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Long-term association of economic inequality and mortality in adult Costa Ricans

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ABSTRACT

Despite the large number of studies, mostly in developed economies, there is limited consensus on the health effects of inequality. Recently a related literature has examined the relationship between relative deprivation and health as a mechanism to explain the economic inequality and health relationship. This study evaluates the relationship between mortality and economic inequality, as measured by area-level Gini coefficients, as well as the relationship between mortality and relative deprivation, in the context of a middle-income country, Costa Rica. We followed a nationally representative prospective cohort of approximately 16,000 individuals aged 30 and over who were randomly selected from the 1984 census. These individuals were then linked to the Costa Rican National Death Registry until Dec. 31, 2007. Hazard models were used to estimate the relative risk of mortality for all-cause and cardiovascular disease mortality for two indicators: canton-level income inequality and relative deprivation based on asset ownership. Results indicate that there was an unexpectedly negative association between canton income inequality and mortality, but the relationship is not robust to the inclusion of canton fixed-effects. In contrast, we find a positive association between relative deprivation and mortality, which is robust to the inclusion of canton fixed-effects. Taken together, these results suggest that deprivation relative to those higher in a hierarchy is more detrimental to health than the overall dispersion of the hierarchy itself, within the Costa Rican context.

Introduction

Despite decades of research there is still much controversy regarding the notion that individuals are less healthy in places where income or wealth is more unequally distributed. A substantial amount of literature documents a positive association between poor health and the level of economic inequality across countries (Wilkinson, 1992), regions or states (Hildebrand & Kerm, 2009; Subramanian & Kawachi, 2003), and counties (Soobader & LeClere, 1999; Subramanian et al., 2003). While empirical literature testing the relationship between inequality and health and its pathways is extensive (Deaton, 2003; Kondo et al., 2009; Lynch, Smith, Harper, & Hillemeier, 2004; Wilkinson & Pickett, 2006), the interpretation of these associations remains contentious. Disagreement remains on the underlying pathways by which inequality could directly (or indirectly) affect health, as well as on the appropriate methods for testing this relationship.

Recently, a related literature has examined the relationship between relative deprivation and poor health. The concept of relative deprivation—which focuses on how an individual compares his circumstance to that of others who are more fortunate than he is—was mathematically formulated by Yitzhaki (1979). Using Yitzhaki's measure of relative deprivation, several studies demonstrate a positive association between higher relative deprivation and poor health, although these results are somewhat dependent on specification. In the United States, Eibner and Evans find that even after controlling for individual income and a number of covariates, relative deprivation appears to be strongly related to the probability of dying in a 5-year follow-up (Eibner & Evans, 2005). Likewise, Subramanyam et al. find that relative deprivation in income is positively associated with poor self-rated health (Subramanyam, Kawachi, Berkman, & Subramanian, 2009). In South Africa, a parallel study finds that relative deprivation, particularly with age as a reference group, is related to mortality after controlling for individual socioeconomic status (SES) (Salti, 2010). In the United Kingdom, Jones and Wildman find mixed support for the association between relative deprivation and self-reported health, depending on the parametric assumptions (Jones & Wildman, 2008). In contrast, Li and Zhu find no relationship between relative deprivation and poor self-reported health in China (Li & Zhu, 2006). Finally, in Sweden, Aberg Yngwe et al. use

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a different measure for relative deprivation, but still find a relationship with poor self-reported health (Aberg Yngwe, Fritzell, Lundberg, Diderichsen, & Burstrom, 2003).

These two literatures are closely linked: if within-group inequality rises, those at the bottom end of the distribution become relatively more deprived. However, relative deprivation varies at the individual-level, whereas inequality measures vary only at the group-level. This difference is important because an individual's relative deprivation is insensitive to the distributional properties for anyone with less income, but is highly sensitive to one's rank relative to those higher within a distribution. In contrast, inequality measures summarize distribution-wide characteristics and exhibit certain properties such as sensitivity to transfers from the poor to the rich (see chapter 3 of Deaton, 1997, for detailed discussion of desirable properties of inequality measures). These differences are more pronounced when the underlying distribution is skewed. Many studies with inequality measures try to bridge these differences by stratifying the analysis based on an individual's rank in the distribution to capture the different effects of hierarchy depending on an individual's position within the hierarchy. It will be important for future studies to make a direct comparison between the different approaches.

Acknowledging the differences in each measure, this study aims to advance empirical understanding of the income inequality/relative deprivation hypothesis by analyzing the effects of income inequality and relative deprivation on mortality in a unique, previously unexplored institutional context: Costa Rica. For decades observers have noted that Costa Rica has a remarkably high life expectancy (higher than the United States) despite its limited resources. While many hypotheses have been suggested to help explain this outlier, one untested notion is that Costa Rica is an unusually equitable society in certain dimensions, and that this has contributed to its good health (Daniels, Kennedy, & Kawachi, 2000).

In addition, Costa Rica provides a setting where the possibility of confounding in the relation of health and inequality is limited because of several characteristics. First, Costa Rica has a highly homogeneous population. Using the sum of the ethnic, linguistic, and religious fractionalization measures created by Alesina, Devleeschauwer, Easterly, Kurlat, and Wacziarg (2003), Costa Rica ranks in the bottom decile in the world in terms of combined ethnic, linguistic, and religious heterogeneity. Comparatively, South Africa ranks in the 90th percentile, the U.S. at about the 70th percentile, and China in the 30th percentile. Second, Costa Rica has a highly centralized political system which allocates health services, education, and most local resources, (Mesa-Lago, 2000) decreasing the possibility of locally endogenous underinvestment in unequal areas. Third, Costa Rica also has a nationally run universal health insurance regime with an independent budget, making political cycles less significant in the delivery of health services. Taken together these unique characteristics make Costa Rica an attractive case to consider.

