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Abstract:
This paper argues that previous cross-country (panel) studies on the relationship between income inequality and health suffer from significant biases due to (i) omitted country-specific factors, (ii) endogeneity, and (iii) cross-country heterogeneity in the impact of inequality on health. Using panel cointegration techniques that are robust to omitted variables, endogenous regressors, and slope heterogeneity, we find that income inequality has, on average, a small, but robust and statistically significant positive impact on population health. Also, there is some evidence that inequality is endogenous in the sense that poor health leads to increased income inequality. Finally, we find that there are large cross-country differences in the effect of income inequality on health (in about 35 percent of the cases, the effect is negative).

Keywords: Health; Inequality; Panel cointegration

JEL classification: I14; C23

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1. Introduction

There is a large literature on the health effects of income inequality. Lynch et al. (2004) review almost 100 studies.[1] Nevertheless, it is still open to question whether more unequal societies are less healthy. The predominant view appears to have shifted over time. Earlier studies led Wilkinson (1996: 4) to conclude that 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." In addition to Wilkinson's own extensive work, several prominent studies supported this view, including Rodgers (1979) and Waldmann (1992). 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."

More recent studies draw on more data and typically go beyond pure cross-sections for one particular point in time. They largely fail to achieve similarly strong results. Once other country, local or personal socio-economic characteristics — such as average income, wealth, education or indicators of social capital — are controlled for, the association between income inequality and health turns out to be elusive and mostly insignificant. This suggests, according to Deaton (2003: 115), that "individuals are no more likely to be sick or to die if they live in places or in periods where income inequality is higher."[2]

Several analyses even find higher income inequality to be associated with better health. Mellor and Milyo (2001) report striking reversals in the sign of the income-inequality variable once education is controlled for in samples of 12 to 47 countries, and also across US states; a higher Gini coefficient, revealing a more unequal income distribution, then has a significantly negative effect on infant mortality and a significantly positive effect on life expectancy.[3] Moreover, increases in Gini coefficients over time tend to be associated with increases in life expectancy in the study of Mellor and Milyo. Likewise, 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; the sign of their inequality measure, the income share of the richest

[1] 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] Similarly, Lynch et al. (2004: 19-20) conclude that earlier claims have become "more circumspect" as research in the field has matured. Subramanian and Kawachi (2004: 89) stress the limitations of cross-section studies; they point to several confounding factors and omitted variables, summarizing that "income-based inequality is, at best, simply one dimension that could be relevant to population health."

[3] According to Miller and Paxson (2006), some demographic and age groups in the United States (in particular working-age black males) benefited in terms of lower mortality from living together with richer neighbors. Lorant et al. (2001) also find that mortality decreased with higher (municipal) Gini coefficients in Belgium.

decile of the population, tends to switch in the preferred fixed-effects specifications. Hence, Leigh and Jencks (2007: 19) do not rule out "the possibility that inequality *raises* life expectancy by a substantively significant amount."

While previous studies reveal strikingly different results, most of them 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. Subramanian and Kawachi (2004) stress endogeneity problems as a major challenge for research on inequality and health. Deaton (2003) and Leigh et al. (2009) discuss the possibility of reverse causation from income inequality to health, but existing empirical research typically fails to establish causal links. Furthermore, earlier studies are highly likely to suffer from omitted variable bias by ignoring socio-economic factors which may drive inequality and health simultaneously. On the other hand, more recent multivariate analyses face a dilemma by controlling for factors through which inequality may affect health. Controlling for educational attainment, for instance, has been criticized by Kawachi et al. (1997: 1497) as "statistical overadjustment" which could deprive inequality of any visible impact on health.[4]

The panel cointegration approach pursued in the following addresses endogeneity concerns head-on. We explicitly account for reverse causality. Moreover, the techniques applied are robust to omitted variables while allowing income inequality to possibly affect health outcomes through different channels. 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 sample of 35 countries over the period 1970-1995, and Section 5 concludes. We find that income inequality has, on average, a small, but robust and significantly positive impact on health outcomes. At the same time, there is some, though weak, evidence for reverse causality. Our findings also indicate considerable heterogeneity in the effects of income inequality on health across countries.

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,

[4] See also Lynch et al. (2004) for a discussion of confounding versus intermediating variables.

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

[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.

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., 2011).[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

[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."

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

[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).

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 model and data

The objective is to analyze the impact of income inequality on health using heterogeneous panel cointegration techniques. In this section, we present the basic empirical model and discuss some econometric issues (Section 3.1). Then, we describe the data and report some descriptive statistics (Section 3.2).

3.1. Empirical specification and econometric issues

Following common practice in panel cointegration studies, we consider a bivariate relationship of the form

[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."

$$LE_{it} = a_i + \delta_i t + bInequality_{it} + \varepsilon_{it}\,, \tag{1}$$

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 $Inequality_{it}$ stands for the estimated household income inequality (EHII) in Gini format. Following previous inequality-health studies (see, e.g., 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 income inequality by one unit. Moreover, we include country-specific fixed effects, a_i, and country-specific deterministic time trends, $\delta_i t$, to control for any omitted factors that are relatively stable over time or evolve relatively smoothly over time.

Given that both life expectancy at birth and income inequality have increased significantly in recent decades, it is reasonable to assume that these two variables are permanent, non-stationary 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 $Inequality_{it}$. Otherwise, there is no long-run relationship between life expectancy at birth and income inequality; Equation (1) would in this case represent a spurious regression in the sense of Granger and Newbold (1974).[17] Thus, the necessary conditions for our model to be a correct description of the data are that LE_{it} and $Inequality_{it}$ are non-stationary or, more specifically, integrated of the same order, and cointegrated.

A regression containing all the variables of a cointegrating vector has a stationary error term, ε_{it}, implying that no relevant integrated variables are omitted; any omitted non-stationary variable that is part of the cointegrating relationship would enter the error term, thereby producing non-stationary residuals and thus a failure to detect cointegration. If, on the other hand, a cointegrating relationship exists among a set of non-stationary variables, the same stationary (cointegrating) relationship also exists in extended variable space (Johansen, 2000). Thus, an important implication of finding cointegration is that no relevant integrated variables are omitted in the cointegrating regression. Cointegration estimators are therefore robust to the omission of non-stationary variables that do not form part of the cointegrating relationship (Pedroni, 2007).

Of course, there are several factors (such as income, education, employment, housing and the environment) that affect the health of the population. Adding further variables may therefore result in further cointegrating relationships. Since the cointegration property is invariant to

[17] The spurious regression problem can also arise in panels when dealing with non-stationary variables. 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.

extensions of the information set, the inclusion of additional variables would, however, not "destroy" the original cointegrating relationship. This justifies considering small subsystems, such as Equation (1) (if the variables are cointegrated).

