

THE CAUSALITY BETWEEN RELATIVE POVERTY AND AGE AND GENDER-SPECIFIC MORTALITY: EVIDENCE FROM G7

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Abstract

Income distribution is one of the key determinants of population health. Associations between population mortality rates and income inequality have been reported in numerous papers comparing data from both developed and developing countries, from areas within countries, cross-sectionally and overtime. In this study, it is analyzed the possibility of a causal relationship between population mortality by age and gender and income inequality for G7 countries over the period 1970-2010. Taking into account both the cross-sectional dependence and the heterogeneity and using the non-causality test developed by Dumitrescu and Hurlin (2012), the empirical results of the study showed that there were unidirectional causal relationships from income inequality to population mortality for both male and female at the ages below 50.

Keywords: Income inequality, population health, mortality rates, panel causality, G7

JEL Classifications: D31, I130, I32, C23

1. Introduction

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In recent years there has been a great deal of interest in whether or not a prominent and controversial hypothesis that higher income inequality in a society is responsible for higher mortality is valid. If inequality in the income distribution produces unequal access to services such as education, health care, and police protection, the negative effects for those at the bottom of the distribution will not be offset by positive outcomes for those at the top of the distribution. Moreover, the negative health outcomes of an unequal income distribution may not be limited to the portion of the population with fewer resources. Differential access to resources and services may result in less effective preventive health care more costly disease control, or higher crime rates, affecting the health and mortality risk of the entire population. But this is an economic evaluation for the income inequality-mortality relationship. Most explanations for the relationship between income inequality and mortality fall into two categories: economic and psychosocial.

The psychosocial side of the relationship between two variables rests on the following explanation: Under this category, income inequality and mortality risk are in a relationship through the effects of emotional and psychological stress on health. The psychosocial hypothesis posits that levels of depression, isolation, hopelessness, insecurity and anxiety, which are known correlates of mortality, are all results of relative economic position. In addition, some researchers argue that income inequality inhibits many of the social behaviors that may reduce stress, insecurity, anxiety and feeling negative about yourself and valued by others such as participation in voluntary organizations or community groups and investment in ongoing education and training (Daly and Wilson 2013).

We analyze the causality relationship between income inequality and population mortality in G7 countries over the years 1970-2010. The data allows us to control for time-invariant country specific factors. We use mortality as the measure for health and the high-quality Human Mortality Database (2014) as the data source. The source for Gini index of income inequality, as a measurement of income inequality is the United Nations University World Institute Development Economics Research (UNU-WIDER) World Income Inequality Database (2014).

We distinguish population mortality in terms of genders and age groups. Age groups are classified according as 0-1 ages, ages 1-14, ages 15-49 and ages above 50.

The rest of this paper is structured as follows. Section 2 outlines the previous literature, Section 3 contains the data set and the methodology and presents the main findings and Section 4 concludes the study.

2. Literature Review

Associations between population mortality rates and income inequality have been reported in the large of number of studies comparing data from both developed and developing countries, from counties within countries, cross-sectionally and over time. Some empirical studies have produced mixed and contradictory results, as well as most empirical studies show a widespread tendency for population death rates to be lower in societies where income differences are smaller.

Using data for the 1960's Fuchs (1974) showed that there was no relationship between per capita income and age-specific adult mortality rates across economically developed countries. Similarly, Preston (1976) confirmed Fuchs's evidence of no correlation between income and life expectancy for economically developed countries in 1960's.

Duulep (1995) test for the effect of income on the mortality of economically developed thirty-seven countries. According to OLS method, the death rate of men aged 50-54 is negatively associated to the average income received by the bottom 10 percent of the population in each country, used as a proxy variable of the income distribution. Moreover, the regression results showed that the estimated effect of average income is statistically insignificant. Therefore, the study concluded that the average income received by the bottom income 10 percent of the population is likely to be much more important in determining the mortality rates of economically developed countries than the average income of population.

Kaplan et al. (1996) examined the relationship between health outcomes and income inequality for the 50 states in the United States in the years of 1980 and 1990. Their findings suggested that

income inequality was associated with mortality trends. Moreover, the findings of Kaplan et al. (1996) showed that income inequality was also significantly associated with age-specific mortalities and rates of low birth weight, homicide, violent crime, work disability, expenditures on medical care and police protection, smoking and sedentary activity.

