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Income Inequality and Health: Evidence from Developed and Developing Countries

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Abstract
The authors assess the effect of income inequality on life expectancy by performing separate estimations for developed and developing countries. Their empirical analysis challenges the widely held view that inequality matters more for health in richer countries than for health in poorer countries. Employing panel cointegration and conventional panel regressions, they find that income inequality increases life expectancy in developed countries. By contrast, the effect on life expectancy is significantly negative in developing countries. While the quantitative effects are small, the striking contrast between the two country groups proves to be robust to modifications in measurement, specification and methodological choices.

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Keywords Health; Inequality; Panel cointegration

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1. INTRODUCTION

Income disparities have widened in various developed and developing countries during the process of economic globalization. Critics have called for redistributive policy interventions, not only for reasons of fairness but also to avoid economic and social costs of wide income gaps. Impaired health could add considerably to such costs.

The earlier literature suggests that the case for health-related redistribution is particularly strong for developed countries.[1] According to Wilkinson (1996: 4), the distribution of income is "one of the most powerful influences on the health of whole populations in the developed world to have come to light."[2] Lynch et al. (1998: 1074) reckoned that the loss of life from income inequality in the United States "is comparable to the combined loss of life from lung cancer, diabetes, motor vehicle crashes, human immunodeficiency virus (HIV) infection, suicide, and homicide in 1995." The so-called Whitehall studies on British civil servants showed "that, even among people who are not poor, there is a social gradient in mortality that runs from the bottom to the top of society" (Marmot, 2003: S10).[3]

In contrast to developed countries, income inequality may play a secondary role in low-income countries with pervasive absolute poverty. Under such conditions, it could be low income per se that matters most for health and mortality, rather than income relative to other peoples' incomes (Deaton, 2003). Importantly, this so-called absolute income or poverty hypothesis implies that previous findings on inequality and health in developed countries do not necessarily hold for developing countries.[4] We account for this possibility by performing separate estimations for two samples of developed and developing countries.

Empirical evidence on the health effects of income inequality continues to be scarce for developing countries, largely because of lacking data on income inequality for a sufficiently large sample and a sufficiently long period of time. Our analysis contributes to filling this important gap by drawing on a relatively new data set, the Standardized World Income Inequality Database

(SWIID, 2013), developed by Solt (2009). This data set combines information from several sources, resulting in greater coverage and better comparability (for details, see Section 3.b). Furthermore, many previous studies share limitations that we attempt to overcome in the subsequent analysis. Most importantly, the endogenous nature of income inequality is often acknowledged, but rarely addressed appropriately in the empirical analysis. The panel cointegration approach pursued in the following accounts for endogeneity concerns and is robust to omitted variables.

Before describing in more detail our methodological approach and the data employed (Section 3), we provide an overview of the analytical background in Section 2, focusing on the theoretical ambiguity of the relationship between income inequality and health outcomes. Section 4 presents the empirical results for our samples of developed and developing countries, and Section 5 concludes. We find that income inequality has a small, but robust and significantly positive impact on health outcomes in developed countries. In contrast, the effect on life expectancy is significantly negative in developing countries. While the quantitative effects are small, the striking contrast between the two country groups proves to be robust to modifications in measurement, specification and methodological choices.

2. ANALYTICAL BACKGROUND

Several lines of reasoning in the relevant literature suggest that a more equal distribution of income is associated with better average health outcomes such as longer life expectancy and lower mortality. Nevertheless, there is considerable theoretical ambiguity in various respects. Preston's (1975) finding of a non-linear relationship between life expectancy and average per-capita incomes across countries provided an important building bloc of the so-called absolute income hypothesis, which has also been coined the poverty hypothesis (e.g., Deaton, 2003). The most obvious explanation for this non-linearity is that it reflects diminishing returns to increases in income

(Preston, 1975: 241). Increases in income would have larger positive effects on health outcomes among poor people than on health outcomes among rich people.

Consequently, mean-preserving income redistribution from the rich to the poor — within countries or between countries — would be associated with better average health. Health conditions among the rich might suffer to some extent from such income transfers, but improved health conditions among the poor would over-compensate any adverse effects on the rich.[5] The fact that diminishing returns to personal income imply a negative association between income inequality and health conditions at the aggregate level has been labeled a "statistical artifact" by Gravelle (1998). This notion is meant to distinguish the absolute income hypothesis from propositions according to which income inequality is directly hazardous to health (see below). In the present context, it is more important to note that the absolute income hypothesis requires the relationship between health and personal income to be concave.

Even though the Preston curve is widely accepted as a stylized empirical observation, the theoretical case for the concave relationship between health and income is open to debate. Grossman (1972) regards health as a durable capital stock that produces an output of healthy time. The marginal product of health capital increases with higher wage rates; "the higher a person's wage rate, the greater the value to him of an increase in healthy time" (Grossman, 1972: 241). Grossman's model thus predicts that the demand for health and medical care should be positively correlated with wage rates and per-capita income. Similarly, Waldmann (1972: 1291) argues that "health care is plausibly a superior good." This could prevent diminishing returns to income to the extent that additional spending on health care translates into better health outcomes.[6] The absolute income hypothesis would not hold under such conditions.

Another line of reasoning expects directly hazardous effects of income inequality on health outcomes. The so-called relative income hypothesis, according to which equal societies are healthier, draws on concepts and insights from several disciplines, notably psychology, politics and

economics.[7] Wilkinson (1996; 1997; 2000), its most prominent proponent, argues that the epidemiological transition from infectious diseases to chronic and degenerative diseases implies that the major reason for differences in mortality and health shifts from (absolute) material deprivation to (relative) social disadvantage. Social disadvantage is supposed to give rise to psychosocial stress and relative deprivation. Unequal societies are characterized, according to Wilkinson (2000: 4), by "much more stressful strategies of dominance, conflict and submission." At the same time, biologists have shown that chronic stress impairs health by permanently perturbing the physiologic balance (Sapolsky, 2004).

This reasoning, though plausible, does not necessarily imply impaired health due to income inequality. First, inequality may be closely linked with relative deprivation, "but there is little that suggests it is *income* inequality" (Deaton, 2003: 152). Second, rank matters and (upward) comparisons of one's own well-being with higher ranked individuals in relevant reference groups may be stressful.[8] However, deprivation and adverse health effects would be contained if inequality within specific reference groups was low compared to economy-wide inequality. Indeed, inequality appears to be lower in groups of people who have much in common, such as co-workers, friends, relatives, and neighbors (Leigh et al., 2009). Furthermore, people typically belong to various reference groups and tend to reduce stress by deriving self-esteem from the reference group where their ranking is highest.[9] Third, while social subordination often involves stress and an increased risk of stress-related diseases, Sapolsky (2004: 397 and 408) concludes from surveying the relevant literature that there are "numerous" and "dramatic" exceptions to this profile. In unstable hierarchies, stress centers on the higher ranks as dominant individuals constantly need to defend their position against emerging competitors.[10]

Income inequality could also impair health conditions by eroding social trust and affecting the political process of delivering public goods. It is widely agreed that mutual trust and social capital are associated with better health (e.g., Kawachi et al., 1997; d'Hombres et al., 2010;

Ronconi et al., 2012).[11] Trust and social capital could also help contain violent crime that may have minor direct effects on mortality and life expectancy, but could have considerable second-order effects by creating chronic stress among potential victims (Leigh et al., 2009). It is less obvious that adverse health effects of a disrupted social fabric can be traced back to income inequality. On the one hand, Alesina and La Ferrara (2002) find that a higher degree of income disparity is one factor among others eroding mutual trust among individuals in US localities. According to Leigh (2006), a negative effect of income inequality on trust can also be observed across countries. On the other hand, reverse causality from trust and social capital to inequality cannot be ruled out. Indeed, Knack (2002: 71) shows that social capital is "progressive, in the sense that it helps the poorer classes more than it helps the richer classes." This seems to suggest that inequality represents the intermediating variable through which social capital affects health, rather than being the ultimate cause of impaired health.[12]

Ambiguity also prevails on whether income inequality is associated with less or more public spending on health.[13] As noted by Leigh et al. (2009), the Meltzer-Richard theorem predicts that an increase in the mean income, relative to the income of the median voter, increases the size of government (Meltzer and Richard, 1981). A wider gap between the poor and the rich would thus encourage redistribution. Saint-Paul and Verdier (1993) model redistribution through public spending on education, showing that more inequality is associated with higher spending on education since the median voter (being poorer than the mean) prefers a higher rate of taxation. The reasoning of Saint-Paul and Verdier would also apply to health-related spending. It cannot be ruled out, however, that more inequality reduces the support for public spending on health or education. This could happen if poorer population segments participate less in the electoral process than richer population segments. The preferences of the poor may also be underrepresented because of the political clout of the rich elite and the associated pressure for lower taxes.[14]

Finally, it is debatable whether the above noted superior-goods character of health care would strengthen the economic justification of the relative income hypothesis. According to Waldmann (1992), infant mortality could be expected to increase if the rich receive a higher income share and health care is a superior good. Waldmann (1992: 1291) argues that the relative cost of health care would increase when the rich demand more medical services, leaving fewer medical resources for the poor. This reasoning rests on fairly restrictive assumptions. In public health systems with universal access, the poor may even benefit from living where income inequality is relatively high and where the demand of the rich results in better medical facilities (Miller and Paxson, 2006). The cost of medical services supplied at the request of the rich must not necessarily rise if the plausibly high fixed costs of sophisticated facilities are distributed among larger numbers of (rich and poor) users. Miller and Paxson (2006) make a similar argument with respect to the provision of health-related public goods such as stricter environmental regulations. Health conditions could improve with income inequality if the demand for environmental quality stems mainly from people whose income exceeds a certain threshold.