The bivariate nature of the model guarantees in addition that the coefficient *b* captures the total effect of income inequality on life expectancy at birth. In a multivariate model, in contrast, the estimate of inequality's impact on life expectancy would leave out any effects operating through the impact on the other included variables. Suppose, for instance, that inequality reduces growth, as several studies suggest (see, e.g., Clarke, 1995; Chamber and Krause, 2010; Herzer and Vollmer, 2011), then by including per capita income in the regression, we would be omitting the negative health effect of inequality that operates via income growth.

Another important feature of Equation (1) is the implicit assumption that life expectancy is endogenous in the sense that, in the long run, changes in inequality cause changes in life expectancy. However, although cointegration implies that long-run Granger causality must run in at least one direction (Granger, 1988), causality may also run from health (and thus life expectancy) to income inequality. As discussed in Section 2, there are several ways in which poor health may increase income inequality, implying that simple OLS estimates of the effect of income inequality on health are very likely to be negatively biased. The empirical implication is that it is crucial to not only examine the time-series properties of the variables and test whether the variables are cointegrated, but also to deal with this endogeneity problem and investigate the direction of causality.

Another important issue is the potential cross-country heterogeneity in the relationship between inequality and health. Countries differ in terms of per capita income, economic, political, and social structures (and other characteristics). The implicit assumption of traditional, homogeneous panel estimators that the coefficients on the variables of interest are the same across all countries can therefore be unduly restrictive. The problem is that homogeneous (within-dimension) estimators may produce inconsistent and potentially misleading estimates in the presence of slope heterogeneity (Pesaran and Smith, 1995). For this reason, we use heterogeneous (between-dimension) panel estimators based on the mean group approach. This allows us, in addition, to explicitly analyze the potential heterogeneity in the effects of inequality across countries.

A final econometric issue is the potential 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 effect of income inequality on health status using both our raw data and demeaned data; in place of LE_{it} and $Inequality_{it}$, we thus also use $LE_{it}' = LE_{it} - \overline{LE_t}$ and $Inequality_{it}' = Inequality_{it} - \overline{Inequality_t}$, where $\overline{LE_t} = N^{-1}\sum_{i=1}^{N} LE_{it}$ and $\overline{Inequality_t} = N^{-1}\sum_{i=1}^{N} Inequality_{it}$ are the cross section means of the variables.

3.2. Data and descriptive statistics

The data on life expectancy at birth are from the World Development Indicators Online database. 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. We therefore prefer LE_{it}, but we will also use alternative measures of health status, such as the infant mortality rate, the under-five mortality rate, and the crude death rate, to examine the robustness of our conclusions.

As far as data on income inequality are concerned, several studies have used the Gini coefficient dataset 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 dataset, 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 (Galbraith, 2009).

In this study, we use the estimated household income inequality (EHII) dataset developed by the University of Texas Inequality Project (UTIP).[18] The EHII data are fully comparable across space and time (Galbraith and Kum, 2005). Another advantage is that they are available for a reasonably large number of countries over a sufficiently long and continuous time period.

The EHII index is in Gini format (measured on a 0 to 100 scale). It is estimated by combining information from the Deininger-Squire dataset with information from the UTIP-UNIDO

[18] Available at http://utip.gov.utexas.edu/data.html.

dataset. The latter is a set of measures of manufacturing wage inequality, using the between-groups component of a Theil index, measured across industrial categories in the manufacturing sector based on the Industrial Statistics database of the United Nations Industrial Development Organization (UNIDO). Specifically, the EHII index is constructed by regressing the Deininger-Squire Gini indices on the UTIP-UNIDO Theil inequality measures (and on several control variables), and then using the predicted values as estimated Gini coefficients. The intention of this procedure is to separate the useful from the doubtful information in the Deininger-Squire dataset (Galbraith and Kum, 2005).

Although many of the more recent income inequality studies use the EHII Gini coefficient (Meschi and Vivarelli, 2009; Herzer and Vollmer, 2011; Gimet and Lagoarde-Segot, 2011), this index has the limitation that it is estimated, and estimates may be biased (for several reasons). Therefore, we will check the sensitivity of the results to the measure of inequality by using the top-decile income share data provided by Leigh (2007).[19] Leigh adjusts top incomes series from different studies to produce a comparable dataset. However, these data are available only for a small number of high-income countries, so that the EHII Gini is our preferred measure of income inequality. Table 1 contains a list of the main variables and their definitions.

Table 1

Variables and sources

Variable	Definition	Source
LE_{it}	Live expectancy at birth is 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.	World Development Indicators Online http://data.worldbank.org (accessed August 22, 2011)
$\log(IMR_{it})$	Log of the infant mortality rate. The infant mortality rate is the probability (expressed as a rate per 1,000 live births) of a child born in a specified year dying before reaching the age of one if subject to current age-specific mortality rates.	Child Mortality Database, http://www.childmortality.org (accessed August 22, 2011)
$\log(U5MR_{it})$	Log of the under-five mortality rate. The under-five mortality rate is the probability (per 1,000) that a newborn baby will die before reaching age five, if subject to current age-specific mortality rates.	Child Mortality Database, http://www.childmortality.org (accessed August 22, 2011)
$\log(CR_{it})$	Log of the crude death rate. The crude death rate indicates the number of deaths (per 1,000) occurring during the year.	World Development Indicators Online http://data.worldbank.org (accessed August 22, 2011)
$Inequality_{it}$	Estimated household income inequality (EHII) in Gini format, measured on a 0 to 100 scale.	University of Texas Inequality Project http://utip.gov.utexas.edu/data.html (accessed August 22, 2011)
$TopDecile_{it}$	Percentage income share of the richest 10% of the population.	Leigh (2007) http://people.anu.edu.au/andrew.leigh (accessed August 22, 2011)

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

In our main analysis, we will focus on the cointegrating relationship between LE_{it} and $Inequality_{it}$. The identification and estimation of cointegrating relationships requires the use of continuous data over a sufficiently long period of time. Since panel cointegration methods can be implemented with shorter data spans than their time-series counterparts (due to exploitation of both the time-series and cross-sectional dimensions of the data), a period of about 25 observations should be more than sufficient for our purpose; several cointegration analyses for individual countries are based on shorter periods (see, e.g., de Crombrugghe et al., 1997; Herzer, 2010). We include all countries for which continuous data are available over a sufficiently long period of time, resulting in a balanced panel of 910 observations on 35 countries over the period 1970-1995 (26 years).

