kawachi, & Levin, 2004). The theory helps explain the persistence of health inequities over time, and how lower SES persons are more likely to be exposed to health risks regardless of their specific nature. A failure to include social and economic context (i.e., to contextualize risk) and to evaluate risk distributions in populations can cause at least two problems. First, individual-level interventions focused on behavioral change intended to reduce risk factors may be ineffective because social constraints on behavior make it

too costly or impossible for individuals to alter their own behaviors in a sustainably positive way. Second, a focus on individual risk factors risks blaming individuals for circumstances over which they have little or no control. However, a limitation of fundamental cause theory is that it does not help to determine the marginal impact of a particular resource (maintained in excess by high-SES persons) on improvements in health. Therefore, it does not provide policy guidance on which particular resource is the best one to invest in, or help us to predict the likely health impact of the specific investment. Nor does it suggest where there may be unintended consequences for particular policies. It seems to suggest that the combined set of resources is important to maintaining health, and that increasing one alone is unlikely to be sufficient. For example, marginal increases in income are unlikely to improve health in the absence of the other resources helping one to utilize that income for better health (Glymour et *al.*, 2014).

The current study both applies fundamental cause theory and the CSDH conceptual framework and attempts to make some headway past its limitations by attempting to determine as accurately as possible (but necessarily with a fair degree of uncertainty), the marginal impact of more household income on premature mortality in the U.S. population aged 25 to 59. Specifically, to what extent will mortality, and inequalities in mortality across SES groupings, be improved by redistributive policies that decrease income inequality? Further, what are the likely costs of such policies and would they exceed the benefits, and are there likely to be unintended consequences? The study is an application of theory in which the results will hopefully feed back to further inform one aspect of the theory to potentially modify it, as all theories are subject to modification in order to accommodate valid and relevant evidence.

Of the resources (money, knowledge, prestige, power, and beneficial social connections) mentioned above, the greatest role for public policy would seem to be with the first two, through

fiscal and monetary policies and education policies. Link and Phelan offer three policy strategies based in fundamental cause theory that will lead to improvements in population health. First, reduce inequalities in the above key resources for health. They note that such policies already constitute a major part of the national policy agenda: minimum wage laws, housing for low-income or homeless people, capital gains and estate taxes, parental leave, social security, head-start and college-admission policies, and regulation of lending practices. Regarding macroeconomic monetary policy, Stiglitz calls for abandoning a single-minded preoccupation with controlling inflation at two percent or less, which mainly benefits bondholders, and a greater focus on the demand side of the economy, and in particular un- and under-employment (Stiglitz, 2012). Baker and Bernstein provide evidence that a sustained one percentage-point decline in the unemployment rate is associated with a 9.4 percent rise in the wages of workers in the bottom quartile of the wage distribution, and that a policy of full employment could reverse income inequality (Bernstein & Baker, 2013). Link and Phelan argue that understanding how these direct SES policies are relevant should become a vital part of the agenda for medical sociology. To the list of fundamental causes of population health inequalities the authors would add stigma (Hatzenbuehler, Phelan, & Link, 2013; Link & Phelan, 2006), which overlaps with racism and discrimination but is much broader, and has tremendous, underappreciated public health implications. It is the focus of Link and Phelan's more recent research, featured in a recent special issue of *Social Science and Medicine* devoted to structural stigma and health (Angermeyer, Matschinger, Link, & Schomerus, 2014; Link & Phelan, 2014; Phelan, Lucas, Ridgeway, & Taylor, 2014). Like income inequality and mortality, these processes operate insidiously across social status groupings, affecting everyone, and more often than not are unconscious, subtle, and hidden from view.

The second policy strategy advocated by Link and Phelan is to contextualize individual risk factors by identifying factors in the environment that place people "at risk of risk," for example, power imbalances that prevent people from engaging in safe sex practices or neighborhoods where healthy foods are unavailable or, more likely, prohibitively expensive. Finally, institute health interventions that benefit individuals regardless of their own resources or behaviors, such as air bags in automobiles, health screenings in communities rather than doctor's offices, and health care access to all citizens rather than only those who can pay for it (Phelan et *al.*, 2010).

Interestingly, an important earlier study by Phelan and colleagues used the NLMS, taking advantage of its cause of death information, to test fundamental cause theory. The authors hypothesized that for less preventable causes of death, for which we know little about prevention or treatment, SES would be less strongly associated with mortality than for more preventable causes. Their hypothesis was supported, lending further support to fundamental cause theory and to approaching health disparities from a sociological perspective emphasizing the importance of SES for mortality (Phelan et *al.*, 2004).

This study follows closely on Link and Phelan's prudent recommendations for improving population health by focusing on income inequality and mechanisms to directly reduce it—a simulation of income redistribution to decrease income inequality and its causal impact on premature mortality risk reduction. We show that the *social gradient in health* with respect to premature mortality is evident in our initial analyses, that is, both the estimated observed and predicted mortality rate ratios demonstrate an upward gradient from highest to lowest income deciles. We find that the *absolute income effect* is in the range of a three to four percent reduction in mortality for a 10% reduction in the Gini coefficient. There are larger mortality

reductions with larger reductions in the Gini, but with diminishing returns. Finally, inequalities in estimated mortality rates are reduced by a larger percentage than overall estimated mortality rates under the same counterfactual redistributions.

Methods

Data and Sample

Data for this study come from the NLMS, a large prospective household survey based on a random sample of the non-institutionalized population of the United States, developed specifically to study the effects of demographic and socioeconomic characteristics on differentials in U.S. mortality rates. The NLMS matches individual records from the Current Population Survey (CPS) (Current Population Survey (CPS), 2012) to the National Death Index (NDI) (National Death Index, 2014). It currently consists of 36 cohorts and includes approximately 3.6 million records with over 250,000 identified mortality cases. A complete description of the dataset is available in the latest edition of the Reference Manual (National Longitudinal Mortality Survey: Extract and Analysis Files, 2012).

The NLMS is based on specific survey months of the CPS, the Annual Social and Economic Supplement, and a subset of the 1980 Census. Because these surveys are one-time data collection processes with no subsequent data collection follow up, a limitation of NLMS data is that they provide a one-time only, baseline, measurement of subjects in follow-up studies. Another limitation is that although the CPS and Census offer rich data in specific subject areas, general or specific health information on subjects is not collected, and smoking status is available on a limited number of subjects.

We use CPS cohort years 2005 to 2008 to allow for three years of follow-up (the most recent data on mortality in the NLMS is for the year 2011). The outcome variable is an indicator

of death versus no death at three-year follow-up. The set of covariates in regression models include inflation-adjusted (in 1990 dollars) annual family income equivalized for the number of persons in the household by dividing by the square root of the number of persons in the household (the method used by the Congressional Budget Office in its publications and the most straightforward). County-level poverty rates come from the 2000 Decennial Census and are used to control for small-area poverty level. The set of demographic indicators measured at the individual level include a continuous measure of age in years at the time of the interview; a set of race/ethnicity indicators (Hispanic, non-Hispanic white, non-Hispanic black, non-Hispanic Asian/Pacific Islander, non-Hispanic American Indian/Alaskan Native, and non-Hispanic other race); a marital status indicator (married, never married, widowed/divorced/separated); highest level of education (dropout, high school or some college, college degree or more); and having a known Social Security Number (an important factor for probability of matching to death records). The analysis is stratified by sex to assess any differential effects by sex.

Empirical model

The analysis employs logistic regression to generate adjusted odds ratios by income categories by regressing an indicator of mortality on the natural logarithm of equivalized household income, controlling for the above confounders. Define x_j as the (row) vector of independent variables, augmented by 1, and b as the corresponding estimated parameter (column) vector. The logistic regression model is fit by the logit transformation. The likelihood function for logit is:

$$\ln L = \sum_{j \in S} w_j \ln F(\mathrm{x}_j \mathrm{b}) + \sum_{j \in S} w_j \ln\{1 - F(\mathrm{x}_j \mathrm{b})\},$$

where S is the set of all observations j, such that $y_j \neq 0$, $F(z) = e^z/(1 + e^z)$, and w_j denotes the optional weights. ln$L$ is maximized using the modified Newton-Raphson algorithm in Stata. The

odds ratio corresponding to the i^{th} coefficient is then $\psi_i = \exp(b_i)$. The standard error of the odds ratio is $s_i = \psi_i s_i$, where s_i is the standard error of b_i estimated by logit in Stata. Predicted probabilities are generated as follows: Define $I_j = x_j b$ as the predicted index of the j^{th} observation. The predicted probability of a positive outcome is:

$$p_j = \frac{\exp(I_j)}{1 + \exp(I_j)}$$

Analysis

NLMS data are converted to person-time data and then logistic regression is used to calculate 3-year mortality rates for each income decile. Therefore, the numbers of persons remain equally spaced across deciles, but person-years are greater in higher income deciles due to longer life expectancies in these deciles. The analysis population is restricted to those aged 25 to 59 years in order to avoid effects of income loss from retirement, and excludes deaths and person-time in the first six months to reduce health selection effects (where poor health causes loss of income and therefore reverse causation in the income-mortality relationship). Total household income is equivalized for the number of children and adults to allow for economies of scale, as noted above. Next, individuals are allocated to one of ten income categories based on roughly equal deciles of equivalized family income, rounding the cut points to the nearest $100, and logistic regression performed to determine the mortality rate ratios compared to that of the reference group containing the mean income of the population (decile seven). Because age and race/ethnicity are major determinants of both income and mortality, baseline estimates of the income–mortality association are initially adjusted for both age and race/ethnicity, and then further adjusted for other important demographic and socioeconomic factors based on the conceptual model and the availability of the above-mentioned covariates in the dataset.

Next, a log transformation of equivalized family income is applied in order to improve the fit of the income-mortality curve. Then, modelling exercises are performed to determine the effects of hypothetical interventions which A) lift everyone living on an equivalized household income which includes the U.S. poverty line in 2000 [(weighted average for family of four people $17,603, or an equivalized income of $8,801) (Dalaker, 2001)] to the income category just above, the targeted policy equivalent of lifting out of poverty the households with the lowest incomes, and B) shift everyone's income by 10–40% to the mean household income, equivalent to reducing the Gini coefficient by 10–40% (see the appendix of Blakely & Wilson, 2006 for the proof of this result). The modelling exercises use the coefficient for the logarithm of equivalized family income in the multivariate regressions noted above. In the first simulation, the income shifts occur through redistribution of dollars from the top decile to the bottom three deciles, which fall below the U.S. poverty threshold (as opposed to an influx of new income to the lower deciles). In the second simulation, dollars are shifted from upper to lower deciles, such that all deciles are moved between 10% and 40% toward the mean household income.

The change in overall mortality rate is calculated using the Population Attributable Risk Percent (PAR%) calculation for a counterfactual scenario, in which the new relative risks of mortality for each of the ten income categories previously specified are calculated using the post-redistribution mean income for each group.

$$PAR = \frac{\sum_i (P_i \text{ x } RR_i) - \sum_i (P_i \text{ x } RR_i\text{^})}{\sum_i (P_i \text{ x } RR_i)},$$

where RR_i is the relative risk of income group i *before* the counterfactual change, RR_i^ is the relative risk of income group i *after* counterfactual change, and P_i is the proportion of population in each income group (Blakely & Wilson, 2006; Murray & Lopez, 1999).

We also estimate the changes in inequalities in mortality associated with these hypothetical interventions, comparing estimated rate ratios for people in the second lowest decile to those in the second highest decile, in order to avoid comparisons based on extreme groups. We apply sensitivity analyses by halving the coefficients for the logarithm of household income. All analyses use survey weights designed to match the approximate non-institutionalized population of 25 to 59 year olds using the 2000 Census (the exact number is 136,187,440 from Table P012). Because the data are in person-years for the logistic regression analyses, the case weights are divided among the person-years.

Results

Table 2.1 shows weighted summary statistics for males and females. Deaths per year and over three years were higher among males (2.8 vs. 1.7 per 1,000 and 6.2 vs. 3.8 per 1,000, respectively). Mean equivalized income adjusted to 1990 dollars was $29,287 among males and $28,107 among females. Mean age was roughly 42 years. Reported race/ethnicity was roughly 15% Hispanic, 68% non-Hispanic white, 11% non-Hispanic black, with the remaining racial groups comprising five percent or less of the sample. Approximately 64% of the sample was married, with more males reporting never being married (22% vs. 17%) and more females reporting being widowed, singled, or divorced (20% vs. 14%). Men reported slightly lower levels of education, with roughly 30% of the sample reporting a college degree or higher. The mean county-level poverty rate (from the 2000 Census) was 12% and county-level median income approximately $52,000. Over 83% of survey participants reported a valid social security number to allow matching to death certificates.

Table 2.2 shows the weighted person-years, deaths, and observed rate ratios for each of the ten deciles of household income in the three NLMS cohorts. Coefficients for the logarithm of

equivalized household income from weighted logistic regressions adjusting for age and ethnicity were -0.315 and -0.348 for males and females, respectively, while coefficients from the multivariate models were -0.232 and -0.271, respectively. Using these coefficients and the mean income in each income decile, predicted mortality rate ratios by income decile are also shown.

Figure 3 shows the density of survey respondents per $1000 range of income (left vertical axis) for both males and females. The highly right-skewed pattern for income distribution is present. Some of the rate ratios from Table 2.2 are plotted with this density distribution (right vertical axis). For males, there is relatively good fit between the observed and modeled rate ratios except for the lowest income categories. For females the fit is good only for the highest five equivalized income deciles, with the observed rate ratio peaking at 4.9. For both males and females, the lower values for modeled multivariate rate ratios indicate attenuation due to confounding among the lowest five equivalized income deciles.

Table 2.3 shows the results for the counterfactual scenarios. As indicated in column three, under scenario A—lifting everyone out of the bottom two income deciles into the third decile—overall mortality is reduce by 3.1% (males) and 6.4% (females), corresponding to an 11.9% and 21.5% reduction in the Gini coefficient, respectively. This scenario reduces relative inequalities in mortality, measured as excess relative risk for the second lowest to second highest income deciles, by 14% (males) and 17% (females). Under scenario B—shifting everyone's household income 10–40% toward the mean—mortality rates decrease by three to seven percent (males) and four to nine percent (females). This scenario reduces relative inequalities by 9–28% (males) and 11–34% (females). Also shown in Table 2.3, in sensitivity analyses in which we halved the coefficient for the log transformation of income from the preferred multivariate model, effectively assuming that the preferred model overestimates the strength of the causal association

of income with mortality by two-fold, all PAR% and reductions in relative inequalities were approximately halved.