In summary, there is considerable theoretical ambiguity so that the health effects of income inequality are essentially an empirical issue. The subsequent panel cointegration analysis provides a particularly useful empirical approach to help clarify the causal links between income inequality and health outcomes. Clearly, cross-section analyses face problems to establish the direction of causality (Kawachi et al., 1997: 1497).[15] More surprisingly perhaps, even recent panel studies do not systematically address causality issues.[16] Reverse causality is possible, or even likely, as ill health may widen income gaps in several ways (Borghesi and Vercelli, 2004; Deaton, 2003; Leigh et al., 2009). Measures that equalize health conditions across the population, e.g., clean water supply in relatively poor countries, are also likely to narrow income gaps. Better health enhances people's earning capacity by reducing absenteeism from work and improving productivity at work. Health conditions within poor families affect the level of education and, thus, the income potential

of their children. Income differences across countries could be reduced if health conditions improved in poorer countries through faster diffusion of superior health technology and drugs. Hence, it appears essential to employ empirical methods that account for bidirectional causality.

3. EMPIRICAL MODELS AND DATA

Our objective is to examine the effect of income inequality on health in developed and developing countries using panel cointegration techniques as well as conventional regression techniques. In this section, we present the empirical models and discuss some econometric issues (Subsection a). Then, we describe the data, report descriptive statistics, and present some preliminary evidence (Subsection b).

(a) *Empirical models and econometric issues*

Following common practice in (panel) cointegration studies (see, for example, Pedroni, 2007; Herzer, 2008; Moscone and Tosetti, 2010), we consider a parsimonious model which includes only the two variables of empirical interest: inequality and health. Thus, the basic model takes the form

$$LE_{it} = a_i + \delta_i t + bGini_{it} + \varepsilon_{it}\,, \qquad (1)$$

where the subscript i refers to one of the N cross-sectional units, $i = 1, 2, ..., N$, and the subscript t refers to one of the T time points, $t = 1, 2, ..., T$. LE_{it} is the most commonly used summary measure of health status—life expectancy at birth (Henderson, 2009), and $Gini_{it}$ represents the standard measure of income inequality—the Gini coefficient (measured on a 0 to 100 scale). Following previous inequality-health studies (see, for instance, Leigh and Jencks, 2007), we use LE_{it} in levels rather than in logs. The coefficient b thus captures the permanent change in life expectancy (in years) associated with an increase in the Gini index by one unit. In the robustness section, we also use the infant mortality rate as an alternative measure of population health and the top-decile

income share as an alternative measure of inequality. Finally, any country-specific omitted factors which are relatively stable in the long run or which evolve smoothly over time are captured by country-specific fixed effects, a_i, and country-specific time trends, $\delta_i t$. The country fixed effects account for factors such as climate/geography, institutions, culture, and norms, while the individual time trends capture factors such as country-specific (medical) technological progress.

Given that all variables exhibit trends (as shown in Figures A.1 and A.2 in Appendix A), it is reasonable to assume that LE_{it} and $Gini_{it}$ are non-stationary integrated processes. If this assumption is correct, the linear combination of the two variables must be stationary, or, in the terminology of Engle and Granger (1987), LE_{it} must be cointegrated with $Gini_{it}$. Otherwise, there is no long-run relationship between life expectancy at birth and income inequality; Eqn. (1) would in this case represent a spurious regression in the sense of Granger and Newbold (1974). Entorf (1997) and Kao (1999) demonstrate that the tendency for spuriously indicating a relationship may even be stronger in panel data regressions than in pure time-series regressions. Thus, the necessary conditions for our model to be a correct description of the data are that LE_{it} and $Gini_{it}$ are non-stationary or, more specifically, integrated of the same order, I(1), and cointegrated.

A regression consisting of (non-stationary) cointegrated variables has the property of superconsistency such that coefficient estimates converge to the true parameter values at a faster rate than they do in standard regressions with stationary variables, namely rate T rather than $\sqrt{T}$ (Stock, 1987). The important point in this context is that the estimated cointegration coefficients are superconsistent even in the presence of temporal and/or contemporaneous correlation between the stationary error term, ε_{it}, and the regressor(s) (Stock, 1987), implying that cointegration estimates are not biased by omitted *stationary* variables (see, for instance, Bonham & Cohen, 2001).

The fact that a regression consisting of cointegrated variables has a stationary error term also implies that no relevant *non-stationary* variables are omitted. Any omitted non-stationary variable that is part of the cointegrating relationship would become part of the error term, thereby producing

non-stationary residuals, and thus leading to a failure to detect cointegration (see also Everaert, 2011).

If there is cointegration between a set of variables, then this stationary relationship also exists in extended variable space. In other words, the cointegration property is invariant to model extensions (see also Lütkepohl, 2007), which is in stark contrast to regression analysis where one new variable can alter the existing estimates dramatically (Juselius, 2006, p. 11). The important implication of finding cointegration is thus that no additional variables are required to account for the classical omitted variables problem. More specifically, the result for the long-run relationship between life expectancy and inequality would also hold if additional variables were included in the model (see also Juselius, 1996).

Of course, there are several other factors that may affect population health and/or income inequality. Therefore, adding further non-stationary variables to the model may, on the one hand, result in further cointegrating relationships. If, however, there is more than one cointegrating relationship, identifying restrictions are required to separate the cointegrating relationships. Otherwise, multicollinearity problems may arise. On the other hand, adding further non-stationary variables to the regression model may result in spurious associations. More specifically, if a non-stationary variable that is not cointegrated with the other variables is added to the cointegrating regression, the error term will no longer be stationary. As a result, the coefficient of the added variable will not converge to zero, as one would expect of an irrelevant variable in a standard regression (Davidson, 1998).

These considerations justify a parsimonious model such as Eqn. (1) (if cointegrated). All the same, we check the robustness of the results to the inclusion of additional control variables. More specifically, we follow Leigh and Jencks (2007) and include GDP per capita and GDP per capita squared.

The superconsistency of the cointegration estimation also implies that the potential endogeneity of the regressors should not affect the estimated long-run coefficients; the estimated long-run coefficients from reverse regressions should be approximately the inverse of each other due to the superconsistency (Engle & Granger, 1987). However, although the standard least-squares dummy variable (LSDV) estimator is superconsistent under panel cointegration, it suffers from a second-order asymptotic bias arising from serial correlation and endogeneity in finite samples. As a consequence, its *t*-ratio is not asymptotically standard normal. To deal with this problem, one has to employ an asymptotically efficient (cointegration) estimator. Examples of such estimators include panel versions of the dynamic OLS (DOLS) and fully modified ordinary least squares (FMOLS) methods. As shown by Wagner and Hlouskova (2010), the panel DOLS estimator of Mark and Sul (2003) outperforms other asymptotically efficient panel cointegration estimators in obtaining reliable long-run coefficients. Therefore, this DOLS estimator is our preferred estimator, but in the robustness section we also present results based on alternative estimation procedures.

The idea behind the DOLS estimator is to account for possible serial correlation and endogeneity of the regressors by augmenting the cointegrating regression (given by Eqn. (1)) with lead, lag, and current values of the first differences of the I(1) regressor(s). Accordingly, in our case, the DOLS regression is given by:

$$LE_{it} = a_i + \delta_i t + b\,Gini_{it} + \sum_{j=-k}^{k} \theta_{ij} \Delta Gini_{it-j} + e_{it}, \qquad (2)$$

where Δ is the difference operator (such that $\Delta Gini_{it} = Gini_{it} - Gini_{it-1}$) and k is the number of leads and lags. We use one lead and lag in the DOLS estimations to preserve degrees of freedom, as is common practice in the literature (see, for instance, Spilimbergo & Vamvakidis, 2003; Thorbecke & Smith, 2010; Herzer et al., 2012).

Another empirical issue is the likely cross-sectional dependence among the variables. Cross-sectional dependence may be the result of a common business cycle and other common factors such

as health shocks. Examples of such shocks that affect health in multiple countries at the same time might include major influenza epidemics, the spread of HIV/AIDS, the introduction of new vaccines, and the diffusion of antibiotics (Leigh et al., 2009). To control for potential cross-sectional dependence, we estimate the long-run effect of income inequality on health status using both the raw data and demeaned data; that is, in place of LE_{it} and $Gini_{it}$, we also use

$LE_{it}' = LE_{it} - \overline{LE_t}$ and

$Gini_{it}' = Gini_{it} - \overline{Gini_t}$, where

$\overline{LE_t} = N^{-1}\sum_{i=1}^{N} LE_{it}$ and

$\overline{Gini_t} = N^{-1}\sum_{i=1}^{N} Gini_{it}$, (3)

which is equivalent to including time dummies. Moreover, we use a battery of panel unit root and cointegration tests, including so-called second-generation panel unit root and cointegration methods that explicitly allow for cross-sectional dependence.

A potential disadvantage of panel cointegration methods is that they typically require balanced panel data over a sufficiently long time period. Continuous time series data on some alternative measures of inequality and health are not available for many countries over long periods of time. To check the robustness of our results to alternative measures of inequality and health, we are thus forced to use conventional panel methods. More specifically, we use data on the tuberculosis incidence rate and the income share of the bottom quintile to estimate a standard autoregressive distributed lag (ARDL) model of the form

$$Health_{it} = a_i + \lambda_t + \sum_{j=1}^{k} \gamma_j Health_{it-j} + \sum_{j=0}^{k} \beta_j Inequality_{it-j} + \sum_{j=0}^{k} \chi_j Controls_{it-j} + e_{it} \quad (4)$$

where $Health_{it}$ and $Inequality_{it}$ stand for the measures of health and inequality, a_i are country-specific fixed effects (as before), and λ_t represents time dummies. As control variables, we include GDP per capita and GDP per capita squared following Leigh and Jencks (2007), as discussed

above. The number of lags is set to $k = 2$ when the time period is sufficiently long (about 30 years); otherwise (when T is about 20 years) we use one lag, $k = 1$. The long-run effect of a change in inequality on health is given by

$$b = \frac{\sum_{j=0}^{k} \beta_j}{1 - \sum_{j=1}^{k} \gamma_j}. \tag{5}$$

As is well known, the dynamic fixed effects model may suffer from the so-called Nickell (1981) bias; that is, the correlation between the lagged dependent variable and the fixed effects may bias the coefficient on the lagged dependent variable toward zero. However, the bias becomes small when T is about 20 or more. Judson and Owen (1999) compare the performance of different estimators in terms of Nickel bias and recommend the LSDV estimator in unbalanced panels with $T = 30$. Bun and Kiviet (2006) examine the performance of several dynamic panel estimators in samples where both T and N are moderate or small and conclude that none of these estimators (including GMM and LSDV) dominates the others in terms of bias or mean squared error. We use the LSDV estimator given the relatively long time dimension of our data.