Table 2

Countries and country summary statistics

Country	Average of LE_{it}	Average of $Inequality_{it}$	Country	Average of LE_{it}	Average of $Inequality_{it}$
Austria	73.27	34.63	Italy	74.81	35.14
Barbados	72.35	43.63	Japan	76.77	36.63
Bolivia	53.61	45.24	Kenya	57.35	47.32
Bulgaria	71.32	29.31	Kuwait	71.76	51.87
Canada	75.53	35.99	Malaysia	67.26	40.72
Chile	69.63	43.95	Mexico	67.42	40.56
Colombia	65.83	42.67	Netherlands	75.79	32.51
Cyprus	74.66	40.66	New Zealand	73.79	34.99
Denmark	74.41	30.94	Norway	75.84	32.18
Ecuador	64.48	42.77	Philippines	62.23	45.46
Egypt	58.14	40.11	Poland	70.81	29.10
Finland	73.54	31.26	Spain	75.35	37.84
Hungary	69.36	29.52	Sweden	76.37	27.54
India	55.32	42.94	Syria	63.90	43.05
Indonesia	56.23	44.28	Turkey	61.39	42.91
Iran	60.36	38.72	United Kingdom	74.30	29.69
Israel	74.29	40.36	United States	73.84	37.19
			Zimbabwe	58.34	43.66

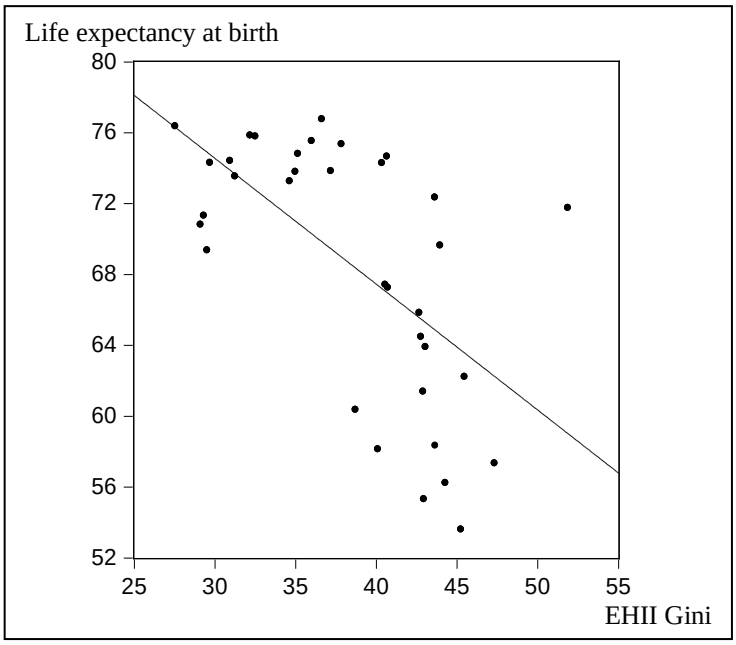
The countries along with the average values for LE_{it} and $Inequality_{it}$ over the period of observation are listed in Table 2. As can be seen, there are considerable differences in the values of these variables across countries. Japan had the highest life expectancy, followed by Sweden, Norway, and the Netherlands; the country with lowest life expectancy in this sample was Bolivia, followed by India, Indonesia, and Kenya. Kuwait was the country with the highest inequality, followed by Kenya, the Philippines, Bolivia, and Indonesia, while Sweden ranked at the bottom of

the inequality scale. Altogether, it appears that countries with lower inequality values tend to have higher live expectancies.

The cross-country relationship between these two variables is depicted graphically in Figure 1. It shows the scatter plot of the period-average of life expectancy at birth versus the period-average of the EHII Gini for the 35 countries in our sample along with the regression line; its slope is strongly negative and statistically highly significant with a *t*-statistic of -4.38. Such correlations have been interpreted by many as support for the hypothesis that inequality has a strong negative effect on health (see, e.g., Wilkinson, 1992; Kaplan et al., 1996). In the next section, we examine this relationship in more detail using heterogeneous panel cointegration techniques to control for omitted variables, endogeneity, and slope heterogeneity.

Figure 1

Scatter plot of life expectancy at birth versus income inequality



4. Empirical analysis

Our pre-tests for unit roots and cointegration suggest that LE_{it} and $Inequality_{it}$ are non-stationary and cointegrated, as assumed in Equation (1). The results of these tests are presented in the Appendix. In this section, we provide estimates of the cointegrating relationship between life expectancy and income inequality (Section 4.1) and test the robustness of our results (Section 4.2). We also discuss possible reasons for the discrepancy between our results and those in the literature, including omitted country-specific fixed effects (Section 4.3), reverse causality (Section 4.4), and cross-country differences in the effects of inequality on health (Section 4.4).

4.1. The long-run relationship between inequality and health

We estimate the long-run effect of LE_{it} on $Inequality_{it}$ using the between-dimension group-mean panel dynamic ordinary least squares (DOLS) estimator that Pedroni (2001) argues has a number of advantages over the within-dimension approach. First, it allows for greater flexibility in the presence of heterogeneous cointegrating vectors, whereas under the within-dimension approach, the cointegrating vectors are constrained to being the same for each country. Another advantage of the between-dimension estimators is that the point estimates provide a more useful interpretation in the case of heterogeneous cointegrating vectors; they can be interpreted as the mean value of the cointegrating vectors, which is not appropriate for the within estimators. Finally, the between-dimension estimators suffer from much lower small-sample-size distortions than is the case with the within-dimension estimators.

The DOLS regression is given by

$$LE_{it} = a_i + \delta_i t + b_i Inequality_{it} + \sum_{j=-p_i}^{p_i} \Phi_{ij} \Delta Inequality_{it-j} + \varepsilon_{it}, \tag{2}$$

where Φ_{ij} are coefficients of lead and lag differences, which account for possible serial correlation and endogeneity of the regressor(s), thus yielding unbiased estimates. Thus, an important feature of the DOLS procedure is that it generates unbiased estimates for variables that cointegrate, even with endogenous regressors. In addition, the DOLS estimator is superconsistent under cointegration, and it is also robust to the omission of variables that do not form part of the cointegrating relationship.

From regression (2), the group-mean DOLS estimator for *b* is constructed as

$$\hat{b} = \left[N^{-1} \sum_{i=1}^{N} \left(\sum_{t=1}^{T} z_{it} z'_{it} \right)^{-1} \left(\sum_{t=1}^{T} z_{it} \tilde{s}'_{it} \right) \right]_1, \tag{3}$$

where z_{it} is the $2(K+1)\times 1$ vector of regressors z_{it} = [$Inequality_{it} - \overline{Inequality_i}$, $\Delta Inequality_{it-K}$, …, $\Delta Inequality_{it+K}$], $\tilde{s}_{it} = s_{it} - \bar{s}_i$, and the subscript 1 outside the brackets indicates that only the first element of the vector is taken to obtain the pooled slope coefficient. Because the expression following the summation over the *i* is identical to the conventional time-series DOLS estimator, the between-dimension estimator for *b* is calculated as

$$\hat{b} = N^{-1} \sum_{i=1}^{N} \hat{b}_i, \tag{4}$$

where $\hat{b}_i$ is the conventional DOLS estimator applied to the *i*th country of the panel and $t_{\hat{b}} = N^{-1/2} \sum_{i=1}^{N} t_{\hat{b}_i}$ is the associated *t*-statistic. According to Stock and Watson (1993), this estimator performs well in short time series compared to other cointegration estimators such as the maximum

likelihood estimator of Johansen (1988), or the fully modified ordinary least squares (FMOLS) estimator of Phillips and Hansen (1990).