Using longitudinal data for the years 1978 to 1982 and 1988 to 1992 in order to investigate the effect of income inequality on state-level mortality, which are identical to data used by Kaplan et al. (1996), Daly et al. (1998) fail to find significant associations between inequality and mortality, except in the case of those with middle incomes between the ages of 25 and 64.

Wilkinson and Pickett (2006) note that the negative association between income inequality and population health was not found in studies using data from between the later 1980s and mid 1990s. Their explanations for these findings have to do with structural processes that evolved in parallel with the increase in income inequality, and which worked toward making population health better, rendering thus the relationship between income inequality and health non significant.

Lobmayer and Wilkinson (2000) tested the link between income inequality and mortality among 14 OECD countries. Using median income as a measurement of income inequality due to their findings that there was a close positive relation between income inequality and median income, Lobmayer and Wilkinson (2000) found that wider income distribution is related to higher premature mortality, and higher age-specific mortality rates below, but not above, age 65 years.

Lynch et al. (2001) investigated cross-sectional associations between the variables such as income inequality, low birth weight, life expectancy, mortality rate for 23 countries over the period 1989-1992. Their empirical results showed that income inequality was strongly and negatively associated with life expectancy. The results also showed that higher income inequality was strongly associated with higher infant mortality, low birth weight and mortality in people aged 1-14 years in both sexes. Associations between income inequality and mortality declined with age at death, and then reversed, so that among those aged 65 years or older. Thus,

associations with mortality above age 65 were the opposite of that predicted by the theory that higher income inequality is bad for health.

Beckfield (2004) analyzed 115 countries over 50 years and used a fixed-effect model to take into account unobserved heterogeneity, but found no evidence for the income inequality-health hypothesis, which posits that levels and changes in income inequality to be negatively related to life expectancy and this relationship to be weaker as the level of economic development is higher, but still significant in the high-developed economies. Babones (2008) analyzed 135 countries over a period from 1970 to 1995 and found that changes in income distribution are associated with changes in life expectancy and infant mortality. However, the association vanished when income per capita per head was added as control.

França-Cavalcante and Paes-Antunes (2007) tested the following hypotheses: a) the health status of the population of metropolitan regions of Brazil is associated to absolute income b) the health status of population of metropolitan regions is not associated to unequal income distribution. According to the results of their regression analysis, it is found that per capita income was an important determinant in explaining the variations in life expectancy at birth and infant mortality rate. On the other hand, no statistically significance evidence was found for the regression coefficients associated to Gini's index as a measurement of income inequality for life expectancy at birth and infant mortality rate.

Wilkinson and Pickett (2009) measured the strength of the relationship between 10 different county mortality rates and state income inequality, conditional on county median incomes across 3139 counties in the United States. Their findings showed that mortality that was more strongly related to county median income was also more strongly associated with state-level income inequality. Moreover they found that mortality was reduced across a wide range of county median incomes in more equal states.

Torre and Myrskylä (2011) tested for 21 developed countries the relative income-health hypothesis, which is postulated that income distribution is one of the key determinants of population health. Covering the period 1975-2006, the study reveals that there exists a positive

link between income inequality and mortality at ages 1-14 both girls and boys. Moreover, the empirical results showed that there exists a strong positive association between income inequality and mortality at ages 15-49 for men, while the association is weaker at ages 15-49 for women.

Pop et al. (2012) investigated the relationship among life expectancy, Gini index and GDP per capita. Covering a number of 140 countries, including 29 high-developed, 61 middle-developed, 50 low-developed countries, and the time period of 1987-2008, the results of the study implied that a decrease in a country's income inequality parallel with an increase in its wealth can help to improve health in economically lesser-developed countries, but not in high-developed countries.

Daly and Wilson (2013) estimated the effects of inequality on mortality risk for the period 1990-2000 in the United States. Using the Gini coefficient as the measure of inequality, Daly and Wilson (2013) found a negative inequality-mortality relationship after taking account of time-invariant characteristics. Their finding that increases in inequality are associated with declines in mortality at the country level was evaluated as part of the psychosocial side of the relationship between income inequality and mortality.