(b) *Data and preliminary evidence*

We estimate both Eqn. (2) and Eqn. (4) for developed and developing countries separately. The data on life expectancy at birth are from the World Development Indicators (WDI) 2013 online database.[17] Life expectancy at birth indicates the number of years a newborn infant would live if prevailing patterns of mortality at the time of its birth were to stay the same throughout its life. Life expectancy is the most widely used indicator of health status and has also several advantages over other measures of health, including the following: (i) it depends on both infant mortality and other mortality rates, thus incorporating mortality rates at all stages in life; (ii) it is not biased by age structure; and (ii) data on life expectancy at birth are available for a reasonably large number of countries and time periods.

However, the use of life expectancy as an indicator of health can be criticized on two major grounds. First, longer life expectancy does not necessarily translate into better health. While this is theoretically correct, we find that life expectancy at birth and healthy life expectancy at birth, i.e., life expectancy at birth adjusted for morbidity and time spent in poor health, are highly correlated. According to data from the Institute for Health Metrics and Evaluation,[18] the cross-country correlation coefficient between life expectancy at birth and healthy life expectancy at birth for women (men) was 0.994 (0.994) in 1990, and 0.990 (0.999) in 2000. Thus, it is very unlikely that the results would change significantly if it were possible to use healthy life expectancy in place of life expectancy. Unfortunately, adequate data on healthy life expectancy are not available to conduct a meaningful panel data analysis. Therefore, we use (unadjusted) life expectancy as our main indicator of population health. As an alternative summary measure of population health, we use the infant mortality rate (per 1,000 live births), and as a specific measure of health status, we use the tuberculosis incidence rate (per 100,000 population). These two variables are also from the WDI 2013 online database.

The second limitation is that average life expectancy does not reveal the variation of health conditions within countries. The health conditions of poorer population segments tend to be worse than those of richer population segments—for economic reasons such as spending on health care and/or for reasons of social or psychic deprivation. There is evidence to this effect from selected case studies, typically for high-income countries, including the well-known Whitehall studies in the United Kingdom (e.g., Marmot, 2003; Anderson & Marmot, 2012) and for the United States (Singh & Siahpush, 2006). Comparable data do not exist for a panel analysis. However, it appears that the results achieved for average life expectancy ought to hold for life expectancy of poor population segments, if such data were available. This can be concluded at least tentatively when considering the prevalence of malnutrition among children under five as a marker of subsequent poor health and low life expectancy. The correlation between malnutrition, which can reasonably be assumed to be

prevalent among the poor and be absent among the rich, and average life expectancy is strongly negative.[19]

As discussed above, we include GDP per capita (in constant 2005 US dollars) and GDP per capita squared as additional explanatory variables. These data are also taken from the WDI.

As far as data on income inequality are concerned, several studies have used the Gini coefficient data set constructed by Deininger and Squire (1996). At least since the work of Atkinson and Brandolini (2001) it is well known, however, that the Deininger-Squire data suffer from deficiencies such as sparse coverage, problematic measurements, and the combination of diverse data types into a single data set, thus limiting the comparability, not only across countries but also over time. Many studies therefore rely on Gini data from the Luxembourg Income Study (LIS) database or the World Income Inequality Database (WIID). The major deficiency of all these sources is the lack of continuous and consistent inequality data over time. More generally, it should be noted that the Gini coefficient, though widely available and often used in empirical studies, is an imperfect measure on inequality. Most importantly, the Gini coefficient is not consistent with the welfare principle.[20]

In this study, we utilize a data source that combines the strengths of the LIS and WIID data—the Standardized World Income Inequality Database (SWIID, 2013) developed by Solt (2009).[21] The SWIID combines information from the LIS and WIID data to create an improved data set with greater coverage than the LIS data and greater comparability than the WIID data. The logic behind the methodology underlying the SWIID can be summarized as follows (see also Morgan & Kelly, 2013). The synchronization process for the SWIID starts by utilizing inequality data from both the LIS and the WIID. The WIID data contain several country-years not available from the LIS and often includes inequality statistics based on multiple income concepts (with some including and others excluding various cash and/or in-kind transfers) for the same country-year. The SWIID synchronization process treats inequality as a latent variable, with data from the LIS and the WIID

acting as imperfect indicators of the underlying concept. With knowledge from country-years in which the two data sets overlap, the SWIID uses inequality estimates from the strongly comparable LIS data set along with inequality estimates and information about the income concept represented in the WIID to adjust the WIID data such that it mimics the comparability of the LIS data. This yields data with greater comparability and more coverage than any other available data set.

Although many of the more recent income inequality studies use the (gross) SWIID Gini coefficient (see, for example, Desbordes & Verardi, 2012; Cole, 2013; Morgan & Kelly, 2013), this index has the limitation that it is estimated, and estimates may be biased (for several reasons). Therefore, we check the sensitivity of the cointegration estimates to the measure of inequality by using the top-decile income share data provided by Leigh (2007).[22] Leigh adjusts top incomes series from different studies to produce a comparable data set. However, these data are available only for a small number of high-income countries. For developing countries, we use the income share of the bottom 20 percent of the population from the WDI. [23] As noted above, these data, as well as the data on the tuberculosis incidence rates, are unbalanced, and cannot be employed in cointegration analysis. Therefore, we estimate the long-run effects from the ARDL model given by Eqn. (4).

In our main analysis, we focus on the cointegrating relationship between LE_{it} and $Gini_{it}$. In order to apply panel cointegration techniques, we need a balanced panel data set. The construction of such a data set involves a trade-off between the time span and number of countries in the sample. For the sample of developed countries, we select all high-income countries for which complete time-series data are available over the period 1976-2010—the longest time period with complete data for a reasonably large number of high-income countries according to World Bank (1995) classification. This yields a sample of 19 developed countries and 35 time-series observations per country (665 total observations). In the robustness section, the long-run relationship between LE_{it} and $Gini_{it}$ is also estimated for 21 developed countries over the period 1981-2005 (525 total observations); data for more countries are not available for this period.

For the period 1976-2010, balanced panel data are available only for 19 developing countries among which there are only three low-income countries. Given this small number of developing (low- and middle-income) countries according to World Bank (1995) classification, we decided not to choose the period 1976-2010 for the sample of developing countries. Instead, we choose the period 1981-2005, resulting in a balanced panel of 59 developing countries (including 18 low-income countries) and 25 time-series observations per country (1475 total observations).

Table 1 lists the countries in our sample of developed countries along with the average values for LE_{it} and $Gini_{it}$ over the period 1976-2010. Japan had the highest life expectancy, followed by New Zealand and the Netherlands. The United States was the country with the highest Gini, followed by the United Kingdom and Switzerland, while Australia had the lowest Gini.

[Table 1]

Table 2 shows the countries in the sample of middle- and low-income countries, their average Gini and their average life expectancy at birth over the period 1981-2005. Life expectancy at birth was highest in Greece and lowest in Sierra Leone. Indonesia had the highest Gini and Mauritius the lowest.

[Table 2]

In Tables 1 and 2 we also report the sample means of the variables used in the analysis, along with the minimum and maximum values of the data. As expected, life expectancy in developed countries is, on average, significantly higher than in developed countries, while income inequality is lower in developed countries compared to developing countries.

Finally, in Table 3 we test whether our data can be used to replicate some of the previous findings reported in the literature. Following Rogers (1979), Waldman (1992), and Beckfield (2004), among others, we use a pooled sample of (21) developed and (59) developing countries. The 80 countries included in this sample are listed in the notes to Figures A1 and A2 in Appendix A, where we plot LE_{it} and $Gini_{it}$ for the sample period 1981-2005. The dependent variable is life

expectancy at birth, and the regressors include the Gini coefficient, GDP per capita, and GDP per capita squared. Columns 1-3 of Table 3 present results from specifications without country and time dummies, while the results in columns 4-6 are based on specifications that include country and time dummies to control for time-invariant omitted-variable bias and common time effects. According to the results without country and time fixed effects, inequality appears to have a strongly negative and statistically significant effect on life expectancy, which is consistent with previous cross-sectional studies (see, for instance, Rogers, 1979; Wilkinson, 1992; Waldman, 1992). The results with country and time fixed effects are also in line with previous studies (see, for instance, Mellor & Milyo, 2001; Beckfield 2004, Leigh & Jencks, 2007): The coefficient on the inequality variable turns out to be insignificant in columns 4-6.

[Table 3]

Columns 3 and 6 show that when we add GDP per capita and GDP per capita squared to the basic specifications in columns 1 and 4, the coefficient on GDP per capita is positive and significant (as in column 2) and the coefficient on GDP per capita squared is negative and significant. Accordingly, increases in GDP per capita are associated with increases in life expectancy, but the effects diminish as GDP per capita rises, which is consistent with the results of Preston (1975), Deaton (2003), and Leigh and Jencks (2007).

4. EMPIRICAL ANALYSIS

In this section, we estimate the long-run effect of income inequality on population health for developed and developing countries separately. We first analyze the effect for developed countries (Subsection a), as most previous studies have done (see, for instance, Wilkinson, 1992; Wennemo, 1993; Judge et al., 1998; Leigh & Jencks, 2007), and check the robustness of our results (Subsection b). Subsequently, we provide estimates of the long-run effect of inequality on health in developing countries (Subsection c).

(a) *The long-run effect of inequality on health in developed countries*

The pre-tests for unit roots and cointegration, reported in Appendix B, suggest that LE_{it} and $Gini_{it}$ are non-stationary and cointegrated. This implies that there is a (non-spurious) long-run relationship between life expectancy at birth and the SWIID Gini coefficient. To estimate this relationship, we use the panel DOLS estimator suggested by Mark and Sul (2003). As discussed above, the DOLS estimator is superconsistent, asymptotically unbiased, and normally distributed, even in the presence of endogenous regressors.