Table 3

Estimates of the long-run effect of inequality on life expectancy at birth

Independent variable	Pooled OLS	Fixed-effects OLS	Group-mean DOLS Raw data	Group-mean DOLS Demeaned data	Group-mean FMOLS Demeaned data
	(1)	(2)	(3)	(4)	(5)
$Inequality_{it}$	-0.558**	-0.016	0.039*	0.017**	0.024**
	(-16.85)	(-0.82)	(2.36)	(5.19)	(4.28)
Countries	35	35	35	35	35
Observations	910	910	805	805	875

Notes: The observation period is 1970-1995. The dependent variable is LE_{it}. The fixed-effects OLS regression includes country and year dummies. The DOLS results are based on a one lead/lag model, as suggested by the usual information criteria. *t*-statistics are in parenthesis. ** (*) indicate significance at the 1% (5%) level. *t*-statistics in parentheses.

However, before estimating the coefficient *b* by DOLS, we are interested in whether our data can be used to replicate some of the previous findings reported in the literature. We first run a pooled regression, which does not account for omitted variables, potential cross-sectional dependence, endogeneity, and slope heterogeneity. The result of this exercise is reported in column (1) of Table 3. As can be seen, the pooled OLS estimate suggests a strong negative and statistically significant effect of inequality on life expectancy, which is consistent with previous cross-sectional studies (and also with the correlation pattern in Figure 1).

Next, we present (in column (2) of Table 3) the result of a fixed-effects regression that includes both country and year dummies to control for time-invariant omitted-variable bias and cross-sectional dependence in the data. In line with previous fixed-effects models such as Leigh and Jencks (2007), the estimated coefficient turns out to be insignificant.

The group-mean DOLS estimates of the effect of income inequality on life expectancy are given in columns (3) and (4). We present results for the raw data, as well as for the data that have been demeaned with respect to the cross-sectional dimension to account for the possible cross-sectional dependence through common time effects.[20] After additionally controlling for the potential endogeneity of inequality and the possible heterogeneity in the slope coefficients via the group-mean DOLS estimator, both the raw and demeaned data suggest that the effect of income inequality on life expectancy is statistically significant and *positive*. According to the DOLS regression with demeaned data, an increase in the EHII Gini coefficient by one unit leads to an increase in life expectancy of 0.017 years — admittedly, a marginal effect. Since the EHII Gini

[20] The cross-sectional correlation induced by a common time effect can be handled by using cross-sectionally demeaned data or by including time dummies.

increased on average from 38.11 in 1970 to 41.43 in 1995, this estimate implies that life expectancy increased by about 21 days due to the increase in income inequality in this period. In fact, life expectancy increased between 1970 and 1995 by about 7.6 years on average in our sample, suggesting that the increase in income inequality was responsible for only about 0.5 percent of the increase in life expectancy in the period of observation.

4.2. Robustness

A major contribution of this paper is the use of estimation techniques that are robust (under cointegration) to a variety of estimation problems that often plague empirical work, including omitted variables, endogeneity, and slope heterogeneity. Hence, we need to ensure that the differences in the results between the present and previous studies are due exclusively to the estimation method, rather than to other factors, such as outliers, sample selection, and different indicators of health status and income inequality. We therefore perform several robustness checks.

To verify that the positive effect of inequality on health is not due to individual outliers, the DOLS regression (with demeaned data)[21] is re-estimated excluding one country at a time from the sample. The sequentially estimated coefficients and their *t*-statistics are presented in Figure 2. They fluctuate between 0.005 (due to the exclusion of Colombia) and 0.049 (due to the exclusion of Zimbabwe)[22] and are always significant at the one percent level, suggesting that the positive effect of inequality on health is not the result of outliers.

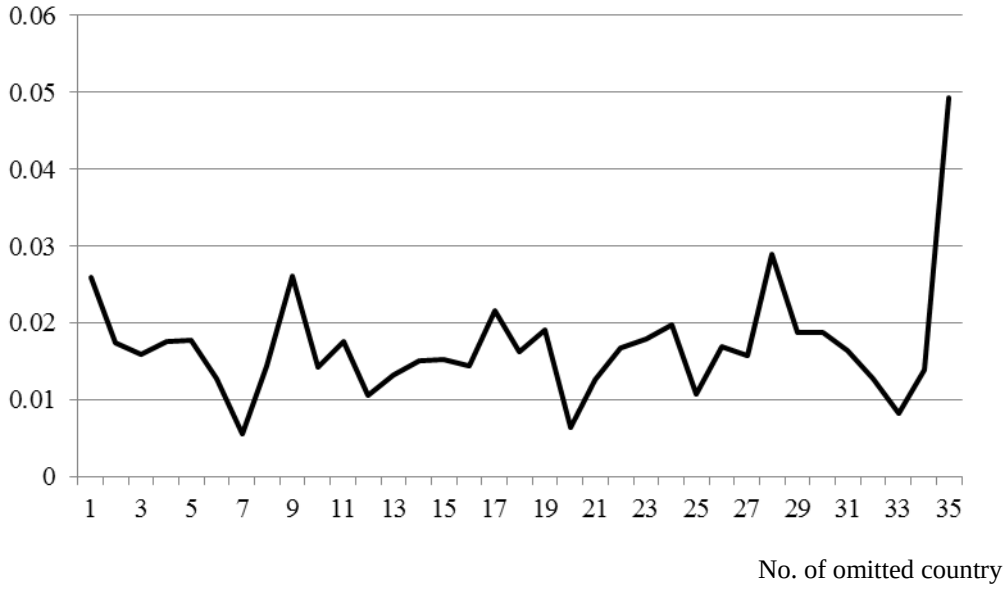
The positive long-run relationship between inequality and life expectancy may be due to sample-selection bias if a group of countries of a certain development level has a significant effect on the results. To examine this, the DOLS regression is re-estimated excluding low-income or, alternatively, high-income countries. The resulting group-mean values for *b* are reported in Table 4. Regardless which of these country groups is excluded from the sample, the long-run relationship between income inequality and life expectancy remains positive and highly significant.[23] Clearly, it would be desirable to also assess whether there are significant differences in the effects of inequality on health between low-income and high-income countries. However, the small sample sizes do not allow statistically meaningful comparisons in this regard.

[21] In the following, we use the demeaned data to account for the likely cross-sectional dependence through common time effects.
[22] The individual country DOLS estimates are presented in Figure 5.
[23] This finding is largely in line with Gravelle et al. (2002), whose results do not suggest that the relationship between income inequality and life expectancy differs between high- and low-income countries.