Discussion and Conclusion

Our modelling scenarios indicate that the absolute income effect on mortality is roughly a 3–4% reduction in mortality for a 10% reduction in the Gini coefficient in this nationally representative study of U.S. adults aged 25 to 59. The analysis indicates that while both overall mortality and inequalities in mortality would be reduced, the percent reductions would be larger for inequalities in mortality. Also, the results suggest that the redistributional effects on mortality in women would be greater compared to the effects in men. Because the income-mortality association is nonlinear and income is taken from the top decile (scenario A) and from households above the mean income (scenario B) there would be health costs in these groups, but the overall modest reductions in mortality rates of 3% to 6% would represent a sizeable improvement in health status in this 25-59 year-old age group. For comparison, a 5% reduction in all-cause mortality would approximate eliminating all unintentional injury deaths in the United States in 2013. An approximately 20% reduction in the Gini would correspond to a return from present levels of inequality in the United States (Gini of 0.476 in 2013) to levels present in the United States 45 years ago (Gini of 0.394 in 1970). Such a reduction might reduce inequalities in mortality by 17% to 20%. The results of this study are very similar to those for the only other study of which we are aware that attempted to quantify the absolute income effect in a developed country (New Zealand), (Blakely & Wilson, 2006) though we found larger disparities in effects by gender. In his recent text on inequality, Tony Atkinson estimates, under reasonable assumptions regarding tax rates and government spending, that to achieve a reduction in the Gini for disposable income would require increasing taxes by 1.6 times the desired percentage point

reduction in the Gini. For example, a 10 percentage-point reduction in the Gini for disposable income would require a 16 percentage-point increase in marginal tax rates. The size of this rate increase suggests that reducing inequality can not rely on fiscal measures such as redistribution alone (Atkinson, 2015).

As indicated in the prior New Zealand cohort study, there are numerous limitations to any modelling exercise of the impact of income redistribution on population health and inequalities in health. First, it is important to evaluate a policy-relevant counterfactual. While income redistribution is but one mechanism to reduce income inequality, it may not be the best and is not sufficient of itself. There are many policy mechanisms to be employed to reduce inequality—for example, policies might focus on making market incomes more equal as opposed to redistributing income through taxes and transfers—but there is little doubt that a host of policy mechanisms will need to be employed simultaneously given the degree of inequality in the United States. The estimates here of the impact of the mechanism of income redistribution nevertheless provide valuable information for policymakers who would chose this mechanism from a list of several others. Another limitation in regression modeling is confounding, whereby the association between the exposure of interest and the outcome of interest is at least partly due to a correlated variable predicting the outcome. Residual confounding in particular refers to the omission of variables from the model which serve as confounders. As George Box noted, "all models are wrong, but some are useful." We have attempted to control for the important variables that might confound the association between income and mortality, but our model may well omit important variables, and include measurement error among the included variables, and/or may over-adjust or under-adjust by including or excluding partial mediators of the association. Additionally, we have utilized the best available U.S. dataset to estimate the absolute

income effect and have followed study participants forward for three years. Another issue is the time lag between the exposure (income level) and outcome (mortality). Given that it may take years to a decade or more for low income to lead to mortality, it is likely that a three-year follow-up period underestimates the full effect of income level on premature mortality. Also, redistribution policies are not costless in that there are dead-weight costs to society from some forms of taxation and redistribution. These costs are not captured here and we assume no overall change in total income and thus no "leaky bucket", the notion put forth by Arthur Okun that any transfer of wealth or income from rich to poor involves loss of total income due to the administrative costs of taxing and transferring, and to incentive effects. However, it should be noted that taxation could be designed in such a way that it encourages activities that are beneficial to society and discourages activities (like rent-seeking) that are costly, while also increasing equity (Stiglitz, 2015).

In summary, the results of this analysis confirm and roughly quantify the absolute income effect in the United States, indicating a modest reduction in overall mortality rates through income redistribution, more so for females compared to males, and that relative inequalities in mortality should reduce even more. The numerous adverse effects of income inequality on economic opportunity, economic growth, etc. are well documented. That nontrivial gains in life expectancy will go to the lower income deciles from redistribution means that the lower income deciles are in effect paying with their lives to sustain high inequality, and this information should, in a democratic society, lead to resistance and movement toward meaningful policy change, and to a politics of health as if all people mattered.

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TABLES

Table 2.1. Descriptive statistics on the 2005-2008 person-year dataset, by sex

	Males (n = 335,283 PY; weighted n = 200,839,899 PY)			Females (n = 366,069 PY; weighted n = 206,464,689 PY)		
Variables	**Mean**	**SD**	**Range**	**Mean**	**SD**	**Range**
Dependent variables						
Deaths over 3 years (%)	0.62	7.88		0.38	6.13	
Deaths per year (%)	0.28	5.26		0.17	4.13	
Days alive	1,093.72	34.40	183–1,096	1,094.66	26.75	183–1,096
New persons entering cohort per year (%)	33.40	47.16		33.38	47.16	
Person-year weight	876.53	472.81	13–5,924	802.04	399.74	12–5,449
Independent variables						
Adjusted income in 1990 dollars	$49,533	$55,030	-$12,188–$2,366,114	$47,933	$54,328	-$12,188–$2,366,114
Family income in dollars	$77,574	$86,181	-$18,800–$3,535,594	$75,120	85,145	-$18,800–$3,535,594
Equivalized adjusted income in 1990 dollars	$29,287	$33,118	-$8,618–$1,183,057	$28,107	31,940	-$8,618.22–$1,183,057
Ln equivalized adjusted income in 1990 dollars	$9.95	$0.92	-$1.10–$13.98	$9.87	$0.10	-$1.10–$13.98
Income decile	5.60	2.83	1–10	5.41	2.91	1–10
Income deciles (% total pop)						
1	8.68	28.16		11.25	31.60	
2	9.68	29.57		10.29	30.38	
3	10.02	30.03		10.00	30.00	
4	10.06	30.08		9.91	29.88	
5	10.31	30.41		9.71	29.60	
6	10.28	30.37		9.74	29.65	
7	10.24	30.32		9.77	29.68	
8	10.29	30.39		9.72	29.62	
9	10.26	30.35		9.76	29.67	
10	10.16	30.22		9.86	29.81	
Demographic variables						
Age (years)	41.73	9.85	25–59	41.96	9.82	25–59
Race/ethnicity (%)						
Hispanic	15.48	36.17		13.77	34.46	
Non-Hispanic white	68.09	46.61		67.66	46.78	
Non-Hispanic black	10.16	30.21		11.97	32.46	
Asian	0.55	7.36		0.57	7.55	
Asian/Pacific Islander	4.70	21.17		5.00	21.79	
Other race/ethnicity	1.02	10.03		1.02	10.07	
Marital status						
Married	63.79	48.06		63.69	48.09	
Never married	22.47	41.74		16.68	37.28	
Widowed/single/divorced	13.73	34.42		19.64	39.72	
Education						
HS dropout	12.96	33.58		10.83	31.07	
HS plus some college	57.05	49.50		57.74	49.40	
College degree or more	30.00	45.82		31.43	46.43	
County-level percent in poverty (2000 Census)	12.01	5.34	2.12–52.32	12.10	5.44	2.12–52.32
County-level median family income in dollars (2000 Census)	$52,102	$12,346	$18,925–$92,146	$51,979	$12,388	$18,925–$92,146
Valid Social Security # (%)	83.20	37.39		84.03	36.63	

Table 2.2. Person-years, deaths and rate ratios of mortality by equivalized household income among 25–59-year-old deaths, 2003–2005 cohorts of the NLMS

Equivalized household income	Weighted person-years	Weighted deaths	Observed rate ratios, using categorical income data (95% CI)		Predicted rate ratios, using coefficients for logarithm of income	
			Age- and race/ethnicity-adjusted	Multivariate[a]	Age- and race/ethnicity-adjusted	Multivariate[a]
Males						
≥ $53,974	20,414,847	29,632	0.50 (0.32–0.77)	0.64 (0.41–1.00)	0.72	0.79
$40,167-53,973	20,612,668	40,091	0.75 (0.49–1.13)	0.86 (0.57–1.32)	0.86	0.90
$32,447-40,166	20,672,875	33,915	0.68 (0.45–1.02)	0.73 (0.48–1.10)	0.94	0.95
$26,837-32,446	20,569,055	48,064	1.0	1.0	1.00	1.00
$22,242-26,836	20,653,152	39,109	0.89 (0.60–1.32)	0.85 (0.58–1.27)	1.06	1.05
$18,072-22,241	20,700,878	58,598	1.41 (0.95–2.07)	1.29 (0.88–1.90)	1.14	1.10
$14,166-18,071	20,207,739	55,825	1.45 (0.99–2.11)	1.25 (0.86–1.83)	1.22	1.15
$10,181-14,165	20,122,653	65,581	1.84 (1.27–2.67)	1.48 (1.01–2.15)	1.34	1.23
$5,969-10,181	19,444,860	92,907	2.72 (1.93–3.84)	2.03 (1.42–2.90)	1.54	1.36
≤ $5,969	17,441,172	93,081	2.79 (1.95–4.00)	2.01 (1.40–2.90)	2.20	1.74
Total	200,839,899	556,803				
Females						
≥ $53,974	20,360,209	9,610	0.41 (0.21–0.79)	0.47 (0.25–0.91)	0.67	0.74
$40,167-53,973	20,144,802	21,208	0.98 (0.56–1.71)	1.07 (0.60–1.89)	0.85	0.88
$32,447-40,166	20,071,340	28,622	1.41 (0.85–2.37)	1.47 (0.88–2.47)	0.93	0.95
$26,837-32,446	20,162,591	19,305	1.0	1.0	1.00	1.00
$22,242-26,836	20,104,265	22,657	1.26 (0.73–2.17)	1.21 (0.70–2.09)	1.07	1.05
$18,072-22,241	20,040,083	29,173	1.68 (1.00–2.83)	1.56 (0.93–2.62)	1.15	1.11
$14,166-18,071	20,461,711	35,181	2.09 (1.28–3.43)	1.87 (1.13–3.08)	1.24	1.18
$10,181-14,165	20,648,475	43,197	2.76 (1.69–4.48)	2.31 (1.40–3.81)	1.38	1.28
$5,969-10,181	21,244,516	59,825	3.93 (2.50–6.19)	3.09 (1.94–4.94)	1.60	1.43
≤ $5,969	23,226,697	83,546	4.91 (3.18–7.60)	3.67 (2.31–5.83)	2.31	1.88
Total	206,464,689	352,324				

[a] In addition to age and race/ethnicity, the multivariate model includes marital status, highest level of education, having a known Social Security Number, and county-level poverty rate in 2000.

Table 2.3. Percentage reductions in overall mortality rates (population attributable risk percent, PAR%) and changes in the relative risk of mortality by income for various income redistributions

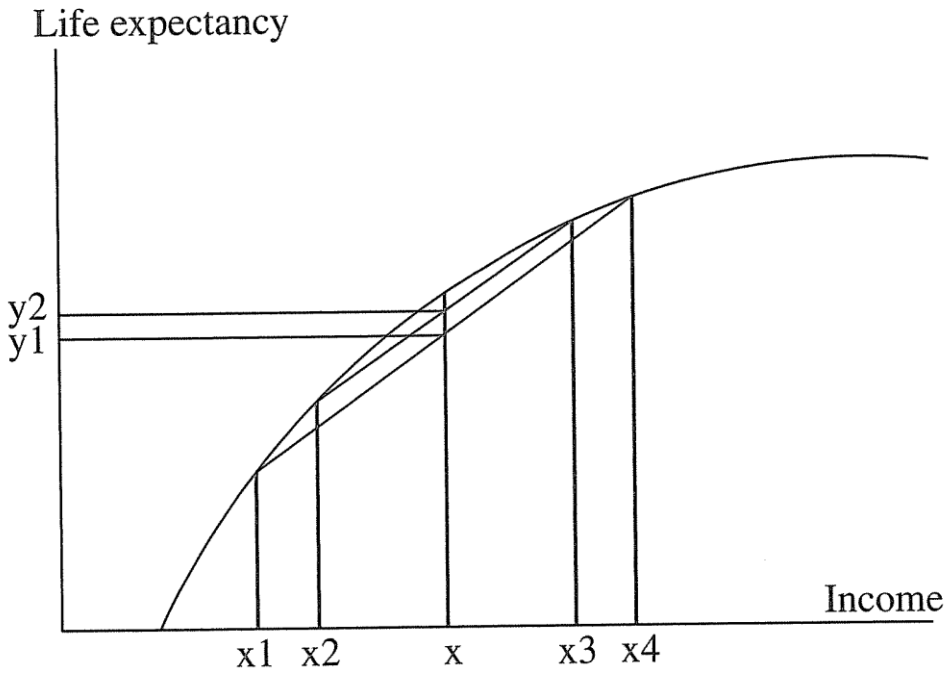
Change of income distribution modelled	Resulting % reduction in Gini coefficient	Using coefficient of the logarithm of household income from the *preferred* multivariate model[a]			Sensitivity analysis: *Halving* the coefficient of the logarithm of household income from the preferred multivariate model[a]		
		% Reduction in mortality rate (i.e., PAR%)	Estimated age- and ethnicity-adjusted rate ratio for people in the 2nd lowest c.f. 2nd highest income group (% reduction of relative inequalities)[b]		% Reduction in mortality rate (i.e., PAR%)	Estimated age- and ethnicity-adjusted rate ratio for people in the 2nd lowest c.f. 2nd highest income group (% reduction of relative inequalities)[b]	
Males							
Do nothing	0	0	1.78	(0)	0	1.78	(0)
A: People with income <$8,900 raised to $10,181-$14,165 category	11.9	3.1	1.64	(14)	1.43	1.72	(6)
B: Everyone's household income moves 'X' % to the mean household income							
X = 10%	10	2.7	1.69	(9)	1.24	1.74	(4)
X = 20%	20	4.6	1.61	(17)	2.15	1.71	(7)
X = 30%	30	6.1	1.55	(23)	2.91	1.68	(10)
X = 40%	40	7.4	1.50	(28)	3.58	1.66	(12)
Females							
Do nothing	0	0	1.88	(0)	0	1.88	(0)
A: People with income <$8,900 raised to $10,181-$14,165 category	21.5	6.4	1.70	(17)	2.76	1.81	(7)
B: Everyone's household income moves 'X' % to the mean household income							
X = 10%	10	3.9	1.76	(11)	1.64	1.83	(5)
X = 20%	20	6.2	1.67	(20)	2.70	1.79	(8)
X = 30%	30	8.0	1.60	(28)	3.48	1.76	(11)
X = 40%	40	9.3	1.53	(34)	4.09	1.73	(14)

[a] The preferred multivariate model adjusted for age, ethnicity, marital status, highest level of education, having a known Social Security Number, and county-level poverty rate.