This study contributes to the income inequality and health literature in several ways. First, it examines the relation between health and inequality using both the income inequality and relative deprivation constructs in a developing country. Second, it employs a high-quality mortality follow-up study that is unique in the developing world in its size and length. Third, by analyzing this relationship in a country with a longstanding social safety net and by accounting for area-level confounders, it aims to minimize the potential for bias in the association between income inequality and mortality. Finally, it examines sensitivity of the results to a range of methodological issues, including controls for individual and area-level covariates, controls for area-level fixed-effects, and alternative inequality measures.

Data

This analysis incorporates a new data source: the Costa Rican Longitudinal Mortality Study (CR-LMS). This dataset links a sample of records from the 1984 Costa Rican census to death records in Costa Rica's civil registration system through December 31, 2007. Rosero-Bixby and Antich detail the methods used to create this dataset (Rosero-Bixby & Antich, 2010). We provide a brief description below and full details of the matching methods in the online data appendix.

The CR-LMS dataset was collected in three phases under the supervision of the Centro Centroamericano de Población. First, a random stratified sample of approximately 20,000 adults, aged 30+, was selected from the 1984 census. Second, the original census questionnaires were consulted in order to retrieve the names of selected individuals. Names were then linked with the Civil Registry to obtain each individual's "*cedula*" (a unique national identification number similar to a Social Security number). The *cedula* was used to conduct a computer follow-up in the Death Registry through December 2007. Finally, a probabilistic linkage was conducted with the vital statistics data file to retrieve information about cause of death.

This method matched 16,315 of the census names to *cedulas* with a high degree of certainty. Of these, 15,276 had non-missing covariates and were alive on January 1, 1989. In this sample, 3746 deaths occurred between January 1, 1989 and December 31, 2007. Of these, 3445 could be linked to the Vital Statistics Records, which further indicate cause of death.

Dependent variables

The outcome of interest is time to death. The origin is birth date, and data are both left and right censored. We started observation on January 1, 1989 (left censoring) and closed it on December 31, 2007, or the date the individual was considered lost to follow-up (right censoring). The longest period of observation was 19 years and the median was 16.86 years. Note that the follow-up period began on January 1, 1989, even though the follow-up in the initial data began on June 28, 1984 (the census date). This was done in order to use the highest-quality early inequality data available and to exclude deaths of individuals already sick or near death at census time.

For theoretical and empirical reasons, we also considered a secondary outcome: cardiovascular disease (CVD) mortality. Prior literature suggests that certain conditions, such as CVD, may respond more quickly to changes in economic conditions that alter both stress and behavior (Adler, Boyce, Chesney, Cohen, & Folkman, 1994). In addition, there were sufficient numbers of CVD deaths (927 deaths) to identify associations in these data, whereas there were not for other causes related to behavior or previously examined, such as chronic respiratory disease, diabetes, suicide, or cirrhosis/liver disease (Modrek & Ahern, 2011). We classified deaths using ICD 9 (period 1980–1996) and ICD 10 (1997–today) codes, and we treated the revisions per guidelines in Anderson, Minino, Hoyert, and Rosenberg (2001), Table C.

Individual-level covariates

Throughout all the models, demographic characteristics—including gender, age at baseline, age at baseline squared, age at baseline cubed, education, health insurance status in 1984, living in the San José metropolitan area, living in an urban area, and whether the individual was married or in a consensual union in 1984—were included as controls. All were measured from the 1984 census. Other controls, detailed below, include household wealth and area of residence.

Wealth

Since there was no income measure in the 1984 census, a wealth measure is included to control for household-level resources. The wealth measure is a count of the number of assets (telephone, hot-water heater, refrigerator, radio, television, and car) and amenities in the household (access to electricity, piped-in water, sewage, and non-dirt floors) in 1984. For each category, ownership increases the wealth measure by 1 and all categories are given equal weight; thus the wealth measure ranges from 0 to 10.

We used this wealth measure in several additional ways. For all models we included this measure as a continuous variable to account for access to resources. We also created a low wealth metric, which is an indicator variable set to 1 if household wealth is in the lowest tertile of wealth distribution. (This measure is used in models where we explore whether income inequality has a differential effect on the poor.) Finally, we used this wealth measure to construct the relative deprivation measures detailed below.

Inequality measures

Two measures of socioeconomic inequality were assessed in this study: (1) area-level income inequality as measured by the canton-level Gini coefficient based on household income, and (2) individual-level wealth relative deprivation based on the distribution of household assets within a canton.

We used 81 cantons, the second hierarchical administrative division, as our geographic unit of analysis in both measures. The average canton population was close to 30,000 (range 3100 to 241,000; median 19,700) in the 1984 census.

Income inequality

We estimated the canton-level Gini coefficients for several periods with data from the Costa Rican Household Surveys for Multiple Purposes (CRHSMP), an annual countrywide cross-sectional household survey of approximately 1% of the population. National Statistics and Census Institute provided the anonymized databases of these surveys. From this survey, total household income was scaled by the square root of household size to account for household size and economies of scales within households (Foster, 2009). Using this metric, we constructed an inequality variable within each canton. For certain years, the geocodes for the cantons were not available, so inequality measures are constructed only for years in which these codes could be ascertained from the data. These measures are made by pooling two or three adjacent years of CRHSMP to ensure a sufficient number of households within each canton. The median number of observations per canton is approximately 200. Because CRHSMP is a 1% sample of the population in each year, the inequality measure in a few small cantons is constructed from a smaller sample with fewer than 60 observations, but the inequality measures from these small cantons account for less than 3% of persons analyzed in the CR-LMS mortality data. Overall, these measures correlate well from year to year and cluster spatially; further simulation evidence indicates that any systematic measurement error attenuation bias is small and does not affect our main conclusions (Modrek, 2009).