3. Data, Empirical Application and Findings

3.1 Data

In this study, we investigated the causality relationship between population mortality by genders and age groups and income inequality in the G7 countries (Germany, France, Canada, Japan, Italy, United Kingdom and United States) for the period 1970-2010.

Eviews 8.0 and Gauss 6.0 statistical packages were used in the econometric analyses. The variables, their symbols and their sources were presented in Table 1. Table 2 and Table 3 provide summary statistics of the variables and correlation matrix, respectively.

Table 1 Data Set

<i>Variables</i>	<i>Explanations</i>	<i>Source</i>
<i>gini</i>	Gini coefficient	the WIID database (2014)
<i>f_infant</i>	Female infant mortality rate- the number of deaths of infants under one year old	the Human Mortality Database (2014)
<i>f_14</i>	Female mortality rate for ages 1-14	
<i>f_49</i>	Female mortality rate for 15-49	
<i>f_over</i>	Female mortality rate for the ages above 50	
<i>m_infant</i>	Male infant mortality rate- the number of deaths of infants under one year old	
<i>m_14</i>	Male mortality rate for ages 1-14	
<i>m_49</i>	Male mortality rate for 15-49	
<i>m_over</i>	Male mortality rate for the ages above 50	
<i>t_infant</i>	Total infant mortality rate- the number of deaths of infants under one year old	
<i>t_14</i>	Total mortality rate for ages 1-14	
<i>t_49</i>	Total mortality rate for 15-49	
<i>t_over</i>	Total mortality rate for the ages above 50	

Following the existing empirical literature on the population mortality and income inequality relationship, in order to investigate the causality between the variables the bivariate models described are as follows:

$f_infant = f(gini)$	$m_infant = f(gini)$	$t_infant = f(gini)$
$f_14 = f(gini)$	$m_14 = f(gini)$	$t_14 = f(gini)$
$f_49 = f(gini)$	$m_49 = f(gini)$	$t_49 = f(gini)$
$f_over = f(gini)$	$m_over = f(gini)$	$t_over = f(gini)$

Table 2 presents descriptive statistics of data used in this paper. According to Table 2, there is no sampling bias in the data. The means of all variables are close neither to their minimum nor

maximum value, which indicates that there is no disproportion. Also, the standard deviations of the variables are widely dispersed around the mean.

Table 2 Descriptive Statistics and Correlation Matrix of the Variables

	<i>Mean</i>	<i>Median</i>	<i>Maximum</i>	<i>Minimum</i>	<i>Std. Dev.</i>
<i>gini</i>	33,951	32,900	52,300	19,800	5,918
<i>f_infant</i>	0,008	0,006	0,027	0,002	0,005
<i>f_14</i>	0,001	0,001	0,002	0,000	0,000
<i>f_49</i>	0,007	0,006	0,012	0,003	0,002
<i>f_over</i>	2,525	2,287	8,188	1,613	0,890
<i>m_infant</i>	0,010	0,008	0,033	0,003	0,006
<i>m_14</i>	0,001	0,001	0,002	0,000	0,000
<i>m_49</i>	0,013	0,013	0,024	0,006	0,004
<i>m_over</i>	2,690	2,393	8,705	1,667	0,992
<i>t_infant</i>	0,009	0,007	0,030	0,002	0,005
<i>t_14</i>	0,001	0,001	0,002	0,000	0,000
<i>t_49</i>	0,010	0,010	0,018	0,005	0,003
<i>t_over</i>	2,579	2,376	8,328	1,707	0,784

According to correlation matrix presented in Table 3, all the mortality rates except for *m_over* are positively correlated with Gini coefficient.