Table 4 presents the results of this estimation procedure both for the raw data (column 1) as well as for the data that have been demeaned over the cross-sectional dimension (column 2). The estimated coefficient based on the raw data is positive but significant only at the 10% level. When the demeaned data are used (to account for the problem of cross sectional dependence induced by common, unobservable factors), the coefficient becomes significant at the one percent level. This suggests that an increase in inequality is associated with an increase in life expectancy in high-income countries, which is in contrast to most previous studies.

[Table 4]

What can be said about the magnitude of the estimated effect in column 2? Multiplying the coefficient of *Gini* (0.0381) with the average value of the change in the Gini coefficient in the sample (0.2075) yields value of 0.0079, implying that inequality has contributed about 0.0079 years to the annual increase in life expectancy for the average country in the sample. With an average increase in life expectancy of 0.2297 years, this means that inequality has been responsible for about 3.5 percent of the annual increase in life expectancy in the country sample considered here—a relatively small effect.

(b) *Robustness*

Given that the finding of a significantly positive relationship between inequality and health in high-income countries contradicts previous findings, we perform several robustness checks. First, we examine whether the positive relationship between inequality and health in developed countries is robust to alternative estimation techniques. A potential problem with the pooled results (in columns 1 and 2 of Table 4) could be that they are based on the implicit assumption of homogeneity of the long-run effects. While efficiency gains from the pooling of observations over the cross-sectional units can be achieved when the individual slope coefficients are the same, pooled estimators may yield inconsistent and potentially misleading estimates of the sample mean of the individual coefficients when the true slope coefficients are heterogeneous. A comparative study by Baltagi and Griffin (1997) concludes that "the efficiency gains from pooling appear to more than offset the biases due to intercountry heterogeneities" (p. 317). Nonetheless, we allow the long-run coefficients to vary across countries by using the group-mean panel DOLS estimator suggested by Pedroni (2001). This estimator involves estimating separate DOLS regressions for each country and averaging the long-run coefficients, $\hat{b} = N^{-1}\sum_{i=1}^{N}\hat{b}_i$. The corresponding *t*-statistic is computed as the sum of the individual *t*-statistics (calculated using heteroskedasticity and autocorrelation-consistent standard errors) divided by the root of the number of cross-sectional units, $t_{\hat{b}} = \sum_{i=1}^{N} t_{\hat{b}_i} / \sqrt{N}$. In addition, we use the pooled FMOLS estimator suggested by Phillips and Moon (1999). Like the time series FMOLS estimator, the panel FMOLS estimator incorporates a semi-parametric correction to the OLS estimator, which eliminates the second order bias induced by the endogeneity of the regressors. We report the results of these estimation methods in columns 3 and 4 of Table 4.

The results show a positive and significant effect of inequality on life expectancy. Interestingly, the panel and group-mean DOLS estimators produce almost identical coefficients, suggesting that slope heterogeneity is not a serious problem in this sample. The FMOLS coefficient estimate in column 4 is somewhat smaller than the DOLS estimate in column 2, but still positive

and significant at the one percent level. As shown by Wagner and Hlouskova (2010), these two estimators perform worse than the pooled DOLS estimator of Mark and Sul (2003). Therefore, we continue our robustness analysis using the pooled DOLS estimator (with the demeaned data).

To verify that the positive effect of inequality on health is not due to individual outliers, the DOLS regression is re-estimated excluding one country at a time from the sample. The sequentially estimated coefficients and their *t*-statistics are presented in Figure 1. They fluctuate between 0.033 (due to the exclusion of Ireland) and 0.043 (due to the exclusion of Australia) and are always significant at the one percent level, suggesting that the positive effect of inequality on health is not the result of individual outliers.

[Figure 1]

It is common practice in conventional panel studies to use time-averaged data to eliminate business cycle effects. However, as pointed out by Attanasio et al. (2000), annual data provide information that is lost when time-averaged observations are used. Moreover, is not obvious that averaging over fixed time intervals will effectively eliminate business cycle effects; the length of the interval over which averages are computed is arbitrary, and there is no guarantee that business cycles are cut in the right way, as their length varies over time and across countries. In addition, the use of time-averaged data decreases the number of observations, and hence statistical power. Despite these concerns, we re-estimate the DOLS regression using five-year averages. The results of this estimation are reported in column 1 of Table 5. As can be seen, the estimate using five-year averages is close to the corresponding estimate based on annual data reported in Table 4, column 2. This is consistent with several studies showing that cointegration estimates are remarkably stable across frequencies (see, for instance, Chambers, 2001; Click & Plummer, 2005; Herzer, 2013).

In column 2 of Table 5, we estimate the coefficient on $Gini_{it}$ using a larger sample of 21 high-income countries from 1981-2005 (which is the period used in the next section to analyze the

effect of inequality on health in developing countries). Once again, the estimated coefficient is positive and highly statistically significant.

As discussed in Section 3, the finding of cointegration implies that there are no missing trending variables and that therefore no additional variables are needed to produce unbiased estimates. Nevertheless, we check the robustness of our results to the inclusion of GDP per capita and GDP per capita squared. A potential problem with this strategy is that it can introduce collinearity among the stochastic regressors or between the additional variables and the individual time trends. To account for this problem, we present estimates with and without individual time trends. As can be seen from columns 3 and 4 of Table 5, the impact of inequality on health remains positive and statistically significant when we include GDP per capita and GDP per capita squared, regardless of whether or not individual time trends are used in the analysis (to represent technological change). While in column 3 the coefficients on GDP per capita and GDP per capita squared have unexpected signs, the signs of these coefficients in column 4 are as we expect: the coefficient on GDP per capita is positive and significant and the coefficient on GDP per capita squared is negative and significant (in the regression without deterministic time trends).

[Table 5]

Next, we examine whether the results are robust to alternative measures of inequality and mortality. Leigh and Jencks (2007) use data on the income share of the richest 10 percent to examine the effect of income inequality on health for 12 high-income countries in an unbalanced panel between 1903 and 2003. Here we construct a balanced panel for 8 high-income countries (Canada, France, Netherlands, New Zealand, Sweden, Switzerland, United Kingdom, and United States) for the period 1961-1996. Regrettably, complete time series on life expectancy at birth and the top income share are not available for more countries and years. We also use the infant mortality rate to check the robustness of our results, following many studies which regard infant mortality as an indicator of the general health status of the population. Table 6 presents the results of the DOLS

regressions using these two different measures, labeled *TopDecile* and *IMR*, both separately and jointly. All estimates suggest that income inequality increases health in high-income countries.

[Table 6]

A potential problem with these estimates is that life expectancy and infant mortality are measures of mortality rather than morbidity. While the two are highly correlated (see Section 3.b), it is morbidity rather than mortality which should be affected most by inequality. In other words, morbidity is, by definition, a better measure of the health response to income inequality than mortality (Soobader & LeClere, 1999). Regrettably, summary measures of morbidity which account for all diseases (whether physical, psychosomatic or psychiatric) are not available to conduct a meaningful panel analysis. A specific measure of morbidity is the tuberculosis incidence rate, which is available for a sufficient number of countries over the period 1990-2011. Because the data are unbalanced, we do not apply the DOLS estimator, but estimate the long-run effect of inequality on the incidence of tuberculosis using the ARDL model. Table 7 presents the results with and without control variables. The estimated long-run coefficient on *Gini* is always negative and highly significant, suggesting that inequality decreases the incidence of tuberculosis. This corroborates the finding that inequality has a positive effect on population health in developed countries.[24]

[Table 7]

In summary, we find that income inequality has a positive and robust effect on population health in developed countries. In the next subsection, we investigate whether this result also holds for developing countries.

(c) *The long-run effect of inequality on health in developing countries*

In Table 8, we present DOLS estimates of the long-run effect of inequality on health in developing countries using life expectancy and infant mortality as measures of health outcomes; the measure of inequality is the Gini index. The estimated coefficient in column 1 is negative and

highly significant, suggesting that inequality is negatively related to life expectancy. Column 2 reports a positive and significant coefficient, suggesting that inequality is positively associated with infant mortality. Thus, in contrast to the results for developed countries, the results for developing countries show that inequality harms health. This is consistent with the results of the few previous studies that have examined the effect of income inequality on mortality in a (sub)sample of developing countries (see, for instance, Rogers, 1997; Flegg, 1982; Waldman, 1992). To quantify the effect, we multiply the coefficient of *Gini* in column 1 with the average change in the Gini index. The result implies that the average loss of life expectancy due to inequality is about one day per year ($-0.003 \times 365 = -1.095$).

[Table 8 about here]

We also report results based on the income share of the bottom quintile, labeled *BottomQuintile*. Balanced panel data on this measure of inequality are not available, which prevents us from using the DOLS procedure. Instead, we employ the ARDL model. The results in Table 9 show that the long-run coefficient on *BottomQuintile* is always positive and significant, indicating that inequality has negative consequences for health in developing countries. Finally, we used the ARDL model to perform estimations with the incidence of tuberculosis as a specific measure of morbidity as in Table 7 above for the sample of developed countries.[25] The Gini index enters with a positive coefficient in the estimations for developing countries with the incidence of tuberculosis as the dependent variable, instead of life expectancy. This is in contrast to the corresponding result for developed countries, even though the coefficient on the Gini index fails to reach statistical significance at conventional levels. In other words, we again find that the results for developed countries do not carry over to developing countries.

The finding that inequality has a positive effect on health in developed countries and a negative effect in developing countries has an interesting implication once it is taken into account that the income distribution in developing countries is more unequal than in developed economies.

Taken together, our findings suggest that the health-impairing effects of inequality are stronger in more unequal countries than in more equal countries. This is consistent with the so-called threshold hypothesis (Kondo et al., 2009), which posits that income inequality harms health only if income gaps are sufficiently wide.