Relative deprivation

Household relative deprivation was estimated for each individual in our sample using the wealth information from the 1984 census microdata, which includes all households within a canton. Following Eibner and Evans (2005) and originally defined by Yitzhaki (1979), relative deprivation was defined as the sum of the differences in the wealth index between person i and all others j who have more wealth than person i in their canton. This is calculated as:

$$RD_i = (1/N)*\Sigma\left(y_j - y_i\right) \forall y_j > y_i \text{ OR equivalently } RD_i$$
$$= \Pr(y > y_i)*[E(y|y > y_i) - y_i]$$

This measure captures the expected difference in household wealth between person i and all other individuals j with greater wealth in his canton. For example, if person i has a wealth score of 9 and 5% of the population have a higher wealth score of 10 in her canton, then her RD is equal to $RD_i = \Pr(y > y_i)*[E(y|y > y_i) - y_i] = 0.05*(1) = 0.05$. We can create this variable only for 1984, so it is fixed over time.

Fig. 1 shows the box plot of the relative deprivation score at each level of wealth. Looking at the mean relative deprivation value at each wealth level also shows the strong correlation between relative deprivation and wealth (the Pearson correlation coefficient is −0.85, $p < 0.0001$). The figure also shows that within each wealth group there is substantial variation in the relative deprivation measure, especially in the lower wealth groups. However, there is no overlap in the distribution of relative deprivation among those with wealth scores 0 and 1 and those with wealth scores of 8, 9, and 10.

In addition, the variation in relative deprivation score relative to the wealth index is highest at lower levels of deprivation, and the distribution of wealth is actually right skewed with most of the mass higher than a wealth of 6.

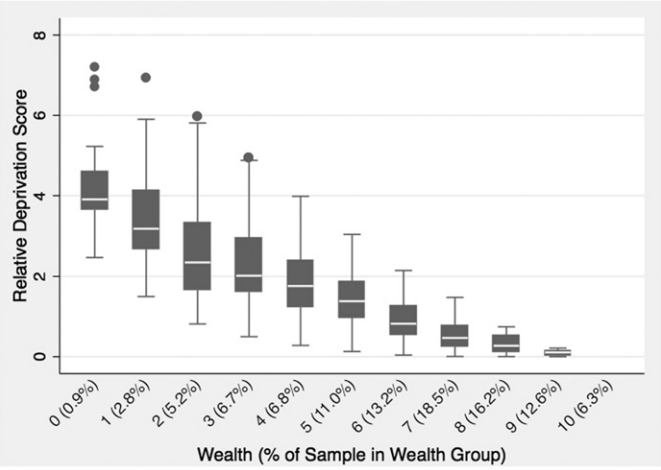
Canton-level controls

Using the same data structure used to construct the canton inequality measure, we also estimated several canton-level area controls based on CRHSMP. Canton-level variables include unemployment rate, percent in-migration into the canton, and mean household equivalent income (Modrek, 2009).

The summary statistics for the individual-level and area-level measures from the from the 1984 census and from the household surveys are included in the online appendix [APPENDIX Table 1 and Table 2].

Statistical methods

This study employs proportional hazard regression models parameterized as a Gompertz hazard distribution (Klein &



Note: Author's calculations based on census mortality data. Relative deprivation score based on age, sex, and canton of residence.

Fig. 1. Relative deprivation box plot by wealth group.

Moeschberger, 2003). The Gompertz distribution has been widely used to describe adult human mortality, and these models are known to fit these data well (Rosero-Bixby & Antich, 2010, p. 22). The key assumption in the Gompertz model is that the underlying rate of mortality increases exponentially with age t at an annual rate γ. Each individual in the sample was split into observational segments of single years of age. The variable age refers to the age in each of these segments.

The Gompertz hazard equation is $h(t) = \exp(\gamma t)\exp(\boldsymbol{\beta X})$ where γ is fixed for all individuals and represents the aging or senescence process in this population (i.e., the increase of mortality with age), $\mathbf{X}$ is a vector of covariates, and $\boldsymbol{\beta}$ is a vector of regression coefficients we estimated using maximum likelihood procedures as implemented in Stata (StataCorp, 11). We modeled inequality with the following three sets of models:

Set 1 of models: constant baseline income inequality

$$\boldsymbol{\beta X} = \beta_0 + \beta_1(\mathrm{I}_{c,1989}) + \boldsymbol{\beta_{2Z}}(\mathbf{Z}_i) + \boldsymbol{\varepsilon}$$

$$\boldsymbol{\beta X} = \beta_0 + \beta_1(\mathrm{I}_{c,1989}) + \boldsymbol{\beta_{2Z}}(\mathbf{Z}_i) + \beta_3(\mathbf{A}_c) + \boldsymbol{\varepsilon}$$

$$\boldsymbol{\beta X} = \beta_0 + \beta_1(\mathrm{I}_{c,1989}) + \boldsymbol{\beta_{2Z}}(\mathbf{Z}_i) + \beta_3(\mathbf{A}_c) + \boldsymbol{\beta_4}\mathrm{I}_c{*}\mathrm{LW}_i + \boldsymbol{\varepsilon}$$

I_c is the income inequality in canton c in 1989, A_c is a vector of the canton-level controls in 1989, $\mathbf{Z}_i$ is a vector of individual controls measured in 1984 (gender, age at baseline, age at baseline squared, age at baseline cubed, insurance status, marital status, wealth, education, and place of residence), and $\mathrm{I}_c{*}\mathrm{LW}_\mathbf{i}$ is an indicator variable for having low wealth interacted with the canton Gini coefficient. The estimate for β_1 is meant to capture the association between income inequality and survival risk. The interaction is included to examine if income inequality has a different impact on the poor; this is the product of an indicator variable for being in the lowest tertile of wealth (having a wealth less than or equal to 5) and the continuous Gini measure.