[b] The estimated age- and ethnicity-adjusted rate ratio was calculated as described in methods. The percentage reduction was that for the excess rate ratio (i.e., RR minus 1), using the "Do nothing" or baseline age and ethnicity adjusted rate ratio as the baseline rate ratio.

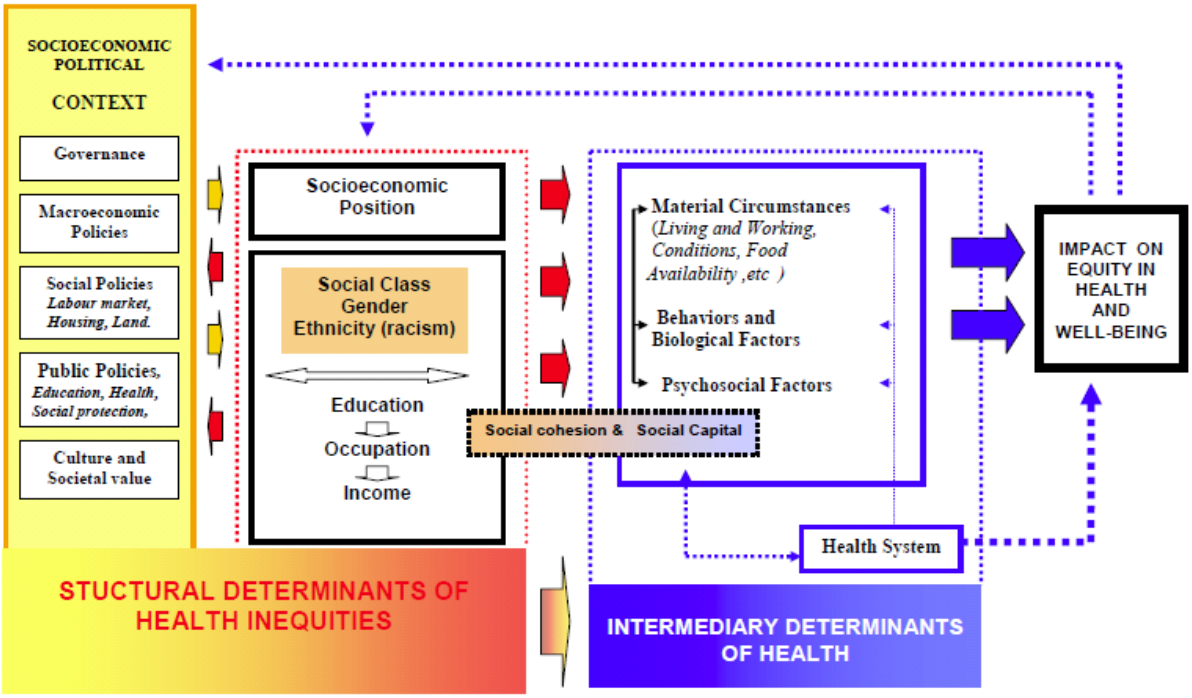
FIGURES

Figure 2.1. Curvilinear relationship between income and life expectancy



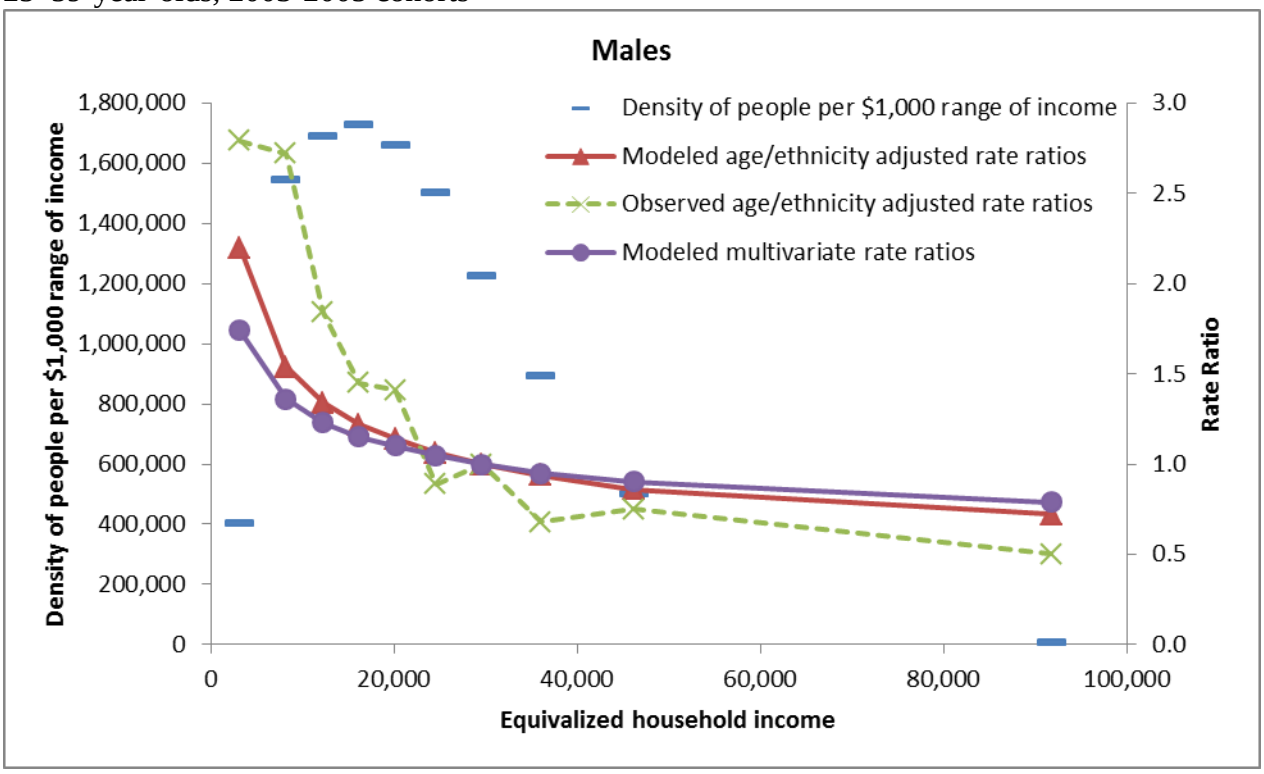
Source: (Kawachi & Subramanian, 2014; Rodgers, 1979)

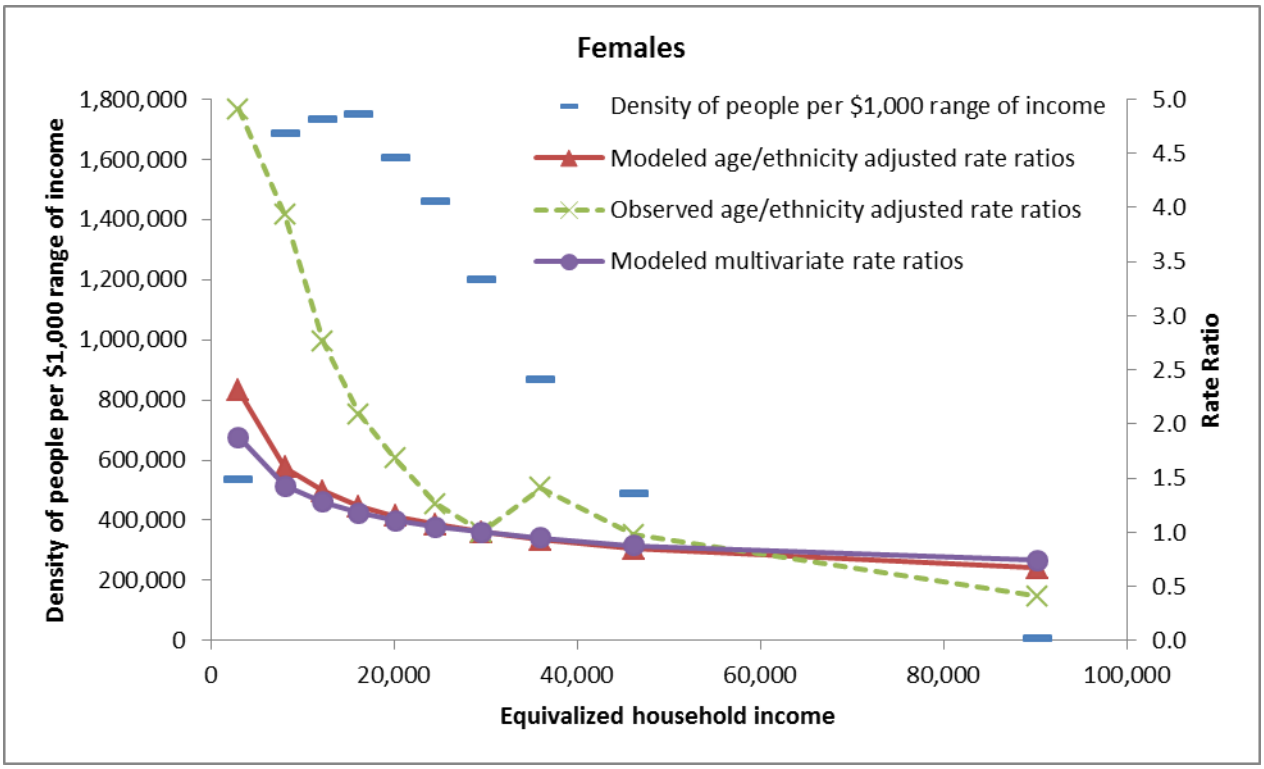
Figure 2.2. Final version of the conceptual framework adopted by the WHO Commission on the Social Determinants of Health



Source: (Solar & Irwin, 2006; Solar & Irwin, 2010)

Figure 2.3. Density of people per centile of household income, and rate ratios of mortality (reference group $26,837-$32,446) for various specifications of the household income variable, 25–59-year-olds, 2003-2005 cohorts





ESSAY 3: TESTING THE CONTEXTUAL THEORY OF INCOME INEQUALITY IN THE UNITED STATES USING THE NATIONAL LONGITUDINAL MORTALITY STUDY

"There is no public health without equality."[3]

"Public health is not a branch of medicine. Rather, medicine is a branch of public health."[4]

Introduction

The contextual effect of income inequality on health posits that income inequality can have direct spillover effects on not just the poor but the more affluent as well, over and above the absolute income effect (Kondo, 2012). The contextual theory claims that health (or life expectancy, in this case) is a function of both individual (or household) income and the *rate* of diminishing marginal returns or the *degree of concavity* in the income-health distribution, most often captured by the Gini coefficient (Figure 1). Among the explanations theorizing a causal effect of income on health, this one is the most controversial, and it has been investigated and debated in the literature since it was first identified in a seminal paper by Richard Wilkinson (Wilkinson, 1992). Wilkinson has suggested people living in highly unequal societies pay a "health tax," which Kawachi and Suburamanian have compared to a miasma or air pollution—it

[3] Health Politics as if People Mattered: 2. Good Practice in Academia. A Politics of Health Guide, 2015. Available: http://pohg.org.uk/support/downloads/goodpractice_ac.pdf. In a similar vein, Richard Wilkinson notes: "equality is substantially a precondition for the other two [liberty and fraternity (friendship)]: for *liberty* because only in the more egalitarian and less hierarchical societies can the burden of social inferiority and subordination be reduced; and for *friendship*, because we tend to choose our friends among our equals and find it difficult to negotiate the social distance created by differences in wealth and power." Wilkinson R. Commentary: Liberty, fraternity, equality. *Int J Epidemiol* 2002;31(3), p. 540.

[4] Navarro V. Why we don't spend enough on public health: an alternative view. *Int J Health Serv* 2011;41(1), p.118. He next writes, "There is overwhelming scientific evidence that the public's health depends primarily on political, economic, social, and cultural factors."

is hard for any individual in a society to escape the adverse contextual effects of income inequality (Subramanian & Kawachi, 2004). Support for this claim has been provided, using the same U.S. dataset (NLMS) for this study, by Wolfson and colleagues, who demonstrated via simulation that the ecological correlation of state-level income inequality and mortality was too large to be explained by the absolute income effect alone (Wolfson, Kaplan, Lynch, Ross, & Backlund, 1999).

There are many different ways that income inequality is measured. The Gini coefficient is a widely used summary measure of income distribution derived from the Lorenz curve, plotting the proportion of aggregate income earned by a population (on the vertical axis), ranked from the poorest to the wealthiest households in that population (on the horizontal axis). The Gini is calculated as the ratio of the area between the 45-degree line of equality among all household incomes and the Lorenz curve, over the total area under the line of equality, and ranges from 0 (perfect equality) to 1 (perfect inequality) (Kawachi & Subramanian, 2014) p. 152. This measure is routinely reported by the U.S. Census Bureau for states and other geographic regions and is the most widely-used measure for operationalizing income inequality in the public health literature (De Maio, 2007). However, the Gini is problematic for at least two reasons. First, when the Lorenz curves intersect, different patterns of income distribution can yield the same value for the Gini coefficient. This property of Lorenz curves makes it difficult to compare Gini values and may confound tests of the income inequality hypothesis. Second, the Gini is most sensitive to inequalities in the middle of the income spectrum, so it is not the best measure when the locus of interest is the bottom or top of the distribution (De Maio, 2007).