Table 3 Correlation Matrix

	<i>gini</i>	<i>f_infant</i>	<i>f_14</i>	<i>f_49</i>	<i>f_over</i>	<i>m_infant</i>	<i>m_14</i>	<i>m_49</i>	<i>m_over</i>
<i>gini</i>	1.00	0.11	0.03	0.12	0.02	0.10	0.03	0.11	-0,03
<i>f_infant</i>	0.11	1.00	0.88	0.79	-0,02	0.99	0.87	0.68	-0,11
<i>f_14</i>	0.03	0.88	1.00	0.86	-0,06	0.89	0.99	0.77	-0,16
<i>f_49</i>	0.12	0.79	0.86	1.00	-0,13	0.80	0.86	0.92	-0,17
<i>f_over</i>	0.02	-0,02	-0,06	-0,13	1.00	-0,02	-0,08	-0,20	0.20
<i>m_infant</i>	0.10	0.99	0.89	0.80	-0,02	1.00	0.88	0.70	-0,11

<i>m_14</i>	0.03	0.87	0.99	0.86	-0,08	0.88	1.00	0.78	-0,16
<i>m_49</i>	0.11	0.68	0.77	0.92	-0,20	0.70	0.78	1.00	-0,17
<i>m_over</i>	-0,03	-0,11	-0,16	-0,17	0.20	-0,11	-0,16	-0,17	1.00

Source: Author' estimations.

3.2 Preliminary Analysis

We tested the presence of the causality relationship between population mortality by genders and age groups and income inequality in the G7 countries. For this aim, we apply panel data techniques, which give more informative data, more variability, less co-linearity among the variables, more degrees of freedom and so, more efficiency (Baltagi 2005).

Cross-sectional dependency and homogeneity analyses

There have been two important issues to be considered in a panel data analysis. These are testing for cross-sectional dependency across the members of panel and determining whether slope coefficients are homogeneous or heterogeneous. In terms of economics, cross-sectional dependence could be explained that a shock affecting individuals forming a panel may also affect other individuals. There are various tests analyzing cross-sectional dependence in panel data. In this study, CD_{BP} , Lagrange multiplier test statistic, developed by Breusch and Pagan (1980) and CD_{LM} , CD tests, developed by Pesaran (2004) are used in order to control cross-sectional dependence across the countries. The three test statistics are formulated as follows:

$$CD_{BP} = T \cdot \sum_{i=1}^{N-1} \sum_{j=i+1}^N \hat{\rho}_{ij}^2 \square \chi_{N.(N-1)/2}^2$$

$$CD_{LM} = \sqrt{\frac{1}{N.(N-1)}} \left[\sum_{i=1}^{N-1} \sum_{j=i+1}^N (T \cdot \hat{\rho}_{ij}^2 - 1) \right] \square N(0,1)$$

$$CD = \sqrt{\frac{2T}{N.(N-1)}} \left[\sum_{i=1}^{N-1} \sum_{j=i+1}^N \hat{\rho}_{ij} \right] \square N(0,1)$$

where $\hat{\rho}_{ij}$ shows the estimation of the correlation coefficient among the residuals obtained from individual OLS estimations. Under the null hypothesis of no cross-sectional dependency the CD_{BP} test, is used when N is fixed and T goes to infinity (T is large relative to N), is asymptotically distributed as chi-squared with $N(N-1)/2$ degrees of freedom. Under the null hypothesis of no cross-sectional dependence CD_{LM} test, which is one of the Pesaran (2004)'s

tests to examine cross-sectional dependence, is useful when N is great and T is small (N is large relative to T) and it is asymptotically distributed as standard normal. Another test of Pesaran (2004) test to examine cross-sectional dependence is CD test, which is used when T and N go to infinity in any order, is asymptotically distributed as standard normal.

The empirical results of three different tests, which are carried out in order to investigate the existence of cross-sectional dependence are illustrated in Table 3. According to Table 3, it is clear that the null of no cross-sectional dependence across the G7 countries is strongly rejected. So, it should be applied the second generation tests, taking into account cross-sectional dependence in analyzing whether the series contain unit root.

Table 3 Results for Cross-Sectional Dependence Tests

<i>Variabl</i>	<i>CD_{BP}</i>	<i>CD_{LM}</i>	<i>CD</i>
<i>e</i>			
<i>gini</i>	54.809 (0.000)***	5.217 (0.000)***	-4.276 (0.000)***
<i>f_infan</i>	62.226 (0.000)***	6.361 (0.000)***	-1.938 (0.026)**
<i>t</i>			
<i>f_14</i>	54.281 (0.000)***	5.135 (0.000)***	-3.629 (0.000)***
<i>f_49</i>	57.094 (0.000)***	5.569 (0.000)***	-2.392 (0.008)***
<i>f_over</i>	53.707 (0.000)***	5.047 (0.000)***	-2.860 (0.002)***
<i>m-</i>			
<i>_infant</i>	86.839 (0.000)***	10.159 (0.000)***	-2.432 (0.008)***
<i>m_14</i>	48.373 (0.001)***	4.224 (0.000)***	-3.832 (0.000)***
<i>m_49</i>	71.970 (0.000)***	7.865 (0.000)***	-1.804 (0.036)**