Set 2 of models: time-varying income inequality with canton fixed-effects

$$\boldsymbol{\beta X} = \beta_0 + \beta_1(\mathrm{I}_{ct}) + \boldsymbol{\beta_{2Z}}(\mathbf{Z}_i) + \mathrm{y}_c + \boldsymbol{\varepsilon}$$

$$\boldsymbol{\beta X} = \beta_0 + \beta_1(\mathrm{I}_{ct}) + \boldsymbol{\beta_{2Z}}(\mathbf{Z}_i) + \beta_3(\mathbf{A}_{ct}) + \mathrm{y}_c + \boldsymbol{\varepsilon}$$

$$\boldsymbol{\beta X} = \beta_0 + \beta_1(\mathrm{I}_{ct}) + \boldsymbol{\beta_{2Z}}(\mathbf{Z}_i) + \beta_3(\mathbf{A}_{ct}) + \beta_4\mathrm{I}_{ct}{*}\mathrm{LW}_i + \mathrm{y}_c + \boldsymbol{\varepsilon}$$

These models examine the same relationship as above, but now allow all the area-level variables, including the Gini, to vary with time. We divide each observation at the time when voting registrations were closed for each election (November 1993, 1997, and 2001); we map area-level variables to the beginning of the period based on the closest year of available data. The addition of time-varying canton-level variables allows us to include canton-level fixed-effect dummy variables y_c. These fixed-effects account for time-invariant canton characteristics that are correlated with both income inequality and mortality risk. If income inequality was systematically related to a canton characteristic such as the allocation of public goods, then we might be incorrectly attributing the effects of the canton characteristic to income inequality. However, these fixed-effects estimates are purged of any time-invariant area-level characteristics. Accordingly, β_1 captures the association of *changes* in income inequality and survival risk.

Set 3 of models: constant baseline relative deprivation

$$\boldsymbol{\beta X} = \beta_0 + \boldsymbol{\beta_{3Z}}(\mathbf{Z}_i) + \beta_6\mathrm{RD}_i + \boldsymbol{\varepsilon}$$

$$\boldsymbol{\beta X} = \beta_0 + \boldsymbol{\beta_{3Z}}(\mathbf{Z}_i) + \beta_6\mathrm{RD}_i + \mathrm{y}_c + \boldsymbol{\varepsilon}$$

These models use the relative deprivation measure described above, which reflects wealth disparity between each individual and those with greater wealth within a canton. Though not directly comparable to the models above, they serve as complementary analyses. Since relative deprivation varies at the individual-level, we can also include canton-level fixed-effects to account for time-invariant canton characteristics that are correlated with relative deprivation and mortalitiy risk in this set of models. Here, $\mathbf{Z}_i$ is again a vector of individual controls measured in 1984, RD_i is the individual's relative deprivation as compared to those living within her canton in 1984, and y_c is a vector of area-level fixed-effects.

Given that the sample followed a clustered design, all estimates of the standard errors from all the models were corrected for clustering at the canton-level by using the "cluster" option in the Stata 11 software package.

Results

Income inequality hypothesis: constant baseline income inequality

Table 1 presents the results for all-cause mortality using the hazard models outlined in the first set of models above. Results presented in column 1 only include individual-level covariates for comparison. Results presented in column 2 include the income inequality Gini measure and the mean household income in the canton. Results presented in column 3 add the other area-level control variables. Finally, results presented in column 4 include an interaction term between income inequality and having low wealth.

Results from these analyses do not support the notion that income inequality is detrimental to health. Rather, the results for all-cause mortality suggest that those residing in areas with greater income inequality actually have *longer* survival. The unscaled coefficients in Table 1 are quite large because they represent a one-unit change in inequality, effectively going from perfect equality to perfect inequality; the scaled coefficients imply that a 0.05-unit increase (a one-standard deviation increase) in inequality is associated with a 5% lower hazard (HR = 0.95, CI 0.91–0.99). The magnitude on the interaction of income inequality and having low wealth in column 4 was not significant. Moreover, higher area-level mean income is associated with lower survival time.

In contrast to the area-level measures, the results for individual-level characteristics are as expected for all-cause mortality. Women have a 30% lower hazard of mortality. Likewise, those who are partnered in 1984 have a survival advantage (HR = 0.82, CI 0.75–0.89), as do the wealthy. Each additional household amenity owned (our measure of wealth) confers a 2–3% lower mortality hazard (HR = 0.98, CI 0.95–1.00). Those who complete college have a 26% lower mortality hazard (HR = 0.74, CI 0.64–0.86). Those who reside in a rural area also have 10% lower hazard of mortality (HR = 0.90, CI 0.81–0.99). Finally, those without insurance in 1984 also live longer, providing some evidence that there was adverse selection into the public insurance program at that time.

Table 2 presents the results for CVD mortality following the same pattern as in Table 1. Results presented in columns 2–4 exhibit the same puzzling association with regard to income inequality. The estimated coefficient suggests that residents in areas with higher income inequality have better survival. A 0.05-unit increase in income inequality is associated with 9% better survival (HR = 0.91, CI

Table 1
Results from hazard models of the association between area-level income inequality in 1989 and mortality 1989–2007.