A variety of indices are available in addition to the Gini, each with benefits and limitations (Allison, 1978; De Maio, 2007). The Atkinson index allows for a weighting of

different parts of the income spectrum by incorporating a sensitivity parameter ϵ, which can range from 0 (where one is indifferent to the nature of the income spectrum) to infinity (where one is concerned only with the income position of the lowest group). Another measure is the coefficient of variation, calculated by dividing the standard deviation of the distribution by its mean. Yet another is the decile ratio, used in the current study, which allows a form of sensitivity analysis by using different ratios (e.g., 10:90, 20:80, and 10:40). The generalized entropy index is a family of indices also incorporating a sensitivity parameter to weight inequalities in different parts of the income spectrum. Finally, there is the proportion of total income earned by the n^{th} percent of the population, also utilized in the current study. Allison showed that three measures in particular—the Gini, the coefficient of variation, and the Theil index (the generalized entropy index where the sensitivity parameter is 2 and most sensitive to inequalities at the top of the income distribution)—meet two key criteria for measures of inequality. These criteria are scale invariance (multiplying everyone's income by a constant leaves the degree of inequality unchanged, and the principle of transfers (the measure increases whenever income is transferred from a poorer person to a richer person, regardless of the amount of income of either person or the amount transferred) (Allison, 1978). In the current study, we use the Gini for its scaling (0-100) properties, its comparability to the analyses in paper two, its general acceptance in the public health literature and its desirable properties. We also supplement it with multiple decile ratios and the proportion of total state income earned by the n^{th} percent of the population where n is the 50^{th} percentile or population below the median, so that we can compensate for the decreased sensitivity of the Gini to the inequalities in the lower and higher ranges of the income inequality distribution.

There have been numerous studies over the past two decades linking contextual effects of income inequality and health outcomes, such as mortality. For example, using the Gini coefficient derived from the 1991-1993 Current Population Survey and constructing state-level Gini categories, and controlling for state-level poverty in a prospective, multilevel study, Lochner et *al.* followed 546,888 respondents to the National Health Interview Survey for eight years. They found that individuals living in high-income-inequality states were at increased risk of mortality (RR = 1.12; 95% CI 1.04 – 1.19) compared to individuals living in low-income-inequality states, supporting the contextual effect of income inequality. However, the authors did not control for state-level percent black. In stratified analyses near-poor non-Hispanic whites were at highest risk (Lochner, Pamuk, Makuc, Kennedy, & Kawachi, 2001).

In a very recent study using the Gini coefficient from the 2000 U.S. Census, Pabayo and colleagues again constructed state-level Gini categories or quartiles of the Gini coefficient to predict prospectively the association between U.S. state income inequality and incidence of heart attack in 34,445 respondents to the National Epidemiologic Survey on Alcohol and Related Conditions. They compared respondents at baseline (2001-2002) and follow-up (2004-2005) and used weighted multilevel modelling controlling for multiple individual- and state-level covariates. The authors found that living in a state with higher income inequality was associated with increased risk of heart attack, with the adjusted odds ratios increasing stepwise with each quartile of increasing income inequality. The authors thus confirmed an association between the leading cause of death in the United States and state-level income inequality. The findings are compatible with both a relative deprivation/social comparisons hypothesized mechanism and with a contextual mechanism (decreased social cohesion and underinvestment in social/public

goods), though neither mechanism was directly tested in the study (Pabayo, Kawachi, & Gilman, 2015).

Despite such evidence, controversy surrounding the claim for a contextual effect or theory should not be understated. The theory has been challenged repeatedly and on a number of fronts, which Kawachi and Subramanian call "flies in the ointment" in their textbook chapter on income inequality (Kawachi & Subramanian, 2014) pp 137-146, addressing each major criticism in turn. Economists Angus Deaton, Christina Paxson, Anne Case, and Darren Lubotsky have been among the more vocal critics of the theory. In 2003 Deaton summarized some of these criticisms or points of differing foci between economists and epidemiologists concerning the evidence for relationships among health, income, and income inequality (Deaton, 2003). Deaton cites his and Paxson's work using both aggregated U.S. birth cohorts and individual-level data. They find that conditional on education, increases in cohort average income are hazardous to health, and claim consistency with the work of Gerdtham and Ruhm (Gerdtham & Ruhm, 2006; Ruhm, 2005, 2006, 2007), and more recently Bezruchka (Bezruchka, 2009), on the effects of business cycles on mortality. This literature finds that for developed countries mortality is procyclical, meaning it goes up with economic expansions and down with contractions, and not countercyclical (the opposite), as might be expected. Deaton notes that the contrast between the positive effects of individual income and negative effects of aggregate income on health is an "unresolved puzzle" (Deaton, 2003). Also, he cites an important article (also cited by Kawachi as causal evidence for an effect from income to health) that effectively eliminated the channel that runs in the opposite direction (from health to income) by studying children, where correlations between poor health and low income cannot be attributed to children's low income (Case, Lubotsky, & Paxson, 2002). Case and colleagues find that children's health is closely associated

with long-run household income, and adverse effects are cumulative, such that children arrive at adulthood with lower health status and educational attainment. These deficits likely compromise poor children's earnings ability in adulthood, such that the "gradient" in health status among adults is due to poor health status and low income in childhood.

The legitimate question then arises, if the damage is already done by adulthood, should the policy focus be on childhood health and education rather than on redistribution of household incomes? It is prudent that policy continue to focus in childhood and especially in the few years of life when the health trajectory across the life-course is largely determined through the processes of biological embedding of early life experience (Bezruchka, 2015; Hertzman, 1999) and cumulative damage or weathering (Shonkoff, 2010; Shonkoff, Boyce, & McEwen, 2009; Shonkoff & Levitt, 2010). However, a policy focus on adulthood is also important because, from a different view health trajectories are determined before conception (through assortive mating/mate selection processes) and in utero through biologic processes in which the developing organism responds to the anticipated type of environment in which he/she will be raised (*op. cit.* above). Further, these trajectories are intergenerational—through both epigenetic mechanisms and the repetition of adverse social conditions in the tail of the social gradient, which exhibits "densely-woven patterns of disadvantage" (Goldberg, 2012; Powers & Faden, 2006). Thus, many of the households potentially affected by income redistribution would be those with young children whose basic needs are not being met and who would be helped, however late in the process of health demise, by the reduction in stress and hardship that income would bring into the household (assuming it is put to use for beneficial purposes). This assumption is reasonable because without it social cash transfers (in developing countries) and various social protection programs (in developed countries) would not work, and yet the

evidence suggests that they do provide health benefits, particularly to young children (Glymour, Avendano, & Kawachi, 2014) and so could potentially help to break an intergenerational transmission of disadvantage.

A focus on education is also important, but it must be education in context. It is easy for researchers to believe that education will improve life chances and social status, but maybe that is because it has for them. The value of education is determined largely by the demand for the knowledge and skills that it affords, and if an economy is non-functioning or contracting, or if growth is confined only to its uppermost echelons rather than diffused more broadly, and if competition for the few available high-paying, high-status jobs is intense, education will not pay off for most. This fact has been mentioned in the developing country context by William Easterly in a chapter titled "Educated for What?" (Easterly, 2001), but it could also be considered true in developed countries, particularly in light of the Boomerang Generation in the United States and the "age of austerity," in which in many cases the opportunities for children in developed countries are less than those afforded their parents (often with less education than their children have). In the United States the income achievement gap has steadily increased over the past 50 years and college enrollment and completion rates have stagnated among lower-income students, so that education has tended to widen inequalities rather than reduce them (Reardon, 2013).

Also, there is an emerging literature in demography using the Human Mortality Database that has examined lifespan inequality (variance of age at death), considered the defining measure of social and health disparity, and life expectancy conditional on survival to certain ages. The usual conditional survival age is 15 [e_{15} for remaining life expectancy at age 15, and $V(15)$ for the dispersion of "adult" deaths, excluding infant and child mortality]. In a study of 40 developed countries covering the time periods 1840 to 2009, Vaupel and colleagues found that in all years

studied the leading countries in life expectancy were also the leaders in life disparity. Though only 38% of adult deaths were premature, 84% of the increase in adult life expectancy resulted from averting premature deaths. Reduction in life disparity resulted from reductions in early-life disparity (premature mortality), while late-life disparities were relatively constant (Vaupel, Zhang, & van Raalte, 2011). Using the same database, Gillespie and colleagues identified a divergence in age patterns of mortality post World War II, which appears to drive international divergence in lifespan inequality. In particular, early in the 1980s a mortality increase in young males led to a continuation of high lifespan inequality in the United States, while in Canada there was a continued decline in lifespan inequality. For the developed countries as a whole since World War II, mortality change varied most at young working ages, particularly for males. From these results, the authors expect that as long as mortality stagnates at young ages through lack of progress on social determinants and declines steadily at old ages, through advances in health technologies, for example, increases in lifespan inequality will be a common feature of future demographic change. This observation is another way of saying that social and health disparities will become an embedded demographic feature of most developed countries, breaking from historical patterns of rising life expectancy coupled with falling lifespan inequality.

A recent study in *Health Affairs* examining trends in U.S. life expectancy disparities from 1990 to 2008, by race and educational level at age 25, lends support to the above findings. Alarmingly, U.S. men and women with less than a high school education had life expectancies not much better than those of all adults in the 1950s and 60s. Even more striking, combining race and education, white men with 16 years or more of education had life expectancies of 14.2 more years than black men, and white women 10.3 more years than black women. The authors suggested there were at least "two Americas" based on education level and racial-group

membership, in terms of life expectancy (Olshansky et *al.*, 2012). Thus, premature mortality among young, working-age adults and particularly males in the United States appears to be an important focus for policy intervention if the goal is to reduce lifespan inequality, in addition to the extant focus on young children.

One line of criticism of the contextual theory is that something correlated with income inequality, rather than income inequality itself, is the real cause of variations in health outcomes. Using ecological data, an important study by Deaton and Lubotsky claimed that, conditional on the fraction black, neither state-level nor Metropolitan Statistical Area (MSA) mortality are correlated with income inequality. They found that mortality rates were higher where the fraction of blacks was higher, both due to higher black mortality rates and lower incomes, but also due to white mortality rates being higher in geographic regions where the fraction of blacks was higher (Deaton & Lubotsky, 2003).

This critique was subsequently tested in two independent epidemiological studies using multilevel models in which race was controlled at both the individual and state level (as fraction of state population that is black) for two health outcomes: self-rated health and individual mortality. Neither study supported the claim that the association was confounded by racial composition. In the first study, using pooled Current Population Survey data on 201,221 adults in all 50 states and controlling for a range of demographic factors, the authors found that a 0.05-increase in the Gini coefficient increases the odds ratio of reporting poor health by 1.39. Controlling for fraction black at the state level reduced the odds ratio to 1.30. There was no significant association between fraction black in a state and poor self-rated health. Rather, it suggested that whites residing in areas such as the Southeast with higher fraction black had worse health not because they lived in proximity to blacks, but because they lived in areas of

higher income inequality (Kawachi & Subramanian, 2014; Subramanian & Kawachi, 2003a). The second study used the data from the NLMS on 521,248 adults in all 50 states. It found that 1990 state-level income inequality was associated with a 22% increase in state-level mortality rates for men aged 25-64 and a 5% increased risk for women aged 25-64 after adjusting for race at both individual and state levels. Also, fraction black was associated with a 14% increased risk of mortality among men and a 22% increased risk among women, controlling for state-level income inequality (Backlund et *al.*, 2007; Kawachi & Subramanian, 2014). Thus, the critique that state-level racial composition explains the contextual association is not supported by more recent and more rigorous epidemiological studies, though there could still be residual confounding by other state-level characteristics.

An important meta-analysis by Kondo and colleagues summarizing the literature through 2009 found that, among the 27 multilevel longitudinal and cross-sectional studies meeting the inclusion criteria for their analysis, each 0.05-unit increase in the Gini coefficient was associated with an increase in total mortality rate of 7.8% (95% CI: 5.8 to 9.8%) (Kondo et *al.*, 2009). The authors also detected what appeared to be a threshold effect at a Gini of about 0.3—they found a stronger relationship between income inequality and mortality for nations with a Gini ≥ 0.3 compared to those with a Gini < 0.3. The 0.05-unit change in the Gini is in line with changes (increases) in this index in recent decades for the United States, with a current post-tax-and-transfer Gini of approximately 0.4. And though some might argue that a 7.8% increase in mortality is a small effect, as opposed to the large effects on mortality attributable to absolute poverty (in the realm of 200% or so), the latter apply only to the 15% of persons below the poverty threshold in the United States, for example. In contrast, the 7.8% excess applies to *all individuals* exposed to higher income inequality in regions with higher Gini coefficients, relative

to those with lower coefficients (Kawachi & Subramanian, 2014). The Kondo et *al.* meta-analysis suggests that one in three deaths in the United States results from high income inequality, as defined by a Gini above the 0.3 threshold (Bezruchka, 2014). The number of excess U.S. deaths exceeds the annual number of deaths from heart disease, the leading cause of death in the United States.

Another important study estimated the number of deaths due to social factors in the United States, using a meta-analysis of 47 studies and calculating a population attributable risk fraction for each social factor. It found that approximately 119,000 deaths or 5.1% could be attributed to area-level income inequality, defined as the percent of the adult population living in counties with a Gini coefficient at or above the 25th percentile (Galea, Tracy, Hoggatt, DiMaggio, & Karpati, 2011). The authors note that past estimates of the attributable fraction for mortality have varied between 9% and 25% depending on the age group studied.

More recently, Torre and colleagues tested the income inequality hypothesis using the Human Mortality Database on panel data for 21 developed countries over 30 years (Torre & Myrskyla, 2014). The analysis used a panel-data regression, with country and time fixed effects, and controlled for GDP per capita. The authors found that income inequality measure by the Gini coefficient was significantly associated with mortality among males and females ages 1-14 and 15-49, and for females aged 65-89, though less strongly than for the younger age groups. They speculated that the stronger relationship among younger persons is plausibly mediated through underinvestment in social services to mothers and children and inadequate monetary support to families, citing Lundberg and Marmot (Lundberg et *al.*, 2008; Marmot, 2003). They also suggested adverse health effects, including premature mortality, in children may reflect parental hardship and tradeoffs in having to invest more time and energy on work and less on their

children's well-being. Further, a stronger effect for men aged 15-49 agrees with the findings of Backlund and colleagues (Backlund et *al.*, 2007) in the United States and may suggest a line of future inquiry concerning cause of death analysis. The authors speculate that deaths due to accidents, violence, or risk-taking behaviors may be more closely related to inequality among males, due to more intense status competition (Torre & Myrskyla, 2014).