Figure 2

DOLS estimation with single country excluded from the sample

Coefficients on $Inequality_{it}$



t-statistics of the coefficients

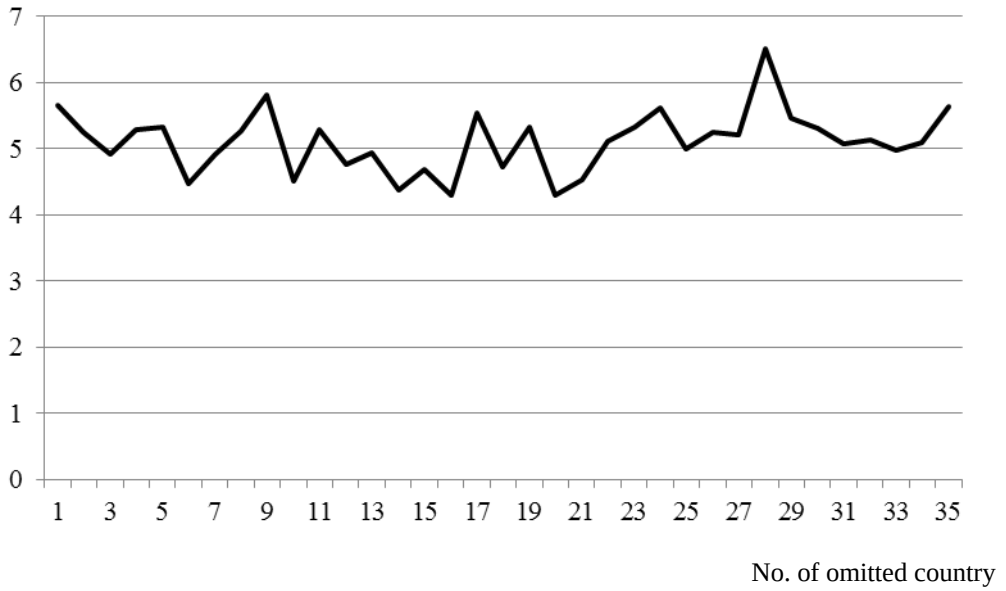


Table 4

DOLS estimation with low-income or high-income countries excluded from the sample

	$Inequality_{it}$	Number of countries in the subsample	Observations
Excluding low-income countries	0.038** (3.91)	32	763
Excluding high-income countries	0.015** (6.29)	19	437

Notes: The DOLS results are based on a one lead/lag model, as suggested by the usual information criteria. The observation period is 1970-1995. The World Bank (1995) classifies 3 countries in 1995 as low-income countries (India, Kenya, and Zimbabwe), while 16 countries are classified as high-income countries (Austria, Canada, Cyprus, Denmark, Finland, Israel, Italy, Japan, Kuwait, Netherlands, New Zealand, Norway, Spain, Sweden, United Kingdom, United States). ** indicate significance at the 1% level. *t*-statistics in parentheses.

We also examine whether our results are robust to alternative measures of health status. More specifically, we replace in Equation (2) life expectancy at birth by the infant mortality rate, log(IMR_{it}), the under-five mortality rate, log($U5MR_{it}$), and the crude death rate, log(CR_{it}), all in logs (as is common practice in the literature). Since the data for these variables cover different countries, we (again) use different country samples. The countries in the samples are listed in the Appendix. Table 5 presents the results. The coefficients on all mortality variables are negative and statistically significant at conventional levels, suggesting that inequality leads to a decrease in mortality. This is consistent with the result that income inequality has a positive effect on life expectancy at birth, or more generally on health.

Table 5

DOLS estimates using different measures of health status

	log(IMR_{it}) (1)	log($U5MR_{it}$) (2)	log(CR_{it}) (3)
$Inequality_{it}$	-0.0045*	-0.0045*	-0.0035**
	(-2.64)	(-2.65)	(-5.04)
Countries	43	43	44
Observations	989	989	1012

Notes: The DOLS results are based on a one lead/lag model, as suggested by the usual information criteria. The observation period is 1970-1995. ** (*) indicate significance at the 1% (5%) level. *t*-statistics in parenthesis.

Next, we test the sensitivity of our results to the measure of income inequality. Leigh and Jencks (2007) use top income share data to examine the effect of income inequality on health for 12 high-income countries in an unbalanced panel between 1903 and 2003. Here we employ continuous data on top income shares for 8 (or 9) high-income countries over the period from 1961 to 1996. Regrettably, balanced data for more countries are not available for a sufficiently long time period, forcing us to limit our sample to these 8 (9) high-income countries (listed in the Appendix). We first re-estimate the original model (with life expectancy at birth as the dependent variable) using the income share of the top decile (labeled $TopDecile_{it}$) as our measure of inequality. The result is reported in column (1) of Table 6. In columns (2)-(4) we present the results from DOLS regressions of $TopDecile_{it}$ on log(IMR_{it}), log($U5MR_{it}$), and log(CR_{it}). All estimates suggest that income inequality increases health. Moreover, the fact that the estimated coefficients for the period from 1961 to 1996 have the same signs as their counterparts in Tables 3 and 5 (for the period 1970-1995) suggests that our results are not sensitive to the sample period.

Table 6

DOLS estimates using different measures of inequality and health status

Regressand Regressor	LE_{it} $TopDecile_{it}$ (1)	$\log(IMR_{it})$ $TopDecile_{it}$ (2)	$\log(U5MR_{it})$ $TopDecile_{it}$ (3)	$\log(CR_{it})$ $TopDecile_{it}$ (4)
Coefficient of the regressor	0.065** (4.25)	-0.014* (-2.18)	-0.013* (-2.25)	-0.007** (-5.60)
Countries	8	9	9	9
Observations	280	315	315	315

Notes: The DOLS regressions were estimated with one lead and one lag. The observation period is 1961-1996. ** (*) indicate significance at the 1% (5%) level. *t*-statistics in parenthesis.

As a final sensitivity test, we employ an alternative cointegration estimator. Specifically, we use the group-mean FMOLS estimator of Pedroni (2001). While the DOLS estimator employs a parametric correction for the endogeneity achieved by augmenting Equation (1) with leads, lags, and contemporaneous values of the differenced $I(1)$ regressors, the FMOLS estimator employs a non-parametric correction to eliminate bias stemming from endogeneity using ε_{it} and $\Delta Inequality_{it}$. Like the group-mean DOLS estimator, the group-mean FMOLS estimator allows the slope coefficients to vary across countries. The FMOLS estimate of the effect of inequality, measured by the EHII Gini, on life expectancy is reported in column (5) of Table 3. Once again, the estimated coefficient is positive and statistically significant. Thus, it can be concluded that the positive effect of inequality on health is robust to potential outliers, sample selection, different measures of health and inequality, and alternative estimation techniques.

4.3. Omitted country-specific effects

Why do we find a robust positive association between inequality and health, while various previous studies suggest that inequality has a negative or no effect on health? One answer is that there are country-specific relatively time-invariant factors — such as norms and institutions — that have a *positive* effect on health and a *negative* effect on inequality. If these factors are omitted from the regression, health will be negatively correlated with the error term, and thus the estimated coefficient on inequality will be downward biased.