<i>m_over</i>	55.184 (0.000)***	5.275 (0.000)***	-3.726 (0.000)***
<i>t_infan</i>	87.437 (0.000)***	10.251 (0.000)***	-1.752 (0.040)**
<i>t_14</i>	61.032 (0.000)***	6.177 (0.000)***	-3.462 (0.000)***
<i>t_49</i>	78.852 (0.000)***	8.927 (0.000)***	-1.671 (0.047)**
<i>t_over</i>	44.259 (0.002)***	3.589 (0.000)***	-3.151 (0.001)***

***, ** indicate rejection of the null hypothesis at the 1%, 5% levels of significance respectively.

Source: Author' estimations.

Determining whether or not slope coefficients are homogeneous is also important in a panel causality analysis to impose causality restrictions on estimated coefficients. Pesaran and Yamagata's (2008) homogeneity tests are applied to test for homogeneity in slope coefficient estimates in this study. Pesaran and Yamagata (2008) proposed a standardized version of Swamy's test of slope homogeneity for panel data models. Pesaran and Yamagata (2008) consider the following panel data model with fixed effects and heterogeneous slopes:

$$y_{it} = \alpha_i + \beta_i' x_{it} + \varepsilon_{it}, \quad i=1, \dots, N, t=1, \dots, T$$

where α_i is unit-specific intercept and bounded on a compact set, x_{it} is a $k \times 1$ vector of strictly exogenous regressors, β_i is a $k \times 1$ vector of slope coefficients. The null hypothesis and the alternative hypothesis of interest are

$$H_0: \beta_i = \beta \text{ for all } i,$$

$$H_1: \beta_i \neq \beta \text{ for a non-zero fraction of pairwise slopes for } i \neq j.$$

Under the null hypothesis $\tilde{\Delta} \xrightarrow{j} N(0,1)$ as $(N,T) \rightarrow \infty$ so long as $\sqrt{N}/T^2 \rightarrow 0$,

where the standardized dispersion statistic, $\tilde{\Delta}$ is defined by $\tilde{\Delta} = \sqrt{N} \cdot \left(\frac{N^{-1}\tilde{S} - k}{\sqrt{2k}} \right)$ where \tilde{S} is the Swamy's statistic, is valid for a fixed N and as $T \rightarrow \infty$. Also, Pesaran and Yamagata (2008) proposed the following mean and variance bias adjusted version of $\tilde{\Delta}$ for the small samples¹.

$$\tilde{\Delta}_{adj} = \sqrt{N} \cdot \left(\frac{N^{-1}\tilde{S} - E(z_{iT})}{\sqrt{Var(z_{iT})}} \right) \text{ where } E(z_{iT}) = k, Var(z_{iT}) = \frac{2k \cdot (T - k - 1)}{T + 1}.$$

The results of the delta test statistics are illustrated in Table 4. The reported results in Table 4 indicate that the homogeneity tests reject the equality hypothesis, supporting that the parameters are heterogeneous.

Table 4 Results for Homogeneity Test

	$\tilde{\Delta}$ test statistic	prob. value	$\tilde{\Delta}_{adj}$ test statistic	prob. value
<i>f_infant</i>	7.239***	0.000	7.512***	0.000
<i>f_14</i>	6.652***	0.000	6.904***	0.000
<i>f_49</i>	6.221***	0.000	6.456***	0.000
<i>f_over</i>	1.495*	0.067	1.551*	0.060
<i>m_infant</i>	7.246***	0.000	7.519***	0.000
<i>m_14</i>	6.794***	0.000	7.050***	0.000
<i>m_49</i>	4.448***	0.000	4.616***	0.000
<i>m_over</i>	-0.038	0.515	-0.039	0.516
<i>t_infant</i>	7.247***	0.000	7.520***	0.000
<i>t_14</i>	6.753***	0.000	7.008***	0.000
<i>t_49</i>	5.121***	0.000	5.315***	0.000
<i>t_over</i>	1.350*	0.088	1.401*	0.081

***, **, * indicate rejection of the null hypothesis at the 1%, 5%, 10% levels of significance respectively.

Source: Author' estimations.