This study uses the 50 states in the United States plus the District of Columbia as the contextual unit of analysis for both data quality and theoretical reasons. First, there are relatively good data for and variance among state Gini coefficients and other measures of income inequality. Second, findings for states have yielded among the most consistent positive associations between income inequality and mortality (Subramanian & Kawachi, 2004). Third, there is significant variation in state-level social protection programs, which tend to vary with the degree of income inequality, and each of which may contribute to variations in levels of premature mortality (Kawachi & Subramanian, 2014). Fourth, as geographic areas studied become smaller, within-area variance declines relative to between-area variance, so that the direction of the association might reverse, where more income-homogenous (lower income inequality) regions might be homogenously poor and therefore have worse health outcomes. Finally, as a republic, the state is a natural focal point because states can design and implement policies significantly affecting the distribution of income—for example, state-level EITC programs, which have been shown to have significant impacts on the state-level percentages of households living in poverty.

This study attempts to address a number of weaknesses among prior studies of the contextual effect of income inequality on mortality. While a contextual effect has been estimated for states in the United States (Backlund et *al.*, 2007), few prior studies have followed a large

enough number of individuals forward for a long enough period of time. This study follows 394,208 persons forward for up to 10 years, the time thought to correspond to the lag required to capture the full effects on mortality (Blakely, Kennedy, Glass, & Kawachi, 2000; Zheng, 2012). This study explicitly addresses confounding by race and by poverty by controlling for them at both the individual and state level. Further, the study period (1995 to 2011) corresponds to one during which national-level income inequality has achieved its highest recorded level in American history, offering a potentially larger signal and more variation with which to detect and potentially quantify the effect. In addition, it complements the study in paper two by including the (aggregate, state-level) household Gini coefficient among the income inequality predictors.

Conceptual Model

Kawachi and Subramanian, citing *The Price of Inequality*, by Nobel Laureate in Economics Joseph Stiglitz (Stiglitz, 2012), describe a mechanism of action for the contextual effect (Kawachi & Subramanian, 2014). Echoing Piketty and Saez, Stiglitz claims that the rent-seeking behavior of the top 1% imposes a tax on the 99%, via a loss of social cohesion and a degrading of the quality of life for most everyone except the wealthiest. This effect proceeds in two steps. First, as wealthy people become wealthier, they functionally secede from the rest of society, for example, by segregating themselves into their own communities, schools, health clinics, etc. Because they do not use public services (public schools, hospitals, etc.) themselves, the wealthy become less and less inclined to subsidize others for their use. Secondly, the wealthy call for tax relief. Because power becomes concentrated in the upper classes, they can use their resources to get policies that benefit themselves at the expense of those lower down the socioeconomic ladder. Princeton economist Angus Deaton, author of his own book on wealth

and income inequality (Deaton, 2013), provides direct evidence for this effect among OECD countries—those with the largest shares of income going to the top earners have also passed the largest tax cuts to those same earners. As Deaton states,

> …if democracy is compromised [through extreme wealth concentration], there is direct loss of well-being because people have good reason to value their ability to participate in political life, and the loss of that ability is instrumental in threatening other harm. The very wealthy have little need for state-provided education or health care; they have every reason to support cuts in Medicare and to fight any increases in taxes…. To worry about these consequences of extreme inequality has nothing to do with being envious of the rich and everything to do with the fear that rapidly growing top incomes are a threat to the wellbeing of everyone else (Deaton, 2013) pp. 213-214.

Extreme societal-wide economic inequalities are a threat to economic growth and democratic governance (Stiglitz, 2012). There is even evidence from social psychology that the rich are less empathetic or caring toward the hardships of the poor, compared to those with lower socioeconomic status (Stellar, Manzo, Kraus, & Keltner, 2012).

The mechanisms linking contextual income inequality specifically to worse health remain incompletely characterized. As Kawachi and Subramanian have noted, "it is an almost universal challenge in social epidemiology to theorize the transition from macro-scale phenomena (such as the societal distribution of income) to micro- or individual-level consequences (in this instance, health outcomes)" (Kawachi & Subramanian, 2014) p. 135. They note that various authors have posited that inequality generates more anxiety, shame, depression and other negative emotions that harm health (Kawachi & Kennedy, 2002; Kawachi & Subramanian, 2014; R. Wilkinson & Pickett, 2009). In the United States, the pervasive belief in the "Horatio Alger myth" leads most Americans to much more readily accept that reward is relative to level of effort and that family wealth has little to do with advancement up the socioeconomic latter (Haskins, Isaacs, & Sawhill, 2008). Yet social mobility in the United States is lower than that in most other OECD nations. For instance, one measure of social mobility—the correlation between incomes of

fathers and sons—is 0.47 in the United States, much higher than in Norway (0.17), Sweden (0.27), or France (0.41) (Corak, 2013). As Kawachi and Subramanian note, the discordance between the myth and reality leads many to strive and fail, and then to blame themselves (Kawachi & Subramanian, 2014). And according to social strain theory (Merton, 1957), this discrepancy between what society asks and what it is (in the vast majority of cases) possible to achieve given the available opportunities, leads to alienation, anxiety, frustration and maladaptive coping behaviors.

As Stiglitz (Stiglitz, 2012) argues, mainstream economics assumes individuals have well-defined, (usually exogenous) preferences and rational expectations and perceptions. But just as advertising firms can shape preferences to induce people to buy products, those in the 1 percent can and have shaped the beliefs among the other 99 percent—beliefs about notions of fairness and efficiency, the proper role of government and the market, and even the degree of inequality that actually exists today. Most people think that the level of wealth inequality is much lower than it actually is, and across the demographic spectrum they express preferences for much, much lower levels. The top 20% of the U.S. population holds about 85% of the wealth, while respondents in one study thought the top 20% held 60% of the wealth and expressed preferences for an ideal distribution of 30% (Norton & Ariely, 2011). The field of behavioral economics and the notion of endogenous preferences, formulated by Samuel Bowles (Bowles, 1998) and others, has begun to provide insights about the importance of social context on decision-making, demonstrating that there are systematic misperceptions and consistent biases in judgments and framing effects, which can be manipulated.

Secondly, psychological research indicates that individuals process information that is consistent with their prior beliefs differently from how they process information that is

inconsistent. A large literature has emerged on this "confirmatory bias"—that information that is consistent with prior beliefs is more likely to be remembered, seen as relevant, and to reinforce beliefs, while that which is inconsistent is more likely to be ignored, discounted, or forgotten. "Equilibrium fictions" can result from this process, which are beliefs maintained strongly because the evidence people see is fully consistent with prior beliefs (Stiglitz, 2012). Thus, in the social sciences beliefs can affect reality, and beliefs about how an economic system functions can affect how it functions.

What is more, ideas and perceptions (beliefs) are *social constructs*, meaning that the willingness to hold certain beliefs is related to other people holding similar beliefs. The social construction of knowledge is fundamental to qualitative research, and it is critical to understanding why and how beliefs and social realities can remain stable or change rapidly. It suggests how social separation among groups can lead to different and incompatible perceptions of reality. If one or more groups has better access to the means for shaping perceptions and beliefs, the evolution of ideas will follow the course that they choose. Also, through processes of social distancing or segregation, those with contrary perspectives can come to be seen (by others and by themselves) as intrinsically different, and may come to act differently and to take on the socially-constructed difference as part of their identity. Thus in some ways, these social constructions can lead to self-fulfilling and self-sustaining prophecies in which social distance and separation on various vitally-important resources for health, such as wealth and income and social connectivity, can become normative. It is a contention of this paper that these less-than-transparent social processes produce contextual effects that can causally determine both the health status of individuals, and the distribution of health in populations.

We hypothesize that state-level income inequality in 2000 will significantly predict individual-level mortality risk up to ten years later, controlling for important potential confounding variables at multiple levels. Second, the measured contextual effect of state-level income inequality will be larger than that reported previously using the same dataset (Backlund et *al.*, 2007) because the data will track subjects over a longer time horizon and from a baseline time point surrounding the year 2000 when state income inequality was higher. We confirm a significant, though subtle contextual effect of income inequality on premature mortality, which varies by inequality measure, gender, and analytical method. The effect sizes are generally in the range of a 1% to 5% increase in the likelihood of premature death for a one standard deviation increase in income inequality, controlling for all other variables in the models, and appear to be larger for males. Because the contextual effect applies to the entire population, it may cause a sizeable number of premature deaths in the United States, especially among males.

Methods

Data and Sample

Data for this study come from the NLMS, a large prospective household survey based on a random sample of the non-institutionalized population of the United States, developed specifically to study the effects of demographic and socioeconomic characteristics on differentials in U.S. mortality rates. The NLMS matches individual records from the Current Population Survey (CPS) (Current Population Survey (CPS), 2012) to the National Death Index (NDI) (National Death Index, 2014). A complete description of the dataset is available in the latest edition of the Reference Manual (National Longitudinal Mortality Survey: Extract and Analysis Files, 2012). We use 12 cohort years from 1995 to 2006, bounding the 2000 U.S. Census, and follow these cohorts forward for 5 to 10 years to investigate death rates in the 25- to

64-year-old age group. We cap follow-up at ten years, which is a common practice when looking for geographical causes because people tend to move around, and after a decade or so, the chances are higher that respondents have moved out of a particular state. State-level data on the household Gini coefficient, percentage of state income for households below the median, percent of residents who are black, and percent of residents in poverty come from U.S. Census Bureau Summary Tables. Sourcing data for state-level decile ratios was problematic because publically available Census Summary Files do not have details on income earned within state-specific deciles, and further, most individual income reporting is top-coded to protect the identity of high income persons. Therefore, we calculate state-level income deciles and aggregate income within each decile using the CPS Annual Social and Economic Supplement (CPS ASEC) data contained within the NLMS for cohort years 1995 to 2004. We then calculate multiple decile ratio measures of income inequality from these data.

Empirical Model

The method used by social epidemiologists to test the contextual theory is multilevel regression modeling comparing exchangeable individuals clustered in communities (states in this case) that differ on the distribution of income (Kawachi & Subramanian, 2014). The multilevel regression model relates the binary outcome death within the five-to-ten-year follow-up period to state-level income inequality. State-level income inequality is measured using: the Gini coefficient; the Palma ratio (ratio of income earned by the top 10% of households to that earned by the bottom 40%); the ratio of income earned by the top 10% (and 20%) of households to that earned by the bottom 90% (and 80%); the ratio of income earned by the top 50% of households to that earned by the bottom 50%; and the percentage of total state income received by households with incomes below the median. We control for individual-level income (or

equivalized household income, in this case) because it is a potential compositional confounder, affecting both area-level income inequality and mortality. By compositional we mean the individual or household-level characteristic (equivalized household income) which comprises the aggregate-level variable of interest (state income inequality)—differences in states could be due to differences in the composition of households within the state, as opposed to contextual effects, which are determined solely at the group or state level. The latter effect is of interest in this analysis.

Additional covariates (demographic controls) at level one included in the model are those known to be associated with individual-level mortality: age in years at time of interview, race/ethnicity, urbanization level, marital status, educational attainment, and presence of a valid social security number. Additional covariates at level two also associated with increased mortality are the percentage of the state residents who are black and state-level poverty rate. The analysis is stratified by sex.

Because these data include follow-up mortality and are therefore time-to-event data over a relatively long time period (up to 10 years) during which individuals will have widely varying times to the event, we adopt a Cox proportional hazards multilevel modelling or mixed effects modelling framework as the most appropriate modelling approach. This method incorporates multilevel regression (a form of mixed or fixed plus random effects modelling—in this case there is a single random intercept for each state clustering the observations) into a survival analysis framework. The Cox proportional hazard multilevel model takes the form:

$$h_{ij}(t) = h_0(t)\exp(X_{ij}^T\beta + Z_j^T\mathrm{b}_j),$$

where $b_j \sim N\ (0, V)$ and X and Z represent the matrices for the fixed and random effects, respectively. It is evident from this equation that the covariates have a multiplicative effect on

the baseline hazard function $h_0(t)$, which we assume is a parametric function with a Weibull distribution, which is commonly used to model time-to-event data in which the probability of the event accelerates rapidly with time.

Analyses

We fit mixed effects proportional hazards random intercepts models using the mestreg estimation command in Stata 14 to estimate all regression models. Full models include the level-1 demographic control variables, the key independent variable (state-level income inequality), and controls at level two for percentage black in the population and percent in poverty for each state. We test for significance of the key independent variable, controlling for the individual-level demographic variables and compositional variable equivalized household income, and for potential confounding by race and poverty at the state level. As a robustness check on these models, we also run Cox proportional hazards models with state fixed effects and standard errors clustered at the state level, and include all other controls as in the multilevel mixed effects models (see Appendix). Due to high multicollinearity potentially influencing other parameters in the fixed effects models, we also tested models for males for the 90:10 ratio and household Gini predictors, including all the other individual and state level controls, with fixed effects for the nine census regions rather than for the 51 state-like entities. We ran tests of the proportional hazards assumption for these models (see Appendix). We also attempted to test the proportional hazards assumption for the multilevel Cox models, assuming it should hold, although it is unclear that it should hold—the documentation for this model indicates only that the random effects are assumed to be normally distributed, but we were unable to test this specific assumption. Also, at least one paper seems to indicate that the effect of non-proportional hazards on the fixed effects is consistent with the standard Cox model (as measuring an average

regression effect), nor do non-proportional hazards bias the estimates of variance components on the baseline hazard (Xu & Gamst, 2007).

Results

Table 3.1 shows weighted summary statistics for males and females from the NLMS 1995 – 2006 cohorts dataset. Though the sample of women was slightly larger, there were 2,617 more deaths among men. Mean ln equivalized adjusted income was slightly higher among men, but most demographic variables were similar between the sexes. Slightly more men reported being currently married but also having never married, and slightly fewer reported being widowed, single, or divorced. More men were employed and in the labor force.