5. CONCLUSIONS

The widely held belief that more unequal societies are less healthy is politically highly relevant. Calls for redistributive policy interventions in order to improve health and ensure longer life expectancy would be justified, particularly if wide income gaps represent a major aspect of inequality within countries. This provided the motivation to re-assess Wilkinson's (1996) verdict that the distribution of income is one of the most powerful determinants of the health of whole populations. While recent studies have increasingly doubted this verdict for developed countries, the scant evidence available so far for developing countries posed the important question of whether the experience of developed countries would also hold for lower-income countries. Our empirical analysis addressed these unresolved issues. In addition, we attempted to overcome several limitations of previous research by employing panel cointegration techniques, which allowed us to account for endogeneity concerns.

We found that income inequality has a significantly positive impact on population health in developed countries. Even though wider income gaps increase life expectancy only by a quantitatively small margin, this result proved to be robust to modifications in measurement, specification and methodological choices. It was surprising to find wider income gaps to cause slightly better health outcomes in developed countries, although some recent studies pointed into the same direction, notably Mellor and Milyo (2001) as well as Leigh and Jencks (2007). Concepts and insights from different disciplines such as psychology, political science and economics offer some tentative explanations. For instance, certain stress-related health risks may center on higher-

income ranks if hierarchies are unstable and dominant individuals constantly need to defend their position. More inequality may be associated with higher government spending on health care, as the Meltzer-Richard theorem would predict. Health conditions, including for poorer population segments, may also improve in line with Miller and Paxson (2006) if the superior-goods character of medical and environmental services induces stronger demand for such services by richer people beyond a certain income threshold.

In contrast to developed countries, we found that people living in developing countries with wider income inequality have a significantly lower life expectancy than people living in developing countries with a more equal distribution of income. The health-impairing effect of income inequality in developing countries is quantitatively small, but fairly robust. The case for redistributive policy interventions to improve health and increase life expectancy thus appears to be considerably stronger for developing countries than for developed countries. This is in some conflict with predictions from the absolute income or poverty hypothesis, according to which health primarily depends on the incidence of poverty in low-income countries. Unfortunately, our findings suggest that progressive income taxation might be advisable for health reasons exactly where wide income gaps tend to be most difficult to redress via taxation – due to insufficient administrative capacity and political resistance of local elites. Consequently, the preferred policy response may still consist of targeted pro-poor interventions with regard to the provision of health services. Improving the education of poor population segments could provide another indirect handle to tackle the health-impairing effects of income inequality.

Generally speaking, health policies in both developing and developed countries should take into account that income disparity is just one manifestation of inequality. Paraphrasing Deaton (2003: 152), inequality may be important for health, even though the quantitative impact of *income* inequality on health is rather small and working in opposite directions. As indicated above, interdisciplinary research could provide further insights into the links between different aspects of

inequality and health conditions in developed and developing countries. In addition, deeper insights may be gained once persistent data constraints are relaxed. Continued efforts to collect information on life expectancy adjusted for morbidity and time spent in poor health are of particular importance in this regard. Furthermore, the measurement of health conditions should be refined in order to reveal differences within countries, notably between particularly poor and richer population segments in developing countries.

APPENDIX A. MAIN VARIABLES BY COUNTRY OVER THE PERIOD 1981-2005

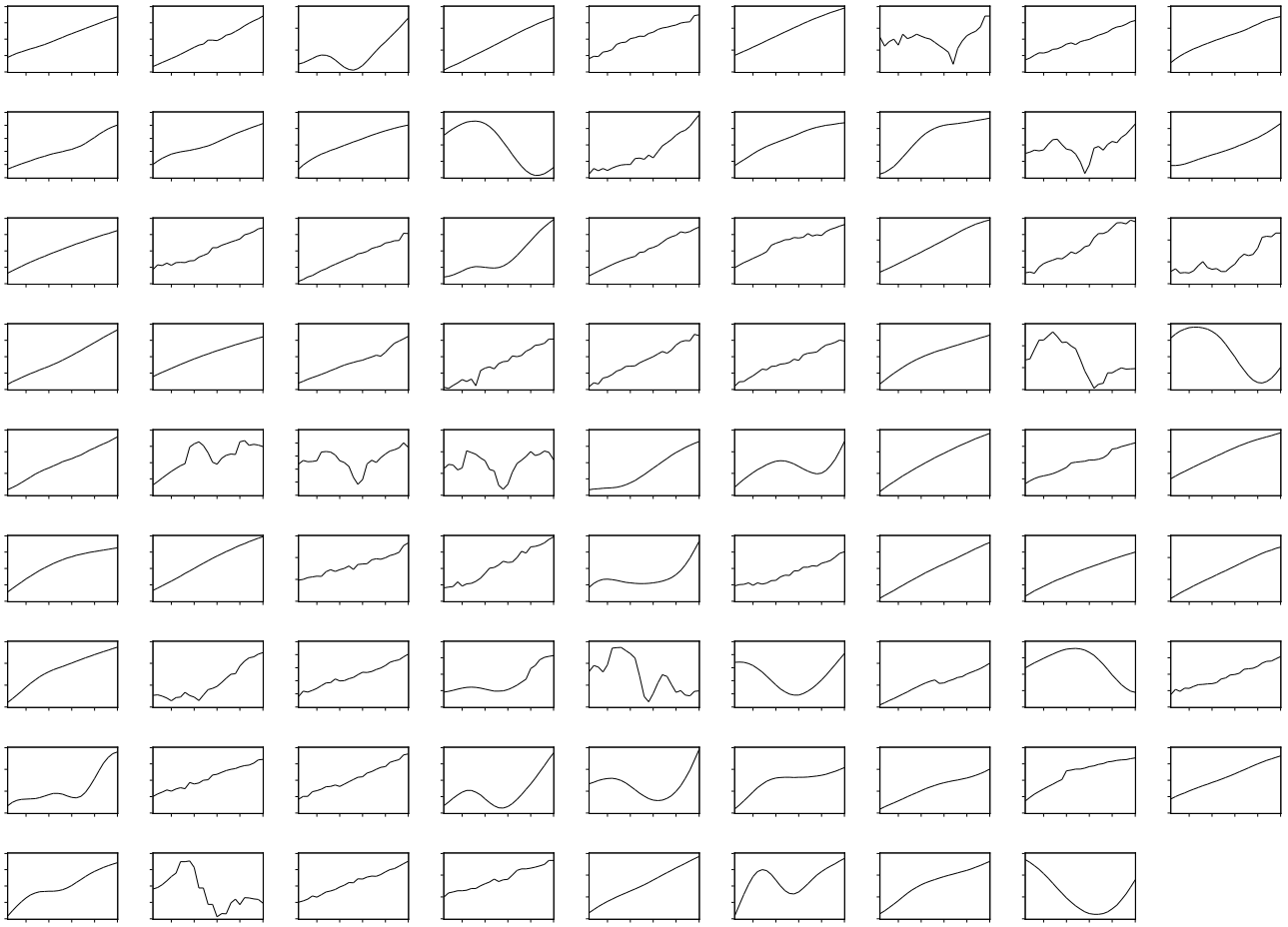

Figure A.1. *Life expectancy at birth by country over the period 1981-2005. Note: The figure includes all (80) countries for which complete time series data on life expectancy and the Gini coefficient are available over the period 1981 - 2005. The countries from left to right are: Argentina, Australia, Azerbaijan, Bangladesh, Belgium, Brazil, Bulgaria, Canada, Chile, China, Colombia, Costa Rica, Cote d`Ivoire, Denmark, Egypt, El Salvador, Estonia, Ethiopia, Fiji, Finland, France, Georgia, Germany, Greece, Guatemala, Hong Kong, Hungary, India, Indonesia, Ireland, Israel, Italy, Japan, Jordan, Kazakhstan, Kenya, Korea, Kyrgyz Republic, Latvia, Lithuania, Madagascar, Malawi, Malaysia, Mauritius, Mexico, Morocco, Nepal, Netherlands, New Zealand, Nigeria, Norway, Pakistan, Panama, Peru, Philippines, Poland, Portugal, Puerto Rico, Russian Federation, Sierra Leone, Singapore, South Africa, Spain, Sri Lanka, Sweden, Switzerland, Tajikistan, Tanzania, Thailand, Trinidad and Tobago, Tunisia, Turkey, Turkmenistan, Ukraine, United Kingdom, United States, Uruguay, Uzbekistan, Venezuela, and Zambia.*