¹ For a detailed proof and information, see Swamy (1970) and Pesaran and Yamagata (2008).

Unit root analysis

After analyzed the cross-section dependency and the homogeneity, it should be controlled the existence of unit root in the series in order to get unbiased estimations. Many recent studies rely on panel unit root tests in order to increase the statistical power of their empirical findings. We applied the panel stationarity tests Z_A^{SPC} and Z_A^{LA} proposed by Hadri and Kurozumi (2012).

Hadri and Kurozumi (2012) consider the following model:

$$y_{it} = z_i' \delta_i + f_t \gamma_i + \varepsilon_{it}, \quad \varepsilon_{it} = \phi_{i1} \varepsilon_{it-1} + \dots + \phi_{ip} \varepsilon_{it-p} + v_{it} \quad \text{for } i=1, \dots, N, t=1, \dots, T$$

where z_i' is deterministic, $z_i' \delta_i$ is the individual effect while f_t is a one-dimensional unobserved common factor, γ_i is the loading factor, and ε_{it} is the individual-specific error, which follows an AR(p) process.

For the correction of cross-sectional dependence, for each I, Hadri and Kurozumi (2012) regress y_{it} on $w_t = [z_i', \bar{y}_t, \bar{y}_{t-1}, \dots, \bar{y}_{t-p}]$ and construct the following test statistic:

$$Z_A = \frac{\sqrt{N}(\overline{ST} - \xi)}{\zeta} \quad \text{where } \overline{ST} = 1/N \cdot \sum_{i=1}^N ST_i \quad \text{with } ST_i = \frac{1}{\hat{\sigma}_i^2 T^2} \sum_{t=1}^T S_{it}^w, \quad \text{where } S_{it}^w = \sum_{s=1}^t \hat{\varepsilon}_{is}, \quad \hat{\sigma}_i^2 \text{ is the estimator of}$$

the long-run variance.

$$\text{and } \begin{cases} \xi = \xi_m = 1/6 & \zeta^2 = \zeta_m^2 = 1/45 & \text{when } z_t = z_t^m = 1 \\ \xi = \xi_\tau = 1/15 & \zeta^2 = \zeta_\tau^2 = 11/6300 & \text{when } z_t = z_t^\tau = [1, t]' \end{cases}$$

Hadri and Kurozumi (2012) called Z_A statistic as the panel-augmented KPSS test statistic, due to the fact that \overline{ST} is the average of the Kwiatkowski et al. (1992) test statistic across i . They construct S_{it}^w using these regression residuals. In this case, it can be seen that numerators of each ST_i weakly converges to

$$\frac{1}{T^2} \sum_{t=1}^T (S_{it}^w)^2 \Rightarrow \sigma_i^2 \int_0^1 [V_i^e(r) + \tilde{\gamma}_i R_N]^2, \quad \text{where } \tilde{\gamma}_i = \gamma_i / \bar{\gamma}, \quad R_N \text{ is } O_p(1/\sqrt{N}) \text{ over } 0 \leq r \leq 1$$

$$\sigma_i^2 = \sigma_{\varepsilon_i}^2 / (1 - \phi_{i1} - \dots - \phi_{ip})^2$$

Hadri and Kurozumi (2012) divide the numerator of each ST_i by a consistent estimator of the long-run variance σ_i^2 to correct for serial correlation and estimate the AR(p) model augmented by the lags of \bar{y}_t for each i by the least-squares method,

$y_{it} = z_i' \hat{\delta}_i + \hat{\phi}_{i1} \cdot y_{it-1} + \dots + \hat{\phi}_{ip} \cdot y_{it-p} + \hat{\Psi}_{i0} \bar{y}_t + \dots + \hat{\Psi}_{ip} \bar{y}_{t-p} + \hat{v}_{it}$. Hadri and Kurozumi (2012) construct the estimator of the long-run variance by