Table 3.2 shows the U.S. Census 2000 state-level characteristics included in the Cox proportional hazard multilevel analyses, ordered by census region and by state. The Gini coefficient for household ranged from 0.402 (Alaska) to 0.549 (District of Columbia, DC). The percentage of state income for households below median ranged from 15.37 (DC) to 22.93 (Utah). The ratio of income held by the top 10% compared to that held by the bottom 90% ranged from 15.52 (Alaska) to 74.05 (DC). The ratio of income held by the top 20% compared to that held by the bottom 80% ranged from 3.15 (Nevada and Utah) to 6.73 (DC). The ratio of income held by the top 50% compared to that held by the bottom 50% ranged from 2.89 (Nevada) to 5.13 (DC). The ratio of income held by the top 10% compared to that held by the bottom 40% (Palma ratio) ranged from 1.50 (Alaska) to 3.67 (DC). The percent of state residents who were black ranged from 0.3% (Montana) to 60.0% (DC). Finally, the state poverty rate ranged from 6.5% (New Hampshire) to 20.2% (DC). Thus, in 2000 by all measures, state-level income inequality was highest in DC (which is not a state and which may help explain why it suffers so, given that the United States is a republic), as was percentage in poverty and the

percentage black, and state-level income inequality was lowest in the western states Alaska, Utah, and Nevada. Table 3.3 summarizes the state-level characteristics in Table 3.2 for males and females (mean, SD, and range).

Table 3.4 shows the hazard ratios for death within the 10-year follow-up period, by income inequality measure and sex, from the Cox proportional hazards multilevel analyses. It is interesting to note that while nearly all state-level measures of income inequality are significant, the hazard ratios tend to be small and somewhat erratic. We note that the hazard ratios for variables at the state level in these multilevel models do not have the standard interpretation of typical hazard ratios. Therefore, we can not precisely judge the actual value of the effects, but rather must make more general statements about their direction and magnitude. First, the direction of the effect of the Gini coefficient is opposite to that predicted by the contextual theory—a higher Gini appears protective against premature mortality, especially for males. This result is difficult to explain, but may involve multicollinearity with the two other state-level control variables—percent black population and state poverty rate, or the failure of the Gini model to satisfy the proportional hazards assumption, which is assumed to hold for a parametric Weibull survival function. To further evaluate this unanticipated result, we plotted raw values for percentage of male respondents by state surviving to five years versus three income inequality measures (Figures 2 & 3), with particular focus on states with large populations (California, New York, and Texas) which would exert disproportionate influence on the model.

Also, we evaluated distributions of both the linear predictors and residuals for the multilevel Cox models for males for the 90:10 ratio and household Gini predictors and including all controls. We found the linear predictors and residuals to be non-normally distributed (right-skewed, with most values concentrated to the left of the mean) and with kurtosis > 3, or having

extreme values. In the Cox models for males with region fixed effects and standard errors clustered at the state level, for both the 90:10 ratio and household Gini predictors the proportional hazards assumption was rejected.

The indicator percentage of state income for households below the median generated significant results in the anticipated direction for both males and females: the less income held by the lower half of the income distribution, the higher the mortality risk, supporting the contextual theory. Among females, the 90:10 ratio was significant in the anticipated direction, however, the magnitude of the effect appears to be extremely small. Among both males and females, the 80:20 ratio was significant in the anticipated direction (HR 1.108, 95% CI 1.0857 – 1.1307; HR 1.087, 95% CI 1.0618 – 1.1125, respectively). Among males and females, the 50:50 ratio models yielded similar results to the above, with slightly larger hazard ratios (HR = 1.138 95% CI 1.1117 – 1.1667; HR = 1.112, 95% CI 1.0803 – 1.1439). For males the 90:40 (Palma) ratio model yielded a statistically significant hazard ratio in the anticipated direction (HR = 1.059, 95% CI 1.0341 – 1.0837). Models including state poverty rate alone as the predictor variable for both sexes did not yield statistically significant hazard ratios.

In Table 3.5 we summarize the results from Table 3.4 in terms of a percentage change in the risk of mortality due to a one standard deviation change in each state-level measure of income inequality. As noted above, the changes for the Gini measure in multilevel models are in the direction opposite to that predicted. The measure "percentage of state income for households below the median" stands out as having extremely large negative percent changes. Otherwise, the percentage changes are roughly in the range of a 1% to 5% increase in mortality for a one standard deviation increase in income inequality, controlling for all other variables in the models. It is unclear why the models for "percentage of state income for households below the median"

produced such large, negative percent changes – we expected negative changes for this measure, but not of this magnitude.

Discussion

We find significant effects for state-level income inequality as a predictor of premature mortality in models controlling for both percent black and percent poverty at the state level and equivalized household income at the individual level and including multiple individual-level demographic controls. However, the associations vary markedly by inequality measure, gender, and the regression method applied, and in some cases (e.g., for the Gini coefficient in multilevel models for both males and females) are opposite to expectation. One explanation for the latter is that the direction of the association might reverse if more income-homogenous (lower income inequality) states are homogenously poor and therefore have worse health outcomes due to poverty. But we control for state poverty and still find a protective effect for the Gini coefficient. Secondly, for the Gini coefficient measure, if the Lorenz curves intersect, different patterns of income distribution can yield the same value for the Gini coefficient and confound tests of the income inequality hypothesis. Certainly, the variances among states for several of the income inequality measures in Tables 3.2 and 3.3 are low and may have contributed to the difficulty estimating a state-level effect in several of the multivariate models. For example, all the Gini coefficients are all upwards of 0.4 and only one (for DC) is above 0.5. That models with state fixed effects seemed to perform better suggests that fixing the between-state variation in order to completely control for unobservable confounding and just using the within-state variation in these models provided a clearer picture of the contextual effect. However, this approach had the possible cost of wiping out an effect among females and of introducing excessive multicollinearity into the models with uncertain effects on the remaining covariates. Finally, as

the proportional hazards assumption tests for the Cox fixed effects models indicated, for both the 90:10 ratio and the Gini coefficient and for both functional forms of time, this assumption was violated, indicating the effect is not stable over time. Therefore, the predictors of interest might be handled differently in future modelling, for example by including an interaction with time, where time is centered around the year 2000. Also, categorizing the Gini coefficient into [k] groups may be a promising modeling approach and is prevalent in the literature.

For some, the debate regarding the contextual effects of income inequality is settled, but for others it is not. Our results do not provide a definitive answer to the question of mortality effects from inequality, but they may make the path to that answer a bit clearer by showing the sources of systematic variability in the hazard ratios for mortality with respect to a particular income inequality measure, and among different measures. Also, the hazard ratios (and percent changes in mortality rates) reported here are, by-and-large, extraordinarily small. How is one to interpret a statistically significant HR of 1.002 for the 90:10 ratio for females in Table 3.4, for example? The mean for this variable is 26.47 with a standard deviation 4.92 or approximately 5. Therefore, a one standard deviation increase in the 90:10 ratio increases the mortality hazard by about 1 percent (5 x 0.002), after controlling for all other variables in the model. If a state with 2 million people and a 90:10 ratio with HR=1.002 has, say, 20,000 deaths a year, this extremely small yet statistically significant increased hazard of premature mortality equates to 200 people (in just that state), and multiplied by 50 states becomes 10,000 premature deaths, a far from trivial effect. The majority of percent changes reported here (in Table 3.5) are consistent with the magnitude of the contextual effect reported in other studies and also with the expectation that we are dealing with a subtle, population-level effect which requires large sample sizes and carefully-

chosen models and compositional controls to detect, let alone quantify. However, we do find support for the contextual effect of income inequality in most of the models reported here.

In addition, we ran state fixed effects models to effectively control for unmeasured state-level contextual factors in addition to those that were measured—state-level poverty rate and percent black, and this method reduced the contextual effect to non-significance only in the models for females, not males. This econometric method has been called a "sledgehammer approach" because we bludgeon out all the between-state variance, including any that might mediate the effect of interest, yet the effect still stands for males. However, it should be noted that these models did not pass global tests of the proportional hazards assumption and therefore the hazard ratios may not be reliable. Additionally, the degree to which individual-level predictors should be considered confounders versus mediators in multilevel models has been questioned in the literature. If individual-level education or income are related to social or political factors that are also related to income inequality, then these factors could be seen as mediators and their inclusion in the models might attenuate the association between contextual inequality and mortality. It is therefore important to base the inclusion of controls at both individual and contextual levels on carefully considered causal pathways. An additional limitation of the current analysis is that the measures of income inequality were all from a single year (2000) while "exposure" to this level of inequality could range from cohort years beginning in 1995 to those in 2006, so that the true duration of exposure is inconsistent and imprecise. However, we have modelled state-level income inequality indicators as time-invariant characteristics measured at the 2000 Census, and it is probably a safe assumption that changes over time in these measures were minimal, and that the rank-ordering of the states with regard to these measures was largely preserved. Further, we followed respondents forward for at least five

and up to ten years to capture the lagged effects reported in the literature on income inequality and mortality. Differential follow-up and/or inadequate length of follow-up must be included as a limitation of this analysis.

Notwithstanding the arbitrary nature of causal criteria, Bezruchka (Bezruchka, 2014) argues that standard epidemiological criteria for assessing causality, originally proffered in the surgeon general's report on smoking and health but most often attributed to Bradford Hill, and anticipated by Mill and Hume (Rothman & Greenland, 2005), are satisfied for a causal relationship between economic inequality and poor population health. These criteria include consistency across a large number of studies involving different investigators, populations and time periods; a dose-response relationship; no other contending explanations posited; and biological plausibility, with likely mechanisms of action, for example the biology of the stress response involved in social comparisons. Bezruchka and others claim that the extent of inequality in a society reflects, or is a marker for, the range of caring and sharing in that society, with less equality indicating less sharing. Poorer persons or families struggle for acceptance by and full participation in society, and rich persons or families pay a price, for example, through increased crime, overburdened health care systems, and increasing health care costs. The whole economy suffers from cumulative wasted potential and idleness associated with high levels of un- and underemployment, sapping demand from the economy, increasing demands and strain on those who remain employed, depressing wages, and deepening the levels of inequality still further through a hollowing out of a robust middle class, a hallmark of a well-functioning economy (Stiglitz, 2012).

Pickett and Wilkinson recently conducted an exhaustive review of income inequality and health within an epidemiological causal framework similar to that noted above and updating

earlier evidence with more recent studies, finding that the body of evidence taken as a whole strongly suggests that income inequality affects population health and well-being (Pickett & Wilkinson, 2015). The authors note that the major causal criterial of temporality, biological plausibility, consistency, and lack of alternative explanations are well supported. They note that a mechanism for the effect has been elucidated and inequality operates through subtle yet powerful, population-wide psychosocial effects. They claim that "larger income differences increase social distances, accentuating social class or status differences" (Pickett & Wilkinson, 2015) p. 324. Interestingly, the authors note, "A paper by Deaton in 2003 (Deaton & Lubotsky, 2003), reported that the proportion of black residents in states and Metropolitan Statistical Areas of the USA explained the income inequality–health association. This paper continues to be cited as evidence that income inequality does not affect health, despite the fact that several more recent studies find that ethnic heterogeneity does *not* confound the income inequality–health association in the USA" (Ash & Robinson, 2009; Ram, 2005; Subramanian & Kawachi, 2003a, 2003b, 2004).

The authors close their causal review with reference to a provocative article by Schrecker titled "Can health equity survive epidemiology? Standards of proof and social determinants of health," in which he likens the issues surrounding social determinants of health to environmental risk factors and health, and notes the need to make value judgments in the face of scientific uncertainty and to entertain methodological pluralism. He notes the need to explicitly engage ethical issues surrounding the choice of standards of proof, but the stakes are high, so this arena quickly becomes political, with many who benefit from the status quo (and from null findings) interested and engaged in manufacturing uncertainty. One is reminded of past tobacco industry tactics or current climate change deniers. In fact, there are many parallels with climate change

and income/social status inequities in that both are subtle, all-encompassing processes and phenomena that if accepted as true would require major transformations of social, political, and economic systems. Yet these changes are both imaginable and feasible with the knowledge we have, and indeed some (e.g., movement towards renewable energy sources) are already underway. As Schrecker notes, adopting too high a standard of evidence may lead to it never being considered strong enough (Schrecker, 2013). One could equally ask, "Can health equity survive public policy?" To the degree that it requires a singular, reductionist econometric or epidemiological methodological approach which attempts to treat social sciences as physics or other "hard sciences," the answer is no. To the degree that it treats theory as fact, rather than as continually evolving and changing terrain riddled with uncertainties, the answer is no.

Echoing Deaton in the first chapter of his 2015 text titled *Inequality: What can be done?*, Sir Anthony B. Atkinson distinguishes the concepts inequality of opportunity and inequality of outcome and argues that both are important. He notes the first is an ex ante concept—that everyone should start from a level playing field, while the second is concerned with ex post outcomes. Though many argue that concern for the latter is illegitimate, Atkinson claims they are wrong for at least three reasons. First, even if ex ante equality of opportunity is present, people may exert plenty of effort but fall on bad luck—a humane society would provide to those for whom the outcome is hardship. Second, there is a difference between competitive and noncompetitive equality of opportunity, the latter ensuring that all have an equal chance to fulfill their *independent* life projects. The former implies that we all have the chance to take part in a race with unequal prizes (the more common case where there are ex post unequal rewards). The existence of a highly unequal distribution of prizes leads us to focus on making sure the race is fair (i.e., on an ex post conceptualization of inequality), and the prize structure is socially

constructed (Atkinson, 2015). Thus, one might argue that in present-day society, the prize structure is such that returns tend to go to already well-endowed individuals (and in turn their offspring), a prescription for increasing inequality far into the future. Third, inequality of outcome is important because it affects equality of opportunity for the next generation, or "today's ex-post outcomes shape tomorrow's ex ante playing field: the beneficiaries of inequality of outcome today can transmit an unfair advantage to their children tomorrow" (Atkinson, 2015) p. 11.