	Regression coefficients [standard errors]			
	All-cause mortality			
Canton-level covariates				
Gini of monthly household income		−0.843** [0.428]	−0.989** [0.439]	−0.895** [0.449]
Mean of the monthly household income		0.00728 [0.0052]	0.0115* [0.0062]	0.0114* [0.0062]
Interaction of wealth & area-level Gini				
Gini × low wealth in 1989				−0.165 [0.171]
Percent unemployed in 1989			0.0928 [0.753]	0.0722 [0.760]
Percent in-migration in 1989			−1.33* [0.719]	−1.35* [0.716]
Individual-level covariates				
Female	−0.354*** [0.040]	−0.354*** [0.040]	−0.355*** [0.039]	−0.353*** [0.039]
Education categories (none/some primary omitted)				
Completed primary	−0.0595 [0.0481]	−0.0559 [0.0490]	−0.0567 [0.0488]	−0.0567 [0.0487]
Some secondary	−0.107** [0.050]	−0.105** [0.052]	−0.106** [0.052]	−0.103** [0.052]
Some college	−0.298*** [0.073]	−0.297*** [0.077]	−0.300*** [0.077]	−0.294*** [0.078]
Live in San Jose Metro area	0.106** [0.0433]	0.0478 [0.0579]	0.0473 [0.0554]	0.0473 [0.0551]
Live in rural area	−0.116** [0.048]	−0.114** [0.050]	−0.114** [0.049]	−0.112** [0.049]
Wealth	−0.0212* [0.0120]	−0.0229* [0.0119]	−0.0228* [0.0119]	−0.0330** [0.0149]
Uninsured	−0.0869*** [0.0304]	−0.0864*** [0.0306]	−0.0858*** [0.0304]	−0.0852*** [0.0303]
Union	−0.199*** [0.046]	−0.201*** [0.046]	−0.200*** [0.046]	−0.201*** [0.046]
Constant	−6.74*** [0.83]	−6.53*** [0.83]	−6.49*** [0.83]	−6.41*** [0.82]
	Hazard ratios (95% confidence intervals)			
HR for one std deviation increase in Gini		0.96	0.95	0.96
95% CI		(0.92–1.00)	(0.91–0.99)	(0.92–1.00)
Canton fixed-effects	No	No	No	No
Log-likelihood	−694	−692	−691	−690
Observations	15276	15276	15276	15276

* Significant at 10%; ** significant at 5%; *** significant at 1%. Robust clustered (at canton-level) standard errors in bracket. Models include controls for age at baseline, age at baseline squared and age at baseline cubed. Canton-level covariates are from 1989/1990/1991 Costa Rican Household Surveys for Multiple Purposes and individual-level covariates are from Costa Rican Longitudinal Mortality Study.

Table 2
Results from hazard models of the association mortality.

	Regression coefficients [standard errors]			
	Cardiovascular disease			
Canton-level covariates				
Gini of monthly household income		−1.98*** [0.73]	−1.92** [0.77]	−1.73** [0.73]
Mean of the monthly household income		0.0287*** [0.0082]	0.0259** [0.0105]	0.0259** [0.0104]
Interaction of wealth and area-level Gini				
Gini × Low Wealth in 1989				−0.353 [0.324]
Percent unemployed in 1989			−1.35 [1.55]	−1.36 [1.54]
Percent in-migration in 1989			0.555 [1.61]	0.491 [1.61]
Individual-level covariates				
Female	−0.359*** [0.075]	−0.364*** [0.076]	−0.365*** [0.076]	−0.362*** [0.076]
Education categories (none/some primary omitted)				
Completed primary	−0.108 [0.094]	−0.108 [0.095]	−0.106 [0.095]	−0.106 [0.095]
Some secondary	−0.224** [0.111]	−0.248** [0.107]	−0.250** [0.108]	−0.241** [0.110]
Some college	−0.539*** [0.146]	−0.581*** [0.128]	−0.583*** [0.129]	−0.567*** [0.128]
Live in San Jose Metro area	0.122 [0.100]	−0.0745 [0.116]	−0.0700 [0.120]	−0.0704 [0.112]
Live in rural area	−0.0700 [0.0845]	−0.0566 [0.0851]	−0.0590 [0.0869]	−0.0545 [0.0872]
Wealth	0.0179 [0.0182]	0.01 [0.0178]	0.0114 [0.0178]	−0.0104 [0.0241]
Uninsured	−0.249*** [0.0806]	−0.248*** [0.0803]	−0.250*** [0.0806]	−0.248*** [0.0807]
Union	−0.109 [0.084]	−0.111 [0.085]	−0.11 [0.085]	−0.113 [0.085]
Constant	−11.4*** [1.6]	−11.0*** [1.6]	−11.0*** [1.6]	−10.8*** [1.6]
	Hazard ratios (95% confidence intervals)			
HR for one std deviation increase in Gini		0.91	0.91	0.92
95% CI		(0.84–0.97)	(0.84–0.98)	0.92(0.85–0.99)
Canton fixed-effects	No	No	No	No
Log-likelihood	−1291	−1285	−1285	−1284
Observations	14975	14975	14975	14975

* Significant at 10%; ** significant at 5%; *** significant at 1%. Robust clustered (at canton-level) standard errors in bracket. Models include controls for age at baseline, age at baseline squared and age at baseline cubed. Canton-level covariates are from 1989/1990/1991 Costa Rican Household Surveys for Multiple Purposes and individual-level covariates are from Costa Rican Longitudinal Mortality Study.

0.84–0.98). The coefficient of the interaction of income inequality and having low wealth was not significant. Finally, area-level mean income is associated with lower survival time.