$\hat{\sigma}_{iSPC}^2 = \frac{\hat{\sigma}_{vi}^2}{(1 - \hat{\phi}_i)^2}$ where $\hat{\sigma}_{vi}^2 = 1/T \cdot \sum_{t=1}^T \hat{v}_{it}^2$ and $\hat{\phi}_i = \min \left\{ 1 - \frac{1}{\sqrt{T}}, \sum_{j=1}^p \hat{\phi}_{ij} \right\}$. The test statistic of Z_A^{SPC} is created using

the formula below by Hadri and Kurozumi (2012):

$$Z_A^{SPC} = \frac{1}{\hat{\sigma}_{iSPC}^2 \cdot T^2} \sum_{t=1}^T (S_{it}^w)^2$$

Hadri and Kurozumi (2012) considered the lag-augmented method proposed by Choi (1993) and Toda and Yamamoto (1995) to obtain the test statistic of Z_A^{LA} and estimated the following an AR(p+1) model:

$$y_{it} = z_i' \tilde{\delta}_i + \tilde{\phi}_{i1} \cdot y_{it-1} + \dots + \tilde{\phi}_{ip} \cdot y_{it-p} + \tilde{\phi}_{ip+1} \cdot y_{it-p-1} + \tilde{\Psi}_{i0} \bar{y}_t + \dots + \tilde{\Psi}_{ip} \bar{y}_{t-p} + \tilde{v}_{it}$$

After this estimation, the test statistic Z_A^{LA} is created using the Formula below by Hadri and Kurozumi (2012):

$$Z_A^{LA} = \frac{1}{\hat{\sigma}_{iLA}^2 \cdot T^2} \sum_{t=1}^T (S_{it}^w)^2, \text{ where } \hat{\sigma}_{iLA}^2 = \frac{\hat{\sigma}_{vi}^2}{(1 - \tilde{\phi}_{i1} - \dots - \tilde{\phi}_{ip})^2}$$

The test of Hadri and Kurozumi (2012) states that under a null hypothesis, series do not contain unit root, while an alternative hypothesis states that series contain unit root. These hypotheses are formulated as follows:

$$H_0 : \phi_i(1) \neq 0 \text{ for all } i$$

$$H_0 : \phi_i(1) = 0 \text{ for some } i$$

The null distributions of both statistic (Z_A^{SPC} and Z_A^{LA}) are asymptotically standard normal, while they diverge to infinity under the alternative hypothesis. The Hadri and Kurozumi panel stationarity test allowing serial correlation and cross-sectional dependence can be used in which both $T < N$ and $T > N$.

The Hadri and Kurozumi (2012) panel stationarity test results related to gini coefficient, population mortality rate in terms of gender and age are shown in Table 5. According to Table 5, it is clear that the null hypothesis of stationarity cannot be rejected for population mortality rate by gender and age, while the null hypothesis can be rejected for the variable of gini in level. But,

after taking first-difference of gini variable, the null hypothesis of no unit root cannot be rejected at 0.05 significance level.

Table 5 Results for the Hadri-Kurozumi (2012) Stationary Test

<i>Constant</i>			<i>Constant</i>		
<i>Variable</i>	<i>Statistic</i>	<i>p-value</i>	<i>Variable</i>	<i>Statistic</i>	<i>p-value</i>
<i>gini</i>			<i>m_49</i>		
Z_A^{SPC}	1.902**	0.028	Z_A^{SPC}	-0.686	0.753
Z_A^{LA}	2.905***	0.001	Z_A^{LA}	-0.692	0.755
<i>f_infant</i>			<i>m_over</i>		
Z_A^{SPC}	-1.041	0.851	Z_A^{SPC}	1.089	0.138
Z_A^{LA}	-0.429	0.661	Z_A^{LA}	1.640*	0.050
<i>f_14</i>			<i>t_infant</i>		
Z_A^{SPC}	-1.694	0.951	Z_A^{SPC}	-1.272	0.891
Z_A^{LA}	-0.829	0.791	Z_A^{LA}	-1.296	0.902
<i>f_49</i>			<i>t_14</i>		
Z_A^{SPC}	-1.097	0.863	Z_A^{SPC}	-