Beyond these practical concerns, there are instrumental reasons to be concerned about inequality of outcomes. Social epidemiologists have argued that unequal outcomes cause a host of social problems, including lack of social cohesion, increased crime, ill health, teen pregnancy, obesity, etc. (Wilkinson & Pickett, 2009). Political scientists and economists have noted how income inequality and money in political arenas bi-directionally poison democratic processes (Stiglitz, 2012). The head of the IMF, Christine Lagarde, noted in 2012 that "recent IMF research tells us that less inequality is associated with greater macroeconomic stability and more sustainable growth" (Atkinson, 2015) p. 12. [See also (Dabla-Norris, Kochhar, Ricka, Suphaphiphat, & Tsounta, 2015) for a recent global perspective on causes and consequences of income inequality.] In addition, Atkinson describes several intrinsic reasons for concern about excessive income inequality, which can be framed in terms of theories of social justice, including concern for the least advantaged in society expressed by Rawls (Rawls, 1971) and the capability theory espoused by Sen, which is concerned with the opportunities open to people given their particular circumstances (Sen, 2000).

Atkinson (and Deaton, above) notes that many economists would argue that the focus should be on poverty rather than on the overall distribution of income or the extent of inequality.

Despite political commitments to reducing poverty and explicit target setting toward that end, wealthy countries have made little progress in reducing domestic poverty rates. For example, since the late 1960's the United States has made little progress on poverty, during which time it has vacillated between approximately 11% to 15%, and at present some 45 million of its citizens live below the poverty line. What's more, the evidence indicates that high poverty in wealthy OECD nations tends to coincide with larger top income shares. In other words, for better or worse, our economic fortunes are interdependent (Atkinson, 2015). The fact that the main determinants of population health are social, economic and political implies that contextual effects (social, economic, or political) will be vitally important to the health of individuals on some level, because we have evolved as highly social animals. Our minds and bodies are exquisitely attuned to the perceived security or threat in our environments—to felt and embodied social integration or hierarchies and inequities. As Stiglitz notes, "the problem of inequality is not so much a matter of technical economics. It's really a problem of practical politics….We are not embracing a politics of envy if we reverse a politics of greed. Inequality is not just about the top marginal tax rate but also about our children's access to food and the right to justice for all. If we spent more on education, health, and infrastructure, we would strengthen our economy, now and in the future…" (Stiglitz, 2015). "Liberty and justice for all" asks that we renew our historical and foundational commitment to a politics of inclusion and equity in our own country.

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TABLES

Table 3.1. Weighted[a] descriptive statistics for the NLMS (1995 – 2006 cohorts) dataset, by sex

	Males (n = 189,416)			Females (n = 204,792)		
Variables	**Mean**	**SD**	**Range**	**Mean**	**SD**	**Range**
Dependent variables						
Deaths over 10 years (n)	10,508			7,891		
Days alive	3,075.51	682.34	1 – 3,653	3,099.68	652.52	2 – 3,653
Sociodemographic variables						
Ln equivalized adjusted income in 1990 dollars	9.90	1.30	0 – 13.98	9.77	1.39	0 – 13.98
Age (years)	42.94	10.65	25 – 64	43.03	10.70	25 – 64
Urban residence	0.72	0.45	0 – 1	0.73	0.44	0 – 1
Race/ethnicity						
Hispanic	0.09	0.29	0 – 1	0.09	0.29	0 – 1
Non-Hispanic white	0.75	0.43	0 – 1	0.74	0.44	0 – 1
Non-Hispanic black	0.10	0.30	0 – 1	0.12	0.32	0 – 1
American Indian	0.01	0.09	0 – 1	0.01	0.09	0 – 1
Asian/Pacific Islander	0.04	0.20	0 – 1	0.04	0.20	0 – 1
Other race/ethnicity						
Marital status						
Married	0.68	0.47	0 – 1	0.65	0.48	0 – 1
Never married	0.19	0.39	0 – 1	0.14	0.35	0 – 1
Widowed/single/divorced	0.14	0.35	0 – 1	0.21	0.41	0 – 1
Education						
HS dropout	0.12	0.33	0 – 1	0.12	0.32	0 – 1
HS plus some college	0.58	0.49	0 – 1	0.61	0.49	0 – 1
College degree or more	0.30	0.46	0 – 1	0.28	0.45	0 – 1
Employment status						
Employed	0.83	0.37	0 – 1	0.71	0.46	0 – 1
Unemployed	0.09	0.28	0 – 1	0.08	0.27	0 – 1
Not in labor force	0.08	0.27	0 – 1	0.21	0.41	0 – 1
Valid Social Security #	0.90	0.30	0 – 1	0.92	0.28	0 – 1

[a] Weighted to represent 147 million persons, an approximation using 2000 Census data for the non-institutionalized population of 25- to 64-year-olds (exact = 146,992,887 from Table P012).

Table 3.2. Income inequality indices, percent black population and percent population in poverty, by census region and state, 2000 U.S. Decennial Census

Census region	State	Household Gini coefficient[a]	Percentage of state income for households below median[b]	90:10 ratio[c]	80:20 ratio[c]	50:50 ratio[c]	90:40 ratio (Palma)[c]	Percent population black[d]	Percent population in poverty[e]
East	Connecticut	0.477	19.00	24.68	4.78	3.70	2.20	9.1	7.9
	Maine	0.434	21.09	22.88	4.17	3.54	2.07	0.5	10.9
	Massachusetts	0.463	19.40	26.87	4.66	3.80	2.28	5.4	9.3
	New Hampshire	0.414	22.55	25.25	4.40	3.69	2.32	0.7	6.5
	New Jersey	0.460	19.69	26.61	4.33	3.53	2.14	13.6	8.5
	New York	0.499	17.50	35.14	5.08	4.04	2.55	15.9	14.6
	Pennsylvania	0.452	20.16	27.74	4.51	3.82	2.33	10.0	11.0
	Rhode Island	0.457	19.49	24.57	5.24	3.93	2.33	4.5	11.9
	Vermont	0.423	21.83	22.07	4.08	3.67	2.13	0.5	9.4
Midwest	Illinois	0.456	20.12	26.74	4.44	3.54	1.96	15.1	10.7
	Indiana	0.424	21.73	21.01	3.63	3.41	1.91	8.4	9.5
	Iowa	0.418	22.32	17.85	3.64	3.13	1.67	2.1	9.1
	Kansas	0.435	21.31	24.95	4.49	3.64	2.12	5.7	9.9
	Michigan	0.440	20.79	24.24	4.80	3.60	1.98	14.2	10.5
	Minnesota	0.426	21.73	22.76	3.73	3.30	1.93	3.5	7.9
	Missouri	0.449	20.37	27.16	4.07	3.54	1.98	11.2	11.7
	Nebraska	0.424	21.89	20.33	4.00	3.30	1.77	4.0	9.7
	North Dakota	0.429	21.53	20.77	4.45	3.35	1.77	0.6	11.9
	Ohio	0.441	20.81	25.03	4.53	3.72	2.11	11.5	10.6
	South Dakota	0.434	21.43	26.98	4.01	3.46	1.99	0.6	13.2
	Wisconsin	0.413	22.44	18.59	3.87	3.04	1.65	5.7	8.7
South	Alabama	0.475	18.86	33.22	5.48	4.04	2.48	26.0	16.1
	Arkansas	0.458	19.81	22.97	4.44	3.67	2.05	15.7	15.8
	Delaware	0.429	21.48	22.25	3.67	3.36	1.81	19.2	9.2
	DC	0.549	15.37	74.05	6.73	5.13	3.67	60.0	20.2
	Florida	0.470	19.61	29.49	4.31	3.74	2.21	14.6	12.5
	Georgia	0.461	19.56	28.49	4.60	3.57	1.93	28.7	13.0
	Kentucky	0.468	19.08	26.10	5.12	3.84	2.24	7.3	15.8
	Louisiana	0.483	18.13	39.72	5.66	4.27	2.54	32.5	19.6
	Maryland	0.434	21.02	32.92	4.50	3.86	2.45	27.9	8.5
	Mississippi	0.478	18.69	32.38	5.09	4.04	2.43	36.3	19.9
	North Carolina	0.452	20.37	27.64	4.42	3.73	2.22	21.6	12.3
	Oklahoma	0.455	19.97	27.34	4.68	3.64	2.09	7.6	14.7
	South Carolina	0.454	20.10	26.58	4.63	3.62	2.06	29.5	14.1
	Tennessee	0.465	19.67	30.92	4.79	4.05	2.65	16.4	13.5
	Texas	0.470	19.38	24.43	3.87	3.34	1.87	11.5	15.4
	Virginia	0.449	20.26	28.08	4.86	3.71	2.14	19.6	9.6
	West Virginia	0.468	19.27	28.05	4.98	3.90	2.21	3.2	17.9
West	Alaska	0.402	22.56	15.52	3.71	3.10	1.50	3.5	9.4
	Arizona	0.450	20.52	29.63	3.77	3.47	2.01	3.1	13.9
	California	0.475	18.82	23.02	3.87	3.25	1.77	6.7	14.2
	Colorado	0.438	21.14	21.20	3.49	3.19	1.72	3.8	9.3
	Hawaii	0.434	21.07	36.20	5.13	3.96	2.44	1.8	10.7
	Idaho	0.427	21.55	16.98	3.56	3.05	1.66	0.4	11.8
	Montana	0.436	21.03	22.65	4.24	3.45	1.79	0.3	14.6
	Nevada	0.436	21.65	17.72	3.15	2.89	1.57	6.8	10.5
	New Mexico	0.460	19.66	29.89	4.92	3.69	1.94	1.9	18.4
	Oregon	0.438	21.04	21.08	4.09	3.54	2.01	1.6	11.6
	Utah	0.410	22.93	16.63	3.15	2.98	1.57	0.8	9.4
	Washington	0.436	21.24	25.60	4.52	3.49	1.94	3.2	10.6
	Wyoming	0.428	21.44	18.40	3.88	3.16	1.61	0.8	11.4

[a] Source: U.S. Census Bureau, Table S4

[b] Sources: Imputed from Tables HCT-013 Aggregate Household Income and QT-P32 Income Distribution in 1999 of Households and Families: 2000, Census 2000 Summary File 3 (SF3)

[c] Source: Imputed from Current Population Survey Annual Social and Economic Supplement (CPS ASEC) for years 1995 to 2004.

[d] Source: Table DP-1, Profile of General Demographic Characteristics: 2000, Census 2000 Summary File 1 (SF1)

[e] Source: Table 3, State and Regional Poverty Rates: 1989 and 1999, Poverty: 1999 Census 2000 Brief, May 2003

Table 3.3. Summary statistics for the state-level variables used in the models, by sex[a]

	Males (n = 189,416)				Females (n = 204,792)			
State-level measure	**Mean**	**SD**	**Min**	**Max**	**Mean**	**SD**	**Min**	**Max**
Household Gini coefficient	0.456	0.021	0.402	0.549	0.456	0.021	0.402	0.549
Percentage of state income for households below the median	19.997	1.207	15.370	22.930	19.978	1.203	15.370	22.930
90:10 ratio	26.372	4.895	15.524	74.048	26.468	4.923	15.524	74.048
80:20 ratio	4.357	0.518	3.146	6.726	4.368	0.518	3.146	6.726
50:50 ratio	3.583	0.293	2.886	5.125	3.589	0.293	2.886	5.125
90:40 ratio (Palma)	2.069	0.273	1.497	3.668	2.075	0.273	1.497	3.668
Percentage in poverty	12.251	2.747	6.500	20.200	12.280	2.759	6.500	20.200
Percentage black	12.062	8.112	0.300	60.000	12.267	8.198	0.300	60.000

[a] Sources: See footnote to Table 3.2.

Table 3.4. Hazard ratios for death within 10-year follow-up period, by income inequality measure and sex[a]

	Males			Females		
State-level inequality measure	**HR**	**95% CI**	**p-value**	**HR**	**95% CI**	**p-value**
Household Gini coefficient	0.016	(0.0064 – 0.0422)	<0.001	0.235	(0.1246 – 0.4455)	0.011
Percentage of state income for households below the median	0.163	(0.1070 – 0.2549)	<0.001	0.201	(0.1056 – 0.3830)	<0.001
90:10 ratio	1.002	(0.9995 – 1.0040)	0.240	1.002	(1.0012 – 1.0078)	0.003
80:20 ratio	1.108	(1.0857 – 1.1307)	< 0.001	1.087	(1.0618 – 1.1125)	< 0.001
50:50 ratio	1.138	(1.1117 – 1.1667)	< 0.001	1.112	(1.0803 – 1.1439)	< 0.001
90:40 ratio (Palma)	1.059	(1.0341 – 1.0837)	< 0.001	1.008	(0.9655 – 1.0536)	0.208
None (poverty alone)	0.999[b]	(0.9941 – 1.0039)	0.681	0.995	(0.9915 – 0.9991)	0.058

[a] Multilevel Cox proportional hazards models controlling for compositional variables at the individual level (age, log equivalized family income, race, Hispanic origin, highest level of education, urbanization, marital status, employment status, valid social security number) and for percent black population and poverty rate at the state level.

[b] Model did not converge after 50 iterations.