We control for such factors through fixed effects, a_i. Because the country fixed effects are part of our health function and, by definition, do not vary over time, they should be correlated with the live expectancy levels of the countries in all 26 periods of our sample. Figure 3 confirms this expectation. It shows the scatter plots of the relationship between live expectancy and the estimated fixed effects (from the group-mean DOLS regression with demeaned data) for the years from 1970

to 1995. The relationship between LE_{it} and $\hat{a}_i$ is always strong and positive, with highly significant correlation coefficients ranging from 0.972 (1970) to 0.776 (1995).

Figure 3

Scatter plots of the relationship between live expectancy and estimated fixed effects for the years from 1970 to 1995

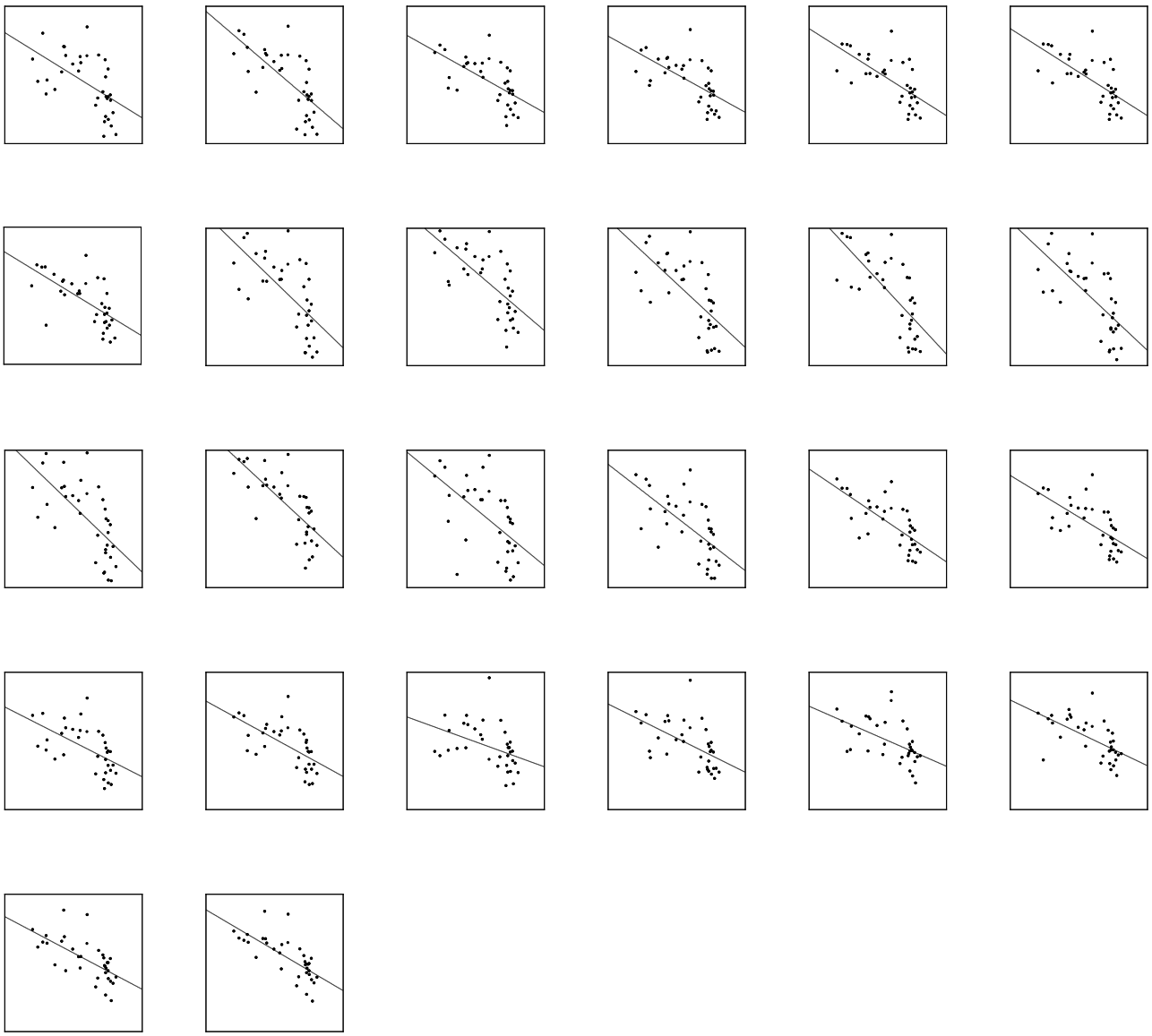
Notes: Vertical axes: life expectancy; horizontal axes: estimated fixed effects. The panels show the scatter plots of life expectancy at birth versus the estimated country-specific fixed effects for the years from 1970 to 1995 (in ascending order from the left to the right).

We now turn to the relationship between the EHII Gini and the estimated country fixed effects. The corresponding scatter plots are shown in Figure 4. We find a strongly negative relationship between inequality and the unobserved time-invariant determinants of health in all periods; the statistically significant correlation coefficients range from -0.412 (1990) to -0.736 (1983). It can be concluded that at least some of the negative correlation found in many studies

between inequality and health is due to omitted unobserved country characteristics. This corroborates Leigh and Jencks (2007) where negative health effects of income equality were no longer observed after accounting for country fixed effects.

Figure 4

Scatter plots of the relationship between inequality and estimated fixed effects for the years from 1970 to 1995



Notes: Vertical axes: EHII Gini; horizontal axes: estimated fixed effects. The panels show the scatter plots of the estimated household inequality versus the estimated country-specific fixed effects for the years from 1970 to 1995 (in ascending order from the left to the right).

4.4. Causality

The negative correlation between income inequality and health found in other studies could also, at least in part, reflect reverse causality from poor health to wider income disparity rather than a negative effect of income inequality on health.

We therefore ask whether the health level helps predict the level of income inequality and vice versa. Table 7 investigates this question using Granger causality tests based on a fixed-effects levels VAR. As shown by Lütkepohl and Reimers (1992), Wald tests for Granger causality in cointegrated bivariate VARs are asymptotically distributed as chi-square. We report the *p*-values of the Granger causality chi-square statistics using one and two lags of each variable.[24] The sum of the lagged coefficients of the ''causal'' variable under consideration is presented in columns (3) and (4).

The null hypothesis of no Granger causality from $Inequality_{it}$ to LE_{it} is rejected at the 6.5% level or better, and the sum of the coefficients on lagged inequality is small but positive in the life expectancy equation. This confirms our result that increased inequality leads, in general, to small (but statistically significant) improvements in population health.

Interestingly, the Granger causality test rejects the null hypothesis that two lags of LE_{it} do not help predict $Inequality_{it}$ at the 8.8% level with a negative sum of the coefficients on life expectancy. Thus, we find some, admittedly weak, evidence that an increase in life expectancy tends to reduce inequality. Failure to account for this endogeneity might also lead to downward biased estimates of the impact of inequality on health.