Women had a lower hazard of cardiovascular mortality, as did those with college education. The results also show that those with no insurance in 1984 have reduced CVD mortality. Moreover, there was no SES gradient in terms of wealth for CVD.

Income inequality hypotheses: time-varying regressions

In Table 3, we examine how *changes* in inequality relate to mortality risk. Table 3 presents the results for all-cause mortality using the models with time-varying area-level covariates and canton-level fixed-effects, as described in the second set of models above. The main difference is that the area-level variables (including inequality Gini) are allowed to change with time and area-level fixed-effects are included. Columns 1–3 present the relationship between changes in income inequality and survival time for all-causes of death, whereas columns 4–6 present the results for CVD mortality. Columns 1 and 4 present the basic model without including the canton-level characteristics controls. Columns 2 and 5 include the area-level control variables. Finally, columns 3 and 6 include an interaction term between income inequality and having low wealth.

We focus on the results for the area-level measures, as the parameter estimates on the individual-level variables do not change substantially. For all-cause mortality, increases over time in the canton Gini coefficients were not significantly related to survival. However, changes in area-level unemployment rates were. A one-standard deviation increase in unemployment, 0.05, was related to an 11% decline in the hazard of mortality (HR = 0.89, CI 0.85–0.93).

For heart disease mortality, the results again suggest that increases in the Gini coefficient are not significantly related to survival. Moreover, with inclusion of fixed-effects, area-level income no longer relates to survival time, either. No other area-level variable was significantly related to cardiovascular mortality risk.

Relative deprivation hypothesis

We next consider an alternate measure of economic inequality: relative deprivation. Table 4 presents the results from the third set of models, examining the relationship between relative deprivation and mortality as described above. Columns 1–2 present the results for all-cause mortality, and columns 3–4 present the results for CVD mortality. For comparisons, columns 1 and 3 do not include canton fixed-effects, while columns 2 and 4 do.

For all-cause mortality, each additional unit of wealth deprivation is associated with 10% higher hazard of death (HR = 1.10, CI 1.00–1.20), even when we include canton fixed-effects. Also noteworthy, the relative deprivation measures eliminate the significance of the protective effects of having high wealth. This suggests that relative deprivation may capture some of the effects of wealth, as those that are wealthier are by definition less deprived, and the correlation between the relative deprivation measure and wealth is quite high (correlation coefficient = −0.82, p-val < .0001). From a theoretical perspective, we are interested in both parameter estimates, so we should include both in the model. But, if we choose to drop the wealth variable, the estimated parameter on relative deprivation is reduced (HR = 1.063, CI 1.04–1.08), though still significant (not shown, but available). The other coefficients for the remaining individual-level covariates remain largely unchanged in these models.

For CVD mortality, each additional unit of deprivation is associated with a 16% increase in the risk of death from CVD (HR = 1.16, CI 1.04–1.29), but this coefficient is not significant with the

Table 3
Results from hazard models of the association between time-varying area-level income inequality and mortality risk.

	Regression coefficients [standard errors]					
	All-cause mortality			Cardiovascular disease		
Individual-level wealth from 1984 census						
Wealth	−0.0286* [0.0126]	−0.0285* [0.0126]	−0.03 [0.0183]	−0.00132 [0.0201]	−0.00126 [0.0201]	−0.0128 [0.0277]
Canton-level covariates from July household surveys- TIME-VARYING						
Mean of the monthly household equivalent salary in 2006/(10,000)	0.00437 [0.00585]	0 [0.00571]	0 [0.00572]	−0.00726 [0.00850]	−0.00632 [0.01080]	−0.00642 [0.01080]
Percent unemployed		−2.35** [0.87]	−2.35** [0.87]		−1.06 [1.76]	−1.02 [1.76]
Percent in-migration		1.16 [0.75]	1.16 [0.75]		−0.198 [1.49]	−0.197 [1.49]
Inequality measure						
Gini of monthly household equivalent salary	−0.402 [0.558]	−0.247 [0.516]	−0.205 [0.540]	0.2 [0.978]	0.162 [1.02]	0.240 [1.08]
Cross-level interaction of wealth & area-level Gini						
Gini × Low Wealth in 1984			−0.0853 [0.182]			−0.178 [0.351]
	Hazard ratios (95% confidence intervals)					
HR for one std deviation increase in Gini	0.98	0.98	0.99	1.01	1.01	1.01
95% CI	(0.93–1.04)	(0.94–1.04)	(0.94–1.04)	(0.92–1.11)	(0.91–1.11)	(0.91–1.13)
Canton fixed-effects	Yes	Yes	Yes	Yes	Yes	Yes
Log-likelihood	−608	−604	−604	−1271	−1271	−1271
Observations	56405	56405	56405	56104	56104	56104

* Significant at 5%; ** significant at 1%. Robust clustered standard errors in bracket. Models include controls for age at baseline, age at baseline squared, age at baseline cubed, gender, education, marital status, and insurance status in 1984.

Table 4
Results from hazard models of the association between individual-level relative deprivation in 1984 and mortality risk.