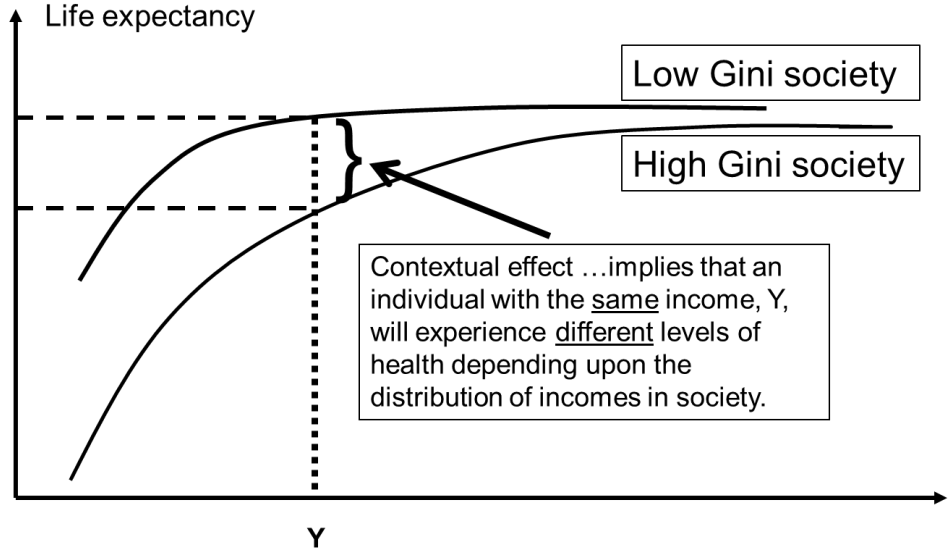
Table 3.5. Percent change in mortality for a one standard deviation increase in income inequality, by sex, at the point estimate for the beta coefficient, and for the 95% CI for beta[a]

	Multilevel Cox proportional hazard models			
	Males		Females	
State-level inequality measure	**% change**	**95% CI**	**% change**	**95% CI**
Household Gini coefficient	-8.34	(-10.09 – -6.45)	-2.99	(-4.28 – -1.68)
Percentage of state income for households below the median	-88.81	(-93.27 – -80.80)	-85.49	(-93.31 – -68.49)
90:10 ratio	0.98	(-0.24 – 1.97)	0.99	(0.59 – 3.90)
80:20 ratio	5.45	(4.35 – 6.56)	4.42	(3.16 – 5.68)
50:50 ratio	3.87	(3.16 – 4.63)	3.16	(2.29 – 4.02)
90:40 ratio (Palma)	1.58	(0.92 – 2.22)	0.22	(-0.96 – 1.44)
None (poverty alone)	-0.27	(-1.61 – 1.08)	-1.37	(-2.33 – -0.25)

[a] Note: All models include state poverty rate and state percent black.

FIGURES

Figure 3.1. The contextual effect of income inequality on life expectancy



Source: (Kawachi, 2011)

Figure 3.2. Scatterplot of NLMS respondent survival to five years versus state-level Gini coefficient (males)

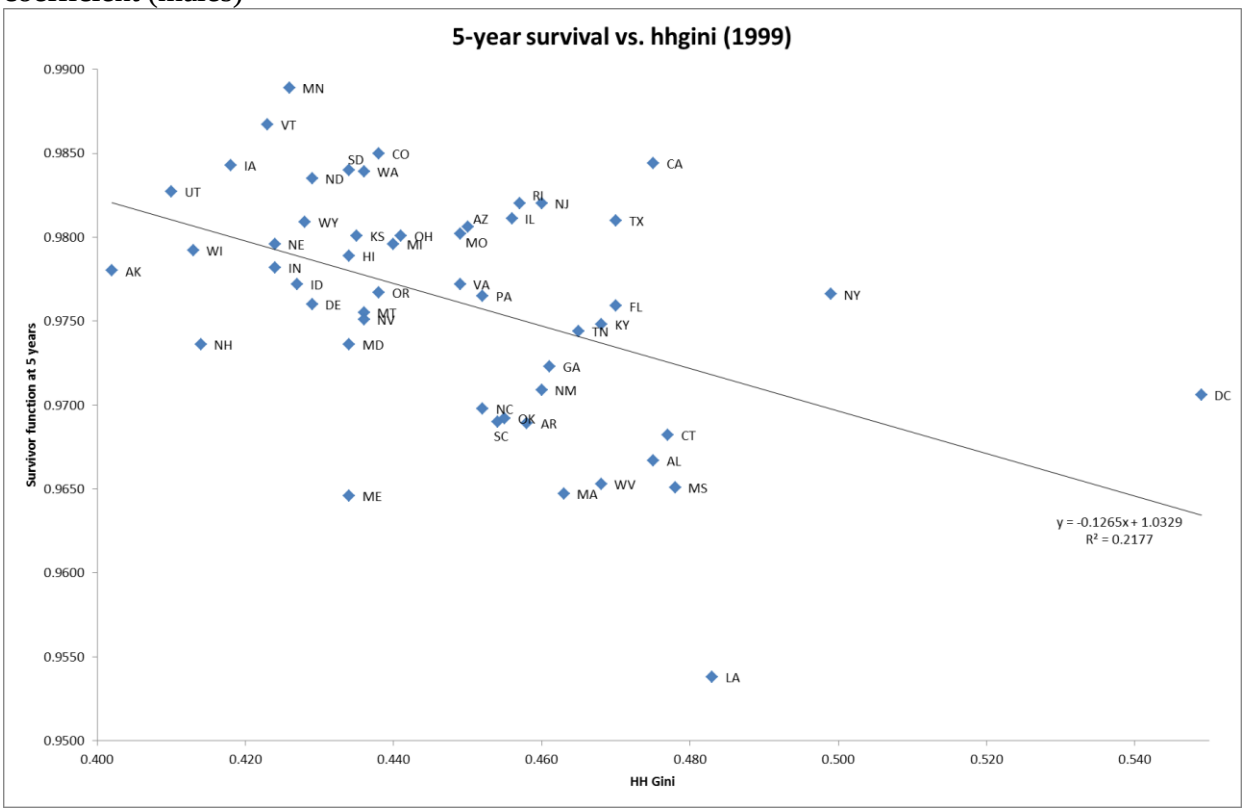
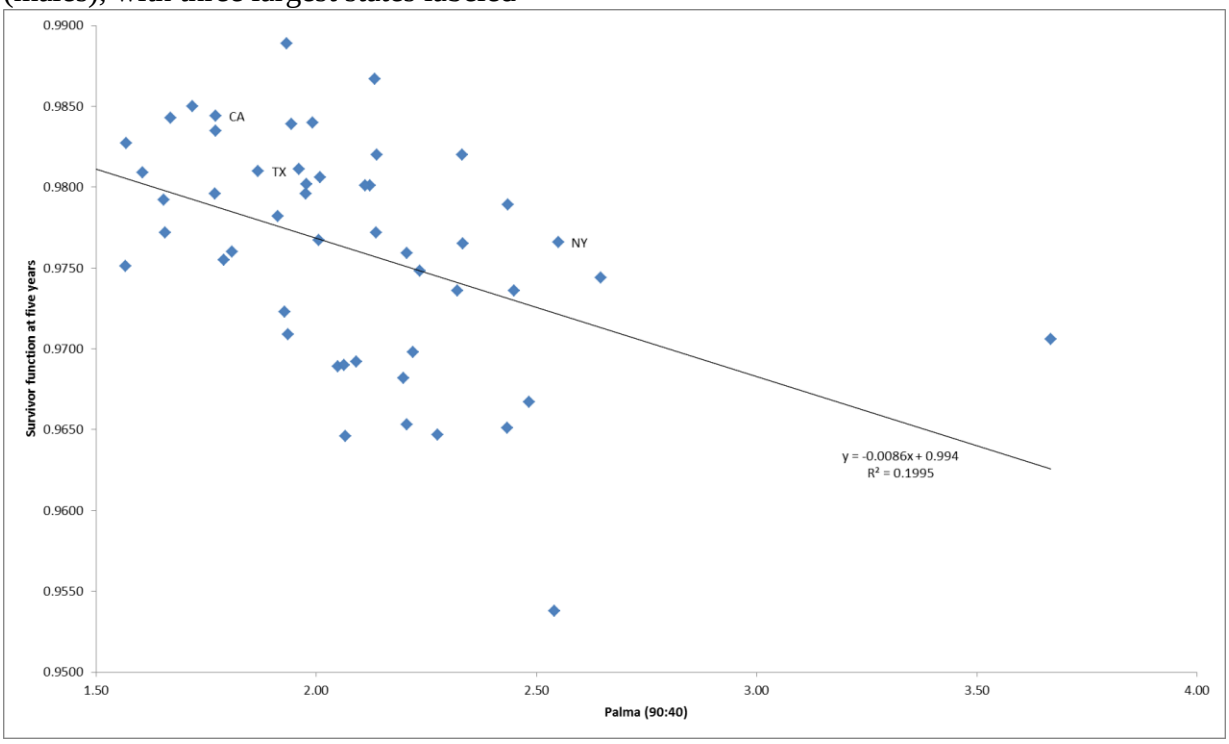
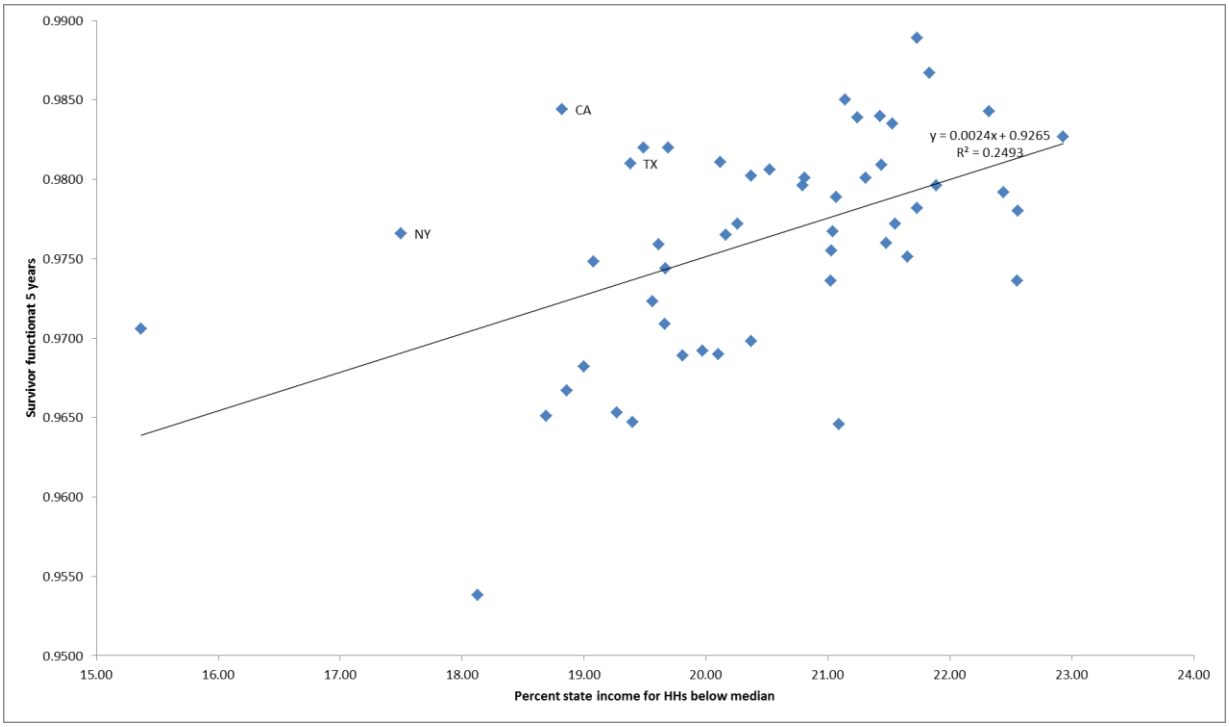


Figure 3.3. Scatterplots of NLMS respondent survival to five years versus (a) state-level Palma (90:40) ratio and (b) percentage of state income going to households below the median income (males), with three largest states labeled





APPENDIX

Table A1. Hazard ratios for death within 10-year follow-up period, by income inequality measure and sex[a]

	Males			Females		
State-level inequality measure	**HR**	**95% CI**	**p-value**	**HR**	**95% CI**	**p-value**
Household Gini coefficient	3.889	(1.7046 – 8.8733)	< 0.001	7.318	(0.3137 – 170.7361)	0.216
Percentage of state income for households below the median	0.301	(0.1504 – 0.6010)	0.001	0.958	(0.0081 – 113.1575)	0.986
90:10 ratio	1.004	(1.0015 – 1.0056)	0.001	1.000	(0.9854 – 1.0144)	0.977
80:20 ratio	1.051	(1.0232 – 1.0794)	< 0.001	0.877	(0.6548 – 1.1750)	0.380
50:50 ratio	1.072	(1.0309 – 1.1149)	0.001	0.982	(0.7246 – 1.3302)	0.905
90:40 ratio (Palma)	1.104	(1.0494 – 1.1604)	< 0.001	0.864	(0.4979 – 1.4996)	0.604
None (poverty alone)	1.013	(1.0097 – 1.0163)	< 0.001	1.000	(0.9918 – 1.0089)	0.941

[a] Cox proportional hazards models with state fixed effects and clustered standard errors, controlling for age, log equivalized family income, race, Hispanic origin, highest level of education, urbanization, marital status, employment status, valid social security number and for state-level percent black population and state-level poverty rate. (Note: four states were excluded from the models due to collinearity).

Table A2. Percent change in mortality for a one standard deviation increase in income inequality, by sex, at the point estimate for the beta coefficient, and for the 95% CI for beta[a]

	Cox proportional hazards models with state fixed effects			
	Males		Females	
State-level inequality measure	**% change**	**95% CI**	**% change**	**95% CI**
Household Gini coefficient	2.90	(1.13 – 4.70)	4.27	(-2.40 – 11.39)
Percentage of state income for households below the median	-76.53	(-89.84 – -45.92)	-5.03	(-99.70 – 29,482.36)
90:10 ratio	1.97	(0.74 – 2.77)	0.00	(-6.98 – 7.29)
80:20 ratio	2.61	(1.19 – 4.03)	-6.57	(-19.70 – 8.71)
50:50 ratio	2.06	(0.90 – 3.24)	-0.53	(-9.02 – 8.73)
90:40 ratio (Palma)	2.74	(1.32 – 4.14)	-3.92	(-17.36 – 11.71)
None (poverty alone)	3.61	(2.69 – 4.54)	0.00	(-2.25 – 2.48)

[a] Note: All models include state poverty rate and state percent black.

Table A3. Hazard ratios for death within 10-year follow-up period, by income inequality measure, males[a]

State-level inequality measure	HR	95% CI	p-value	Proportional Hazards Tests: Functional form for time	rho	Chi-square	p-value
Household Gini coefficient	0.0257	(0.0026 – 0.2524)	0.002	Linear	-0.1246	475.18	<0.001
				Log	-0.1088	362.53	<0.001
90:10 ratio	1.001	(0.9933 – 1.0091)	0.773	Linear	-0.0379	63.12	<0.001
				Log	-0.0279	34.19	<0.001

[a] Cox proportional hazards models with region fixed effects and standard errors clustered at the state level, controlling for age, log equivalized family income, race, Hispanic origin, highest level of education, urbanization, marital status, employment status, valid social security number and for state-level percent black population and state-level poverty rate.