Table 7

Bivariate Granger causality tests

Null hypothesis	Lags	*p*-value of the Granger causality chi-square statistic	Sum of the lagged coefficients of inequality	Sum of the lagged coefficients of life expectancy
	(1)	(2)	(3)	(4)
$Inequality_{it}$ does not cause LE_{it}	1	0.023	0.007	
	2	0.065	0.004	
LE_{it} does not cause $Inequality_{it}$	1	0.224		-0.069
	2	0.088		-0.054

Notes: This table reports the *p*-values of Granger-causality VAR tests. The null hypothesis is that one or two lags of the (demeaned) series of $Inequality_{it}$ (LE_{it}) do not help predict the series of LE_{it} ($Inequality_{it}$).

4.5. Cross-country differences in the effect of inequality on health

The apparent discrepancy between our results and many previous results may be partially due to the fact that, by using between-dimension (group mean) estimators, we allow for slope heterogeneity. Previous studies, in contrast, implicitly assume that the effect of income inequality on health is the same for all countries. Figure 5 illustrates that this assumption is not justified. The

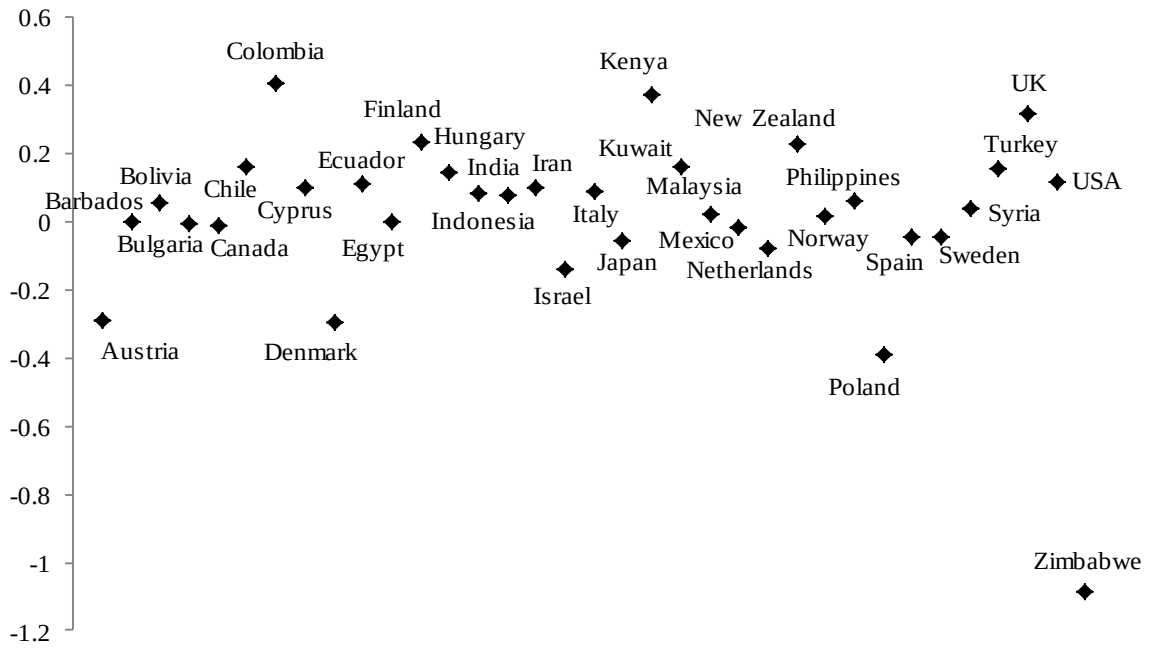
[24] OLS estimates are biased in the presence of country fixed effects and lagged dependent variables. However, Nickell (1981) has shown that the bias is inversely related to the number of years in the sample. Since our sample covers 26 years, the bias in the estimates is likely to be small. Therefore, we follow common practice and estimate the VAR by OLS.

figure plots the individual country DOLS estimates of the coefficients on $Inequality_{it}$. These estimates should of course be treated with caution given the relatively short time span of our data. What can be safely concluded is that there is considerable heterogeneity: the coefficients range from -1.08 in Zimbabwe to 0.41 in Colombia. More specifically, we find that in 13 countries (Austria, Bulgaria, Canada, Denmark, Egypt, Israel, Japan, Mexico, Netherlands, Poland, Spain, Sweden, and Zimbabwe), an increase in inequality is associated with a reduction in life expectancy. In 22 cases (Barbados, Bolivia, Chile, Colombia, Cyprus, Ecuador, Finland, Hungary, India, Indonesia, Iran, Italy, Kenya, Kuwait, Malaysia, New Zealand, Norway, Philippines, Syria, Turkey, United Kingdom, United States), an increase in inequality is associated with an increase in life expectancy. Thus, although the long-run effect of inequality on health is positive in general or on average, income inequality adversely affects health in some countries.

Another striking feature of Figure 5 is that widening income gaps are associated with better health in the most unequal countries in our sample (Kuwait, Kenya, the Philippines, Bolivia, and Indonesia). By contrast, widening income gaps are associated with poorer health in the three most equal countries (Sweden, Poland, and Bulgaria). This appears to be in conflict with the so-called threshold hypothesis (Kondo et al., 2009), which posits that income inequality harms health only if income gaps are sufficiently wide.

Figure 5

Individual country DOLS estimates



Notes: Sample countries listed in alphabetical order from the left to the right.

5. Conclusions

It was widely accepted until recently that more unequal societies are less healthy. However, Wilkinson's (1996) verdict that the distribution of income is one of the most powerful determinants of the health of whole populations rested on poor data and weak statistical tests. More recent studies increasingly eroded the previously held view. Yet important issues have remained unresolved so far.

In the present analysis, we attempted to overcome several limitations by employing panel cointegration techniques. Most importantly, this approach allowed us to account for endogeneity concerns. Reverse causality running from health to income inequality has been largely ignored in previous empirical research. In addition, we included country-specific fixed effects to mitigate omitted variable bias. Another advantage was that we identified cross-country heterogeneity, whereas alternative approaches are typically based on the unduly restrictive assumption that the coefficients of the variables of interest are the same across all countries.

We found that income inequality has, on average, a statistically significant positive impact on population health. Even though wider income gaps improve health conditions only by a quantitatively small margin, our major result proved to be extremely robust. Findings were hardly affected when excluding individual sample countries, or removing either low- or high-income countries from the sample. This also held when employing different measures of health outcomes or income inequality. At the same time, we found some evidence that inequality is endogenous in the sense that poor health widens income gaps. Finally, the effect of income inequality on health differed considerably across countries. Widening income gaps impaired health outcomes in about one third of our sample.