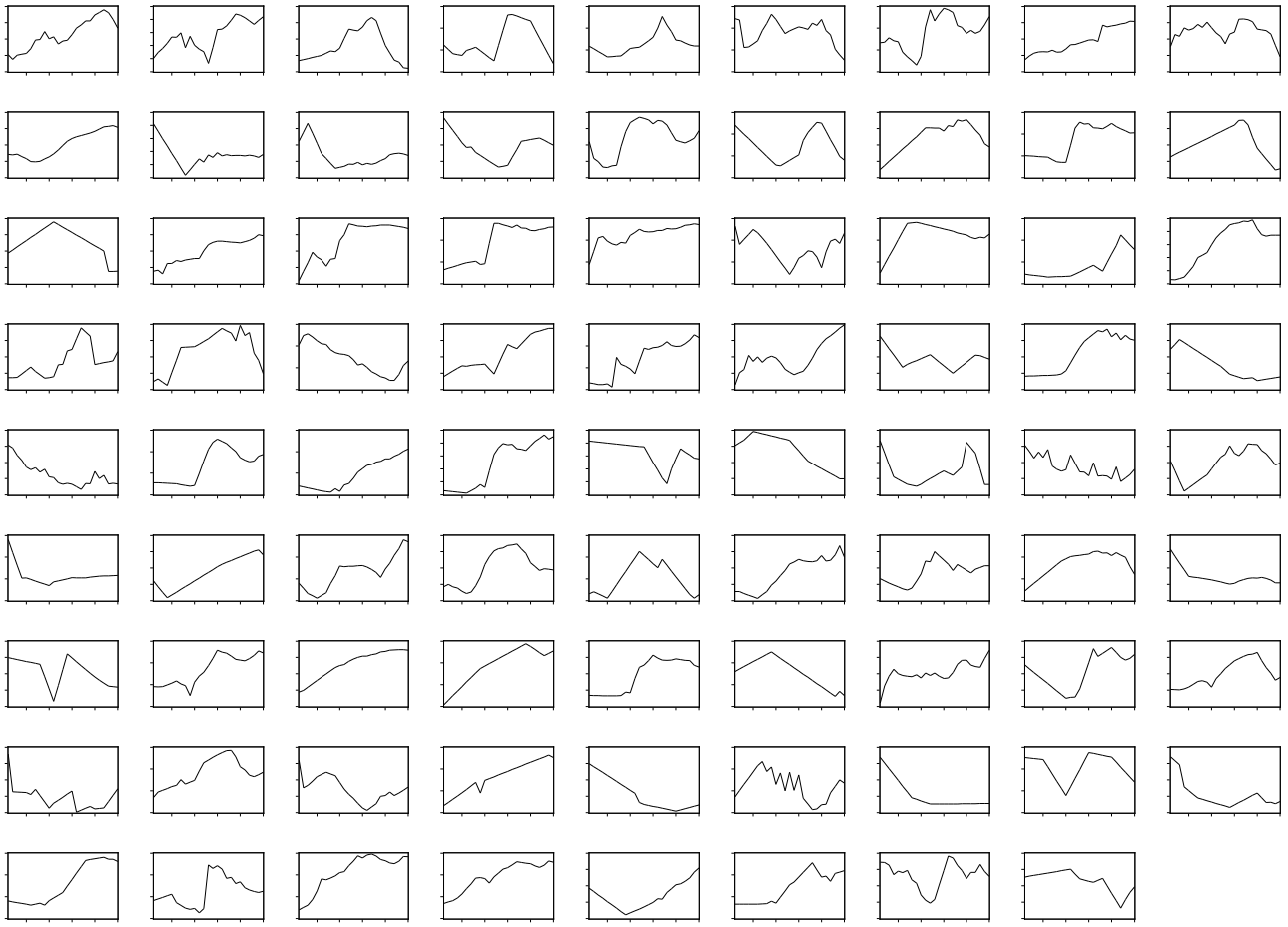

Figure A.2. *Gini coefficient by country over the period 1981-2005. Note: The figure includes all (80) countries for which complete time series data on life expectancy and the Gini coefficient are available over the period 1981 - 2005. The countries from left to right are: Argentina, Australia, Azerbaijan, Bangladesh, Belgium, Brazil, Bulgaria, Canada, Chile, China, Colombia, Costa Rica, Cote d`Ivoire, Denmark, Egypt, El Salvador, Estonia, Ethiopia, Fiji, Finland, France, Georgia, Germany, Greece, Guatemala, Hong Kong, Hungary, India, Indonesia, Ireland, Israel, Italy, Japan, Jordan, Kazakhstan, Kenya, Korea, Kyrgyz Republic, Latvia, Lithuania, Madagascar, Malawi, Malaysia, Mauritius, Mexico, Morocco, Nepal, Netherlands, New Zealand, Nigeria, Norway, Pakistan, Panama, Peru, Philippines, Poland, Portugal, Puerto Rico, Russian Federation, Sierra Leone, Singapore, South Africa, Spain, Sri Lanka, Sweden, Switzerland, Tajikistan, Tanzania, Thailand, Trinidad and Tobago, Tunisia, Turkey, Turkmenistan, Ukraine, United Kingdom, United States, Uruguay, Uzbekistan, Venezuela, and Zambia.*

APPENDIX B. PANEL UNIT ROOT AND COINTEGRATION TESTS

(a) *Panel unit root tests*

One of the most commonly employed tests for unit roots in panels is that of Im, Pesaran and Shin (2003), the IPS test. It tests the null hypothesis that all of the individuals of the panel have a unit root against the alternative that some fractions are (trend) stationary using the augmented Dickey-Fuller (ADF) regression for the *i*th cross-section unit

$$\Delta x_{it} = z_{it}\gamma_i + \rho_i x_{it-1} + \sum_{j=1}^{k_i} \varphi_{ij} \Delta x_{it-j} + \varepsilon_{it} \tag{A.1}$$

here k_i is the lag order, z_{it} represents deterministic terms, such as fixed effects or fixed effects combined with individual time trends, and Δ is the first-difference operator. To test the unit root null hypothesis, $H_0 : \rho_i = 0$, $\forall i = 1, 2, \ldots, N$, against the alternative of (trend) stationarity, $H_1 : \rho_i < 0$, $i = 1, 2, \ldots, N_1$; $\rho_i = 0$, $i = N_1 + 1, N_1 + 2, \ldots, N$, a standardized *t*-bar statistic is constructed as

$$\Gamma_{\bar{t}} = \frac{\sqrt{N}\left[\bar{t}_{NT} - \mu\right]}{\sqrt{v}}, \tag{A.2}$$

where $\bar{t}_{NT}$ is the average of the N (=19) cross-sectional ADF *t*-statistics, and μ and v are, respectively, the mean and variance of the average of the individual *t*-statistics, tabulated by Im et al. (2003).

However, the IPS test procedure assumes cross-sectional independence and can thus lead to spurious inferences if the errors, ε_{it}, are not independent across *i* (for instance, due to common shocks or spillovers between countries). Therefore, we also employ the cross-sectionally augmented IPS test proposed by Pesaran (2007). This test is designed to filter out the cross-section dependency by augmenting the ADF regression with the cross-section averages of lagged levels and first differences of the individual series. Accordingly, the cross-sectionally augmented ADF (CADF) regression is given by

$$\Delta x_{it} = z_{it}'\gamma_i + \rho_i x_{it-1} + \sum_{j=1}^{k_i} \varphi_{ij} \Delta x_{it-j} + \alpha_i \bar{x}_{t-1} + \sum_{j=0}^{k_i} \eta_{ij} \Delta \bar{x}_{t-j} + v_{it}, \quad \text{(A.3)}$$

where $\bar{x}_t$ is the cross-section mean of x_{it}, $\bar{x}_t = N^{-1}\sum_{i=1}^{N} x_{it}$. The cross-sectionally augmented IPS statistic is the simple average of the individual CADF statistics and is defined as

$$CIPS = t\text{-bar} = N^{-1}\sum_{i=1}^{N_i} t_i, \quad \text{(A.4)}$$

where t_i is the OLS t ratio of ρ_i in Eqn. (A.3). The corresponding critical values are given by Pesaran (2007).

The results of the two tests for the variables in levels and in first differences are reported in Table A.1. Both tests fail to reject the unit root null hypothesis in levels, whereas the unit root hypothesis is rejected for the first differences. From this we conclude that LE_{it} and $Gini_{it}$ are integrated of order 1, $I(1)$ — the necessary condition for cointegration in a bivariate context.

Table A.1. *Panel unit root tests*

Variables	Deterministic terms	IPS statistics	CIPS statistics
Levels			
LE	Constant, trend	0.58	-2.16
Gini	Constant, trend	-0.35	-2.37
First differences			
Δ*LE*	Constant	-6.00***	-2.41***
Δ*Gini*	Constant	-7.30***	-3.01***

Note: Three lags were selected to adjust for autocorrelation. The IPS statistic is distributed as N(0, 1). The relevant five- (one-) percent critical value for the CIPS statistics is -2.71 (-2.85) with an intercept and a linear trend, and -2.20 (-2.36) with an intercept. *** denote significance at the one percent level.

(b) *Panel cointegration tests*

We use several panel cointegration tests to examine whether there is a long-run relationship between live expectancy at birth and income inequality. The first is the two-step residual-based procedure suggested by Pedroni (1999, 2004), which can be intuitively described as follows. In the first step, the hypothesized cointegrating relationship

$$LE_{it} = a_i + \delta_i t + b_i Inequality_{it} + \varepsilon_{it} \quad \text{(A.5)}$$

is estimated separately for each country. In the second step, the residuals from these regressions are tested for stationarity based on

$$\hat{\varepsilon}_{it} = \rho_i \hat{\varepsilon}_{it-1} + \sum_{j=1}^{k_i} \varphi_{ij} \Delta \hat{\varepsilon}_{it-j} + w_{it} \tag{A.6}$$

To test the null hypothesis of a unit root or no cointegration, $H_0 : \rho_i = 1$, Pedroni proposes two classes of test statistics. The first category pools the autoregressive coefficients across different countries during the unit-root test and thus constrains the autoregressive parameters to be homogeneous across countries, $\rho_i = \rho$. Pedroni refers to these within-dimension-based statistics as panel cointegration statistics. The second class of statistics averages the individually estimated autoregressive coefficients for each country, thus allowing the autoregressive parameter to be heterogeneous across countries. Pedroni refers to these between-dimension-based statistics as group-mean panel cointegration statistics. The panel cointegration statistics include a non-parametric variance ratio statistic (panel *v*), a non-parametric Phillips and Perron type ρ-statistic (panel ρ), a non-parametric Phillips and Perron type *t*-statistic (panel PP) and a Dickey-Fuller type *t*-statistic (panel ADF). Similarly, the group-mean panel cointegration statistics include a Phillips and Perron type ρ-statistic (group ρ), a Phillips and Perron type *t*-statistic (group PP) and an ADF type *t*-statistic (group ADF). The standardized distributions for the panel and group statistics are given by

$$\kappa = \frac{\varphi - \mu\sqrt{N}}{\sqrt{\nu}} \Rightarrow N(0,1)\,, \tag{A.7}$$

where φ is the respective panel or group statistic, and μ and ν are the expected mean and variance of the corresponding statistic, tabulated by Pedroni (1999).

However, standard panel cointegration tests such as those of Pedroni (1999, 2004) assume cross-sectional independence and can have size distortions when this assumption is violated. To test for cointegration in the presence of possible cross-sectional dependence, we use a two-step residual-

based procedure in the style of Holly et al. (2010). In the first step, we apply the common correlated effects (CCE) estimator of Pesaran (2006) to the static cointegrating regression. Like the cross-sectionally augmented IPS test, the CCE estimator allows for cross-sectional dependencies that potentially arise from multiple unobserved common factors. The cross-sectionally augmented cointegrating regression for the *i*th cross-section is given by

$$LE_{it} = a_i + \delta_i t + b_i Inequality_{it} + g_{1i}\overline{LE_t} + g_{2i}\overline{Inequality_t} + e_{it}, \quad \text{(A.8)}$$

where the cross-sectional averages $\overline{LE_t} = N^{-1}\sum_i^N LE_{it}$ and $\overline{Inequality_t} = N^{-1}\sum_i^N Inequality_{it}$ serve as proxies for the unobserved factors. In the second step, we compute the cross-sectionally augmented IPS statistic for the residuals from the individual CCE long-run relations, $\hat{\mu} = LE_{it} - \hat{\delta}_i t - \hat{b}_i\, Inequality_{it}$, including an intercept. In doing so, we account for unobserved common factors that could be correlated with the observed regressors in both steps.