	Regression coefficients [standard errors]			
	All-cause mortality		Cardiovascular disease	
Individual-level wealth from 1984 census				
Wealth	0.0249 [0.0199]	0.0252 [0.0299]	0.0881* [0.0349]	0.0912 [0.0626]
Inequality measure				
Relative deprivation	0.0937** [0.0300]	0.0950* [0.0462]	0.145** [0.056]	0.161 [0.101]
	Hazard ratios (95% confidence intervals)			
HR for 1 unit change in RD	1.10	1.10	1.16	1.18
95% CI	(1.03–1.16)	(1.00–1.20)	(1.04–1.29)	(0.97–1.43)
Canton fixed-effects	No	Yes	No	Yes
Log-likelihood	−690	−643	−1288	−1250
Observations	15276	15276	14975	14975

* Significant at 5%; ** significant at 1%. Robust clustered standard errors in bracket. Models include controls for age at baseline, age at baseline squared, age at baseline cubed, gender, education, marital status, insurance status in 1984, living in San Jose Metro area in 1984, and living in a rural area in 1984. The FE regressions do not include the living in San Jose Metro area in 1984, and living in a rural area in 1984 indicator.

inclusion of canton fixed-effects because the standard errors nearly double. When we exclude the wealth variable and examine the parameter estimate on the relative deprivation variable, the magnitude of the estimated coefficient becomes essentially zero, and the standard errors increase (HR = 1.021, CI = 0.989–1.05); other coefficients remain largely unchanged in this exercise (not shown, but available).

Sensitivity analyses

In order to ensure these results were not too reliant on any single assumptions, several additional analyses were done. We explore whether our results are sensitive to: (1) the underlying data used to construct the income inequality metrics, (2) the reference group used to create the relative deprivation measure, (3) our choice of survival model and different model specifications, and (4) different ways of modeling the standard errors.

First, to further explore whether the conflicting results between income inequality and wealth-based relative deprivation measures are due to the different underlying data used to construct these measures, we consider an intermediary measure. The income inequality measures were based on monthly household income, whereas the relative deprivation measures were based on the wealth metric based on the census. To account for these differences, we construct an intermediary measure: a wealth-based inequality measure. Using the 100% microsample of the 1984 census and the wealth measure described above, a wealth Gini coefficient was constructed for each canton using all households (except group homes). Since the wealth measures only range from 0–10, these measures are top-coded; hence the estimated wealth Ginis are smaller on average than the estimated income Ginis (see appendix). Moreover, the wealth Gini measures are constructed from all households in the census within a canton and thus have less measurement error than the income Ginis described above.

Results using this intermediate construct echo those found with the income Gini and are presented in Table 5. Using these measures, we find a negative association between the wealth Gini and both all-cause and CVD mortality hazard (all-cause HR = 0.90, CI 0.84–0.96; CVD HR = 0.89, CI = 0.78–1.03). While the magnitudes are not directly comparable, a one-standard deviation change in wealth Gini provides a similar effect size as a one-standard deviation change in the income Gini, and, if anything, the results from the wealth Gini are slightly more negative for all-cause mortality.

Second, we examined multiple versions of the relative deprivation measure and different specifications in the relative deprivation models. In line with Eibner and Evans (2005) and Salti (2010), we created different measures to account for potentially different relationships based on reference group. Since we lack detailed information on individuals' networks, we used observable demographic characteristics to construct these measures. One relative deprivation measure was based on just canton, one on sex and canton, and another on age, sex, and canton. The first variant is presented in the results above. These three measures were highly correlated (correlation coefficients ranging from 0.90 to 0.98), and the results were very stable across all three measures.

Third, we considered our choice of survival model. We first compared several parametric models (Weibull, Gompertz, Lognormal, Log-Logistic, and Gamma models) in terms of their fit to the data using the Akaike's information criterion (AIC) (Akaike, 1981). The Gompertz had the lowest AIC score for both all-cause mortality and CVD mortality, confirming that the Gompertz model is the best parametric survival model for these data. We then compared the parametric Gompertz models to semi-parametric Cox Proportional Hazard models. The coefficients across these two sets of models were very similar.

We also compared several specifications regarding the relative deprivation measure. We modeled the mortality outcome using a discrete time model, annualizing the data then running a logistic regression of death in each year and clustering the standard errors for individuals. This specification produced similar results as the Gompertz model. We also examined whether the inclusion of area-level controls altered the results for the relative deprivation models. (They did not change overall results; relative deprivation was still positively and significantly associated with mortality risk.)

Table 5
Results from hazard models of the association between census-based canton-level wealth inequality in 1984 and mortality.

	All-cause	Cardiovascular disease
Wealth	−0.0312** [0.0118]	0.00299 [0.0186]
Wealth Gini in 1984	−1.51** [0.48]	−1.63 [1.02]
	Hazard ratios (95% confidence intervals)	
HR for one std deviation increase in Gini	0.90	0.89
95% CI	(0.84–0.96)	(0.78–1.03)
Canton fixed-effects	No	No
Log-likelihood	−689	−1288
Observations	15276	14975

** Significant at 1%. Robust clustered standard errors in bracket. Models include controls for age at baseline, age at baseline squared, age at baseline cubed, gender, education, marital status, insurance status in 1984, living in San Jose Metro area in 1984, living in a rural area in 1984, and area-level mean income.

Finally, we estimated the second set of models with the time-varying canton-level characteristics and canton fixed-effects, but also included the baseline relative deprivation measure. The coefficients on both the income Ginis and relative deprivation remained largely the same.

Finally, to account for potential interdependence among observations within canton, we tried different ways of modeling this clustering and estimating appropriate standard errors. Regardless of method, either shared frailty models or clustering the standard errors, the standard errors were very similar. We chose to use clustering.

Discussion

We found that baseline income inequality was unexpectedly associated with *lower* mortality risk for both all-cause and CVD related mortality, but that the relationship largely disappears when we examine *changes* in income inequality and account for canton fixed-effects. In contrast, baseline relative deprivation was related to higher all-cause mortality risk even when controlling for canton fixed-effects. Likewise, baseline relative deprivation was related to higher CVD mortality risk, though when we controlled for canton fixed-effects, the standard errors nearly doubled and the relationship was no longer statistically significant (though magnitudes remained large and positive).