Although some recent studies pointed into the same direction, notably Mellor and Milyo (2001) as well as Leigh and Jencks (2007), it was still surprising to find wider income gaps to cause slightly better health outcomes. 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.

Interdisciplinary research could provide further insights in this respect. Furthermore, it has to be stressed that our approach is not exhaustive. Additional cointegrating relationships with health

outcomes are likely to exist; possible candidates include education, social capital and the environment. They deserve attention in future research; but it would not "destroy" the cointegrating relationship with income inequality if additional variables were considered. Finally, the observed country-specific heterogeneity in the long-run effect of income inequality on health outcomes poses new questions related to the factors that can help explain this heterogeneity.

Acknowledgements

We thank Christopher Sandy Jencks and S.V. (Subu) Subramanian for helpful comments on an earlier draft of this paper.

Appendix

A.1. 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

$$\Delta x_{it} = z_{it}'\gamma + \rho_i x_{it-1} + \sum_{j=1}^{p_i} \varphi_{ij} \Delta x_{it-j} + \varepsilon_{it}, \tag{A.1}$$

where p_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 (=35) 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

$$\Delta x_{it} = z_{it}'\gamma + \rho_i x_{it-1} + \sum_{j=1}^{p_i} \varphi_{ij} \Delta x_{it-j} + \alpha_i \bar{x}_{t-1} + \sum_{j=0}^{p_i} \eta_{ij} \Delta \bar{x}_{t-j} + v_{it}\,, \tag{A.3}$$

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

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

where t_i is the OLS t ratio of ρ_i in Equation 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 A1. 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 $Inequality_{it}$ 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_{it}	constant, trend	-0.15	-2.21
$Inequality_{it}$	constant, trend	1.81	-1.99
First differences			
ΔLE_{it}	Constant	-2.02*	-5.42**
$\Delta Inequality_{it}$	Constant	-6.54**	-2.55**

Notes: Three lags were selected to adjust for autocorrelation. The IPS statistic is distributed as N(0, 1). The relevant 5% (1%) critical value for the CIPS statistics is -2.67 (-2.83) with an intercept and a linear trend, and -2.15 (-2.32) with an intercept. ** (*) denote significance at the 1% (5%) level.

A.2. 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

$$LE_{it} = a_i + \delta_i t + b_i Inequality_{it} + \varepsilon_{it} \tag{A.5}$$

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

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

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

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

where φ is the respective panel or group statistic, and μ and v are the expected mean and variance of the corresponding statistic, tabulated by Pedroni (1999). Note that under the alternative hypothesis, the panel *v*-statistic diverges to positive infinity, and the right tail of the normal distribution is used to reject the null hypothesis of no cointegration, while for the remaining six statistics, the left tail of the normal distribution is used to reject the null hypothesis.

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

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

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:

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

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 countries in the panel have a common co-integrating rank, i.e., at most r (possibly heterogeneous) cointegrating relationships among the p variables: $H_0 : rank(\Pi_i) = r_i \le r$ 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.

Table A.2

Panel cointegration tests

Panel v-statistic	24.36**	
Panel ρ-statistic	-3.88**	
Panel PP-statistic	-8.10**	
Panel ADF-statistic	-9.20**	
Group ρ-statistic	-1.27	
Group PP-statistic	-1.88*	
Group ADF-statistic	-2.34**	
CIPS statistic for the residuals of the CCE long-run relations	-3.33**	
	Cointegration rank	
	$r = 0$	$r = 1$
Panel trace statistics (unadjusted)	18.59**	0.61
Panel trace statistics (adjusted)	13.47**	-1.01

Notes: ** (*) indicate a rejection of the null of no cointegration at the 1% (5%) level. Under the alternative hypothesis, the panel v and the standardized panel trace statistics diverge to positive infinity so that the right tail of the normal distribution is used to reject the null hypothesis. The relevant 1% critical value for the CIPS statistic is -2.32. The number of lags was determined by the Schwarz criterion with a maximum of three lags. The panel statistics are weighted by the member specific long-run conditional variances (see Pedroni (1999) for details).

The results of these panel cointegration tests are presented in Table A2. Six of the seven Pedroni statistics reject the null of no cointegration. Specifically, the ADF-type tests reject the null hypothesis at the one percent level. Given that these tests have been shown to have the highest power for smaller sample sizes, such as T = 26 (Pedroni, 2004), the ADF test results, in particular, provide strong evidence of cointegration. This conclusion is supported by the CIPS and the

(unadjusted and small sample adjusted) trace statistics, which show that LE_{it} and $Inequality_{it}$ are cointegrated (or exhibit a single cointegrating vector).

A.3. Countries in the samples

Table A.3

List of countries included in this study

Australia	1, 2, 3, 5	Japan	1, 2, 3
Austria	2, 3	Kenya	1, 2, 3
Barbados	1, 3	Korea, Republic of	2, 3
Belgium	2, 3	Kuwait	1, 2, 3
Bolivia	1, 2, 3	Luxembourg	2, 3
Bulgaria	1, 2, 3	Malaysia	1, 2, 3
Canada	1, 2, 3, 4, 5	Malta	2, 3
Chile	1, 2, 3	Mauritius	2, 3
Colombia	1, 2, 3	Mexico	1, 2, 3
Cyprus	1, 3	Netherlands	1, 2, 3, 4, 5
Denmark	1, 2, 3	New Zealand	1, 2, 3, 4, 5
Ecuador	1, 2, 3	Norway	1, 2, 3
Egypt	1, 2, 3	Philippines	1, 2, 3
Finland	1, 2, 3	Poland	1, 2, 3
France	4, 5	Singapore	2, 3
Greece	2, 3	Spain	1, 2, 3
Hungary	1, 2, 3	Sweden	1, 2, 3, 4, 5
Iceland	2, 3	Switzerland	4, 5
India	1, 2	Syria	1, 2, 3
Indonesia	1, 2, 3	Turkey	1, 2, 3
Iran	1, 2, 3	United Kingdom	1, 2, 3, 4, 5
Ireland	2, 3	United States	1, 2, 3, 4, 5
Israel	1, 3	Venezuela	2, 3
Italy	1, 2, 3	Zimbabwe	1, 2, 3

1. Sample of 35 countries for which continuous data on LE_{it} and $Inequality_{it}$ are available over the period from 1970 to 1995.
2. Sample of 43 countries for which continuous data on $\log(IMR_{it})$, $\log(U5MR_{it})$, and $Inequality_{it}$ are available over the period from 1970 to 1995.
3. Sample of 44 countries for which continuous data on $\log(CR_{it})$ and $Inequality_{it}$ are available over the period from 1970 to 1995.

4. Sample of 8 countries for which continuous data on LE_{it} and $TopDecile_{it}$ are available over the period from 1961 to 1996.
5. Sample of 9 countries for which continuous data on log(IMR_{it}), log($U5MR_{it}$), log(CR_{it}), and $TopDecile_{it}$ are available over the period from 1961 to 1996.

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