A drawback of residual-based (panel) cointegration tests is that they are generally not invariant to the normalization of the cointegrating regression. Therefore, we also use the Larsson et al. (2001) procedure, which is based on Johansen's (1988) maximum likelihood estimation procedure. Like the Johansen time-series cointegration test, the Larsson et al. panel test treats all variables as potentially endogenous, thus avoiding the normalization problems inherent in residual-based cointegration tests. In addition, the Larsson et al. procedure allows the long-run elasticities to differ from the short-run elasticities and hence does not impose a possibly invalid common factor restriction. It involves estimating the Johansen vector-error-correction model for each individual country:

$$\Delta y_{it} = \prod_i y_{it-1} + \sum_{i=1}^{k_i} \Gamma_{ik} \Delta y_{it-k} + z_{it}\gamma_i + \varepsilon_{it}, \quad \text{(A.9)}$$

where y_{it} is a $p \times 1$ vector of endogenous variables ($y_{it} = [\, LE_{it}\,;\ Inequality_{it}\,]'$; p is the number of variables), and Π_i is the long-run matrix of order $p \times p$. If Π_i is of reduced rank, $r_i < p$, it is

possible to let $\Pi_i = \alpha_i \beta_i$, where β_i is a $p \times r_i$ matrix, the r_i columns of which represent the cointegrating vectors, and α_i is a $p \times r_i$ matrix having p rows which represent the error-correction coefficients. The null hypothesis is that all of the N (=19) countries in the panel have a common co-integrating rank, i.e., at most r (possibly heterogeneous) cointegrating relationships among the p variables: $H_0 : rank(\Pi_i) = r_i \leq r$ for all $i = 1, \ldots, N$. The alternative hypothesis is that all the cross-sections have a higher rank: $H_1 : rank(\Pi_i) = p$ for all $i = 1, \ldots, N$. To test H_0 against H_1, a panel cointegration rank trace-test statistic is computed by calculating the average of the individual trace statistics, $LR_{iT}\{H(r)|H(p)\}$:

$$\overline{LR}_{NT}\{H(r)|H(p)\} = \frac{1}{N}\sum_{i=1}^{N} LR_{iT}\{H(r)|H(p)\}, \tag{A.10}$$

and then standardizing it as follows:

$$\Psi_{\overline{LR}}\{H(r)|H(p)\} = \frac{\sqrt{N}\left(\overline{LR}_{NT}\{H(r)|H(p)\} - E(Z_k)\right)}{\sqrt{Var(Z_k)}} \Rightarrow N(0,1). \tag{A.11}$$

The mean $E(Z_k)$ and variance $Var(Z_k)$ of the asymptotic trace statistic are tabulated by Breitung (2005) for the model (with an intercept and a trend) we use.

However, it is well known that the Johansen trace statistics are biased toward rejecting the null hypothesis in small samples. To avoid the Larsson et al. test, as a consequence of this bias, also overestimating the cointegrating rank, we additionally compute the standardized panel trace statistics based on small-sample corrected country-specific trace statistics. Specifically, we use the small-sample correction factor suggested by Reinsel and Ahn (1992) to adjust the individual trace statistics as follows:

$$LR_{iT}\{H(r)|H(p)\} \times \left[\frac{T - k_i \times p}{T}\right], \tag{A.12}$$

where k_i is the lag length of the models used in the test.

The results of these tests are presented in Table A.2. All test statistics indicate that LE_{it} and $Gini_{it}$ are cointegrated (or exhibit a single cointegrating vector).

Table A.2. *Panel cointegration tests*

Panel *v*-statistic	31.45***	
Panel ρ-statistic	-1.91**	
Panel PP-statistic	-4.46***	
Panel ADF-statistic	-7.35***	
Group ρ-statistic	-1.77**	
Group PP-statistic	-3.85***	
Group ADF-statistic	-4.20***	
CIPS statistic for the residuals of the CCE long-run relations	-2.84***	
	Cointegration rank	
	$r = 0$	$r = 1$
Panel trace statistics (unadjusted)	5.51***	-0.31
Panel trace statistics (adjusted)	4.39***	-0.78

Note: *** (**) indicate a rejection of the null hypothesis of no cointegration/no cointegrating vector at the one (five) percent level. The relevant one-percent critical value for the CIPS statistic is -2.36. All other test statistics are asymptotically normally distributed. The right tail of the normal distribution is used to reject the null hypothesis in the panel *v*-statistic and the panel trace statistic, while the left tail is used for the other statistics. One lag was used to form the panel trace and the CIPS statistics. For all other statistics, the number of lags was determined by the Schwarz criterion with a maximum of seven lags.

NOTES

[1] Lynch et al. (2004) review almost 100 studies addressing the question of whether more unequal societies are less healthy. Informative reviews of the relevant literature are also presented by Judge et al. (1998), Wagstaff and van Doorslaer (2000), Deaton (2003), and Subramanian and Kawachi (2004).

[2] In addition to Wilkinson's own extensive work, several prominent studies supported this view, including Rodgers (1979) and Waldmann (1992).

[3] More recently, Anderson and Marmot (2012) used data from the Whitehall II study to show that promotions reduce the probability of developing heart disease. However, the pattern across OECD countries shown by Leigh et al. (2009: Figure 3) indicates that the increase in life expectancy and the decline in infant mortality were more pronounced where inequality widened. Leigh and Jencks (2007) present long-run evidence from a panel of 12 advanced countries; they do not rule out "the possibility that inequality *raises* life expectancy by a substantively significant amount" (page 19).

[4] On the other hand, Deaton (2003: 114) argues that "many of the arguments that income inequality is a health risk are as plausible for poor as for rich countries."

[5] The rich may not even suffer at all from impaired health if the so-called Preston curve becomes a horizontal line once income exceeds a certain threshold.

[6] Deaton (2003: 119) mentions that skepticism exists about the degree to which medical care results in higher life expectancy. We will return to Waldmann's point on the superior-goods character of health care in the context of the relative income hypothesis below.

[7] See Kawachi and Kennedy (1999) for an overview of important concepts and pathways.

[8] On the other hand, Leigh and Jencks (2007: 3) suspect that upward comparisons may be even "soothing if they lead people whose current economic circumstances are stressful to think that their future circumstances could be better."

[9] Sapolsky (2004: 408) provides the example of a low-paid clerk who is the best player in the firm's sports team; "the place in the former hierarchy may be dismissed as 'just a job,' whereas the latter may be emphasized and become a source of considerable self-esteem." Along similar lines, Miller and Paxson (2006) conclude from previous research that subjective social status is a better predictor of psychological stress and health than objective measures of socioeconomic status.

[10] Sapolsky (2004) also notes that humans reduce stress by adjusting the psychological meaning of rank. For instance, an anticipated winner of a tournament tends to suffer more stress and deprivation when failing to win than a novice player surviving just the first rounds. See also Lynch et al. (2004: 18): "The stress effects of dominance hierarchies

seem not even to be generalizable across primate species, let alone generally applicable to the health effects of hierarchical human social organization."

[11] Putnam (1995: 67) defines social capital as "features of social organization such as networks, norms, and social trust that facilitate coordination and cooperation for mutual benefit."

[12] Note that d'Hombres et al. (2010) consider income inequality as an instrumental variable for social capital.

[13] Recall from Deaton (2003) that is also disputed that higher spending on medical care would necessarily result in better health outcomes.

[14] Kawachi and Kennedy (1999: 221) quote Paul Krugman to this effect. Leigh et al. (2009) also note that public spending on health could decline with more heterogeneous preferences of voters. This argument is based on Alesina et al. (1999) who show that the average value of public goods to members of a community diminishes with more pronounced heterogeneity. However, heterogeneity in Alesina et al.'s analysis is mainly linked to ethnic fractionalization, while they do not find robust negative effects of income inequality on public spending (see Deaton, 2003: 131-2).

[15] Most cross-section studies conclude with similar caveats.

[16] For instance, Leigh and Jencks (2007) do not pursue Granger causality tests as they find no statistically significant relationship between inequality and health. Etienne et al. (2007: 19) conclude that "there is still at least one important dimension which needs to be investigated namely the issue of causality."

[17] Available at: http://data.worldbank.org/data-catalog/world-development-indicators.

[18] Available at: http://ghdx.healthmetricsandevaluation.org/global-burden-disease-study-2010-gbd-2010-data-downloads.

[19] The simple correlation coefficient is -0.45, based on 622 observations for 128 countries. Moreover, the coefficient on malnutrition enters negative and highly significant in simple panel regressions with average life expectancy as the dependent variable (details available upon request).

[20] According to the welfare principle, "income transfers among the poor are more consequential than income transfers among the rich" (Firebaugh, 2003: 79). Moreover, the Gini coefficient is "more sensitive to inequality (or to measurement error) at the top of the income distribution" (Deaton, 2003: 135). We are grateful to an anonymous reviewer for having alerted us to this point.

[21] Available at: http://thedata.harvard.edu/dvn/dv/fsolt/faces/study/StudyPage.xhtml?studyId=36908.

[22] Available at: http://people.anu.edu.au/andrew.leigh/.

[23] Data availability does not allow us to conduct a panel data analysis of the effect of the share of the bottom 20 percent on health in developed countries.

[24] Again, the coefficients of GDP per capita and GDP squared have the "wrong" sign. The positive association between GDP per capita and tuberculosis may be due to endogeneity bias if higher morbidity increases future per-capita income by reducing population size.

[25] For the sake of brevity, the results are not shown in detail. They are available from the authors on request.