How do we reconcile results from the two measures of socioeconomic inequality? Focusing only on results where we controlled for canton fixed-effects, our results are in line with others who have compared income inequality and relative deprivation and have found a stronger relationship for relative deprivation measures than inequality measures (Eibner & Evans, 2005). We scrutinized this further in the sensitivity analysis by examining how a wealth-based inequality Gini relates to mortality risk. Regardless of the underlying data, the results were generally consistent across the wealth-based and income-based Gini measures. The results taken together suggest that canton-level economic inequality is not robustly related to all-cause or CVD mortality in Costa Rica, but that relative deprivation may be.

To further interpret our results, we compared them to the recent literature review by Kondo et al. (2009), which presented meta-regressions of relative mortality risks associated with income inequality Gini coefficients across cohort studies from numerous countries. They found that, in a set of countries with Ginis above 0.3 (U.S, New Zealand, and Norway), the overall relative risk confidence interval ranged from 1.07 to 1.12, though in a set of Scandinavian countries with Ginis below 0.3 the confidence interval ranged from 0.97 to 1.07. Our comparable point estimate for all-cause mortality is 0.96 [CI 0.92–1.00], though it increases to 0.99 [CI 0.94–1.04] in our fixed-effects regression. The Costa Rican nationwide Gini is in the 0.4 range; thus our results are substantially smaller than those from the comparably higher inequality countries, but are more similar to those of lower inequality countries. One hypothesis deserving future exploration is that Costa Rica's ethnic homogeneity, centralized resource allocation, and relatively strong social safety net (including universal health insurance) have helped buffer health outcomes against the adverse effects of income inequality. The fact that the wealth relative deprivation is still significantly associated with higher mortality suggests that the Costa Rican safety net has not been able to *fully* offset the effects of inequality, though the Costa Rican relative deprivation effects are smaller than those found in other contexts, such as the U.S. (Eibner & Evans, 2005) and South Africa (Salti, 2010).

Regarding area income and employment, we found several other seemingly paradoxical results that merit further discussion. First, we found that higher area-level mean income was associated with lower survival time. This result contradicts initial expectations and some of the results for individual-level wealth where those with greater individual wealth had longer survival. In addition, individual-level wealth was not a robust predictor of hazard of dying across the models. These unexpected SES relationships have been documented before in Costa Rica (see Rosero-Bixby and Dow (2009) for further discussion of unexpected SES relationships with health and mortality in Costa Rica). From the second set of models, with the area-level fixed-effects, we again found counterintuitive results with regard to unemployment rate and mortality. Yet there is an emerging literature regarding the health benefits of area-level unemployment, especially in the U.S. In a series of papers, Ruhm and others suggest changes in area-level unemployment modify an individual's time-use preferences and lead to better health behaviors and more social interactions for both the employed and unemployed (Edwards, 2011; Miller, Page, Stevens, & Filipski, 2009; Ruhm, 2000, 2005, 2007).

Several weaknesses should be noted. First, using canton as a reference group may be problematic if individuals do not compare themselves to those living nearby, but rather to the nation as a whole. (An inherent problem in much of the literature, this requires further theoretical and empirical clarification.) Second, it may be that unobservable characteristics—such as an individual's discount rate or level of risk-aversion—are related to both socioeconomic status and health behaviors, thereby confounding our results (though not if the direction of these relationships is in the opposite direction or if these characteristics are along the causal pathway). Third, another limitation of this type of longitudinal cohort study is potential bias from migration across cantons. If people move randomly across cantons, then the baseline inequality measure will not accurately reflect the actual inequality exposures across the follow-up period, which could result in attenuated inequality estimates on mortality. In our sample we observe 18% of people moving cantons between our electoral roll observations of 1989 and 2005. There would also have been limitations of measuring contemporaneous inequality in a person's canton as he moves, because it is likely that some migration is endogenous to both health behaviors and health outcomes (i.e., elderly will often move closer to urban children and hospitals as their health fails). Thus, we chose to focus on the more exogenous baseline residence. Fourth, with regard to the income inequality analysis, there are issues of measurement error. The measures of income inequality were based on household surveys done on a 1% sample; for some cantons the resultant sample size of households is small. We examined this in the sensitivity analysis by using a wealth inequality measure that does not suffer from measurement error. Our analysis suggests that the measurement error problem is not driving our results. Finally, with regard to the relative deprivation analysis, we have issues with top-coding and timing. While the relative deprivation measures are unlikely to be mismeasured (because they are based on a full census and all amenities were fully observable by the enumerators), we have only 10 levels of wealth. Accordingly, we probably underestimate the level of heterogeneity of real socioeconomic assets within cantons. In addition, ownership of these amenities is likely to change over the life course—especially at younger ages—and we cannot capture that adequately.

Notwithstanding these limitations, this study makes several contributions. First, it has an exceptionally long follow-up period. There are few studies with as many observations followed for so long on a nationally representative sample. This is by far the longest cohort study for a developing-country population. Second, Costa Rica's institutional setting and homogenous population make it an appealing case to study the inequality–health relationship; these characteristics may explain why we find smaller effects for relative deprivation than in other contexts where political channels and

race relations are likely to be important. Third, this study compares multiple measures of socioeconomic inequality. While comparing the results from these different measures of economic inequality may hinder making strong conclusions because results may seem contradictory, it is precisely this type of nuanced picture that suggests direction for future work.

Based on the present analysis, it appears that relative deprivation is robustly related to adverse mortality outcomes, but the relationship with income inequality is not consistent. This suggests that having less than people ranked above oneself in the Costa Rican hierarchy may be more detrimental to health than the overall dispersion of the hierarchy.

Appendix. Supplementary material

Supplementary data related to this article can be found online at doi:10.1016/j.socscimed.2011.10.034.

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