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Table 1. *Countries in the sample of 19 developed countries and summary statistics, 1976-2010*

	Average of *Gini*	Average of *LE*
Australia	39.17	77.60
Canada	39.50	77.75
Denmark	43.36	75.78
Finland	40.36	76.06
France	42.76	77.25
Germany	42.81	75.99
Hong Kong	47.71	78.40
Ireland	40.97	75.49
Israel	41.87	77.35
Italy	41.99	77.54
Japan	31.93	79.40
Netherlands	37.02	77.43
New Zealand	34.21	76.42
Norway	37.97	77.63
Singapore	43.88	76.22
Sweden	43.06	78.28
Switzerland	39.30	78.37
United Kingdom	43.58	76.44
United States	43.32	75.67
Mean	40.78	77.11
Minimum	27.16 (Japan, 1976)	70.59 (Singapore, 1976)
Maximum	62.41 (Hong Kong, 2002)	85.16 (Israel, 2006)

Table 2. *Countries in the sample of 59 developing countries and summary statistics, 1981-2005*

	Average of *Gini*	Average of *LE*		Average of *Gini*	Average of *LE*
Argentina	44.55	72.26	Malawi∗	57.02	46.55
Azerbaijan	35.58	65.71	Malaysia	49.79	71.29
Bangladesh∗	37.52	61.58	Mauritius	22.93	69.98
Brazil	55.62	67.59	Mexico	47.88	71.72
Bulgaria	27.14	71.52	Morocco	41.46	65.16
Chile	51.46	74.37	Nepal∗	47.18	57.00
China∗	40.24	70.31	Nigeria∗	48.15	46.50
Colombia	50.94	69.19	Pakistan∗	38.71	61.92
Costa Rica	45.43	76.22	Panama	51.41	73.58
Cote d`Ivoire∗	44.44	49.97	Peru	54.67	66.92
Egypt∗	34.96	65.37	Philippines	52.51	65.45
El Salvador	46.87	65.80	Poland	36.93	72.17
Estonia	37.01	69.74	Portugal	45.86	74.76
Ethiopia∗	36.42	48.80	Puerto Rico	51.53	75.10
Fiji	47.88	66.11	Russian Federation	38.92	66.88
Georgia∗	38.78	70.61	Sierra Leone∗	55.76	38.59
Greece	39.23	76.96	South Africa	55.81	58.65
Guatemala	52.87	63.84	Sri Lanka∗	42.25	70.23
Hungary	40.14	70.23	Tajikistan∗	33.20	63.31
India∗	49.36	59.72	Tanzania∗	40.49	50.59
Indonesia	64.10	64.44	Thailand	52.29	69.77
Jordan	48.57	70.23	Trinidad & Tobago	40.36	68.16
Kazakhstan	32.76	66.74	Tunisia	40.15	69.71
Kenya∗	54.91	56.66	Turkey	50.34	66.00
Korea	32.50	72.48	Turkmenistan	35.89	62.99
Kyrgyz Republic	33.50	66.65	Ukraine	32.43	68.69
Latvia	39.37	69.47	Uruguay	47.36	73.18
Lithuania	41.61	70.91	Uzbekistan	34.14	66.67
Madagascar∗	46.48	54.01	Venezuela	43.30	71.27
			Zambia∗	53.17	44.58

	Gini	*LE*
Mean	43.93	65.34
Minimum	19.24 (Mauritius, 2002)	35.79 (Sierra Leone, 1994)
Maximum	79.35 (Indonesia, 2000)	79.24 (Greece, 2005)

Note: Low-income countries (according to World Bank (1995) classification) are marked with an asterisk (∗).

Table 3. *Preliminary results, 1981-2005*

Independent variable	(1)	(2)	(3)	(4)	(5)	(6)
Gini	-0.2411***	-0.1467***	-0.1199***	-0.0122	0.0121	0.0131
	(-10.85)	(-7.57)	(-6.74)	(-1.54)	(1.44)	(1.58)
GDP per capita ($ 1000s)		0.4330***	1.0556***		0.0158	0.2683***
		(33.32)	(30.32)		(0.87)	(6.38)
GDP per capita squared			-0.0158***			-0.0039***
($ 1000s)			(-19.03)			(-6.65)
Year dummies	No	No	No	Yes	Yes	Yes
Country dummies	No	No	No	Yes	Yes	Yes
Countries	80	80	80	80	80	80
Observations	2000	1889	1889	2000	1889	1889

Note: The dependent variable is *LE*. *t*-statistics are in parenthesis. *** indicate significance at the one percent level.

Table 4. *Estimates of the long-run effect of inequality on life expectancy at birth in developed countries, 1976-2010*

Independent variable	(1) Pooled panel DOLS estimator (Mark and Sul, 2003)	(2) Pooled panel DOLS estimator (Mark and Sul, 2003)	(3) Group-mean panel DOLS estimator (Pedroni, 2001)	(4) Pooled panel FMOLS estimator (Phillips and Moon, 1999)
Gini	0.0113*	0.0381***	0.0407***	0.0274***
	(1.73)	(5.92)	(3.99)	(3.18)
Demeaned data	No	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes	Yes
Individual trends	Yes	Yes	Yes	Yes
Countries	19	19	19	19
Observations	608	608	608	646

Note: The dependent variable is *LE*. *t*-statistics are in parenthesis. *** (*) indicate significance at the one (ten) percent level. The DOLS results are based on a one lead/lag model.

Table 5. *DOLS estimates for five-year averages, for a larger sample of countries, and for specifications with control variables*

Independent variable	(1) 5-year averages	(2) Larger sample over a shorter time period	(3) With GDP p.c. and GDP p.c. squared	(4) With GDP p.c. and GDP p.c. squared
Gini	0.0463***	0.0230***	0.0172**	0.0585***
	(10.48)	(3.37)	(2.12)	(4.09)
GDP per capita ($ 1000s)			-0.2047***	0.2102***
			(-5.94)	(6.83)
GDP per capita squared ($ 1000s)			0.0027***	-0.0031***
			(6.12)	(-9.86)
Demeaned data	Yes	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes	Yes
Individual trends	Yes	Yes	Yes	No
Sample period	1976-2010	1981-2005	1976-2010	1976-2010
Countries	19	21	16	16
Observations	95	462	512	512

Note: The dependent variable is *LE*. *t*-statistics are in parenthesis. *** (**) indicate significance at the one (five) percent level. Given the very small number of time series observations, the DOLS result in column 1 is based on a model with one lag and no leads; all other results are based on equations with one lead and one lag. The estimates in column 2 are based on a sample that includes the 19 countries of our main sample plus Belgium and Spain. For Ireland, New Zealand, and Switzerland, complete time series data on real GDP per capita are not available for the period 1976-2010. Therefore, these countries are excluded from the sample used to estimate the specifications in columns 3 and 4.

Table 6. *DOLS estimates using different measures of inequality and health*

Independent variable	(1) Different inequality measure [Dependent variable: *LE*]	(2) Different health measure [Dependent variable: *IMR*]	(3) Different inequality and different health measure [Dependent variable: *IMR*]
TopDecile			
	0.1032***		-0.2076***
	(4.90)		(-3.30)
		-0.0679***	
Gini		(-3.97)	
Demeaned data	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes
Individual trends	Yes	Yes	Yes
Sample period	1961-1996	1976-2010	1961-1996
Countries	8	18	8
Observations	264	576	264

Note: t-statistics are in parenthesis. *** indicate significance at the one percent level. The DOLS results are based on a one lead/lag model. For Hong Kong, complete time series data on infant mortality are not available for the period 1976-2010. Therefore, Hong Kong is not included in the sample used to estimate the coefficient in column 2.

Table 7. *Estimates of the long-run effect of inequality on tuberculosis incidence in developed countries, 1990-2011*

Exogenous variable	(1)	(2)	(3)
Gini	-0.4692***	-0.5253***	-0.5004***
	(0.0971)	(0.0860)	(0.0817)
GDP per capita ($ 1000s)		0.6948***	2.9457***
		(0.1150)	(0.7664)
GDP per capita squared			-0.0166***
($ 1000s)			(0.0056)
Year dummies	Yes	Yes	Yes
Country dummies	Yes	Yes	Yes
Countries	30	30	30
Observations	547	537	537

Note: Standard errors (calculated by the Delta method) are in parentheses. *** indicate significance at the one percent level. The effects were estimated from an autoregressive distributed lag model with one lag on the endogenous variable and one lag on the exogenous variable(s).

Table 8. *DOLS estimates of the long-run effect of inequality on life expectancy at birth and in developing countries, 1981-2005*

Independent variable	(1) Dependent variable: *LE*	(2) Dependent variable: *IMR*
Gini	-0.0680***	0.1173***
	(-10.82)	(8.62)
Demeaned data	Yes	Yes
Fixed effects	Yes	Yes
Individual trends	Yes	Yes
Countries	59	56
Observations	1298	1232

Note: t-statistics are in parenthesis. *** indicate significance at the one percent level. The DOLS results are based on a one lead/lag model. For Azerbaijan, Georgia, and Russia, complete time series data on infant mortality are not available for the period 1976-2010. Therefore, these countries are not included in the sample used to estimate the coefficient in column 2.

Table 9. *Long-run-estimates for developing countries using the income share of the bottom quintile of the population as the inequality measure, 1983-2011*

Exogenous variable	(1)	(2)	(3)
BottomQuintile	0.4091*	0.4082**	0.5086**
	(0.0538)	(0.1974)	(0.2481)
GDP per capita ($ 1000s)		-0.3345	0.0372
		(0.3012)	(1.0622)
GDP per capita squared ($ 1000s)			-0.0515
			(0.0865)
Year dummies	Yes	Yes	Yes
Country dummies	Yes	Yes	Yes
Countries	32	32	32
Observations	230	226	226

Note: The dependent variable is *LE*. Standard errors (calculated by the Delta method) are in parentheses. ** (*) indicate significance at the five (ten) percent level. The long-run effects were estimated from an autoregressive distributed lag model with two lags on the endogenous variable and two lags on the exogenous variable(s), given the relatively long period covered in the data.

Figure 1. DOLS estimation with single country excluded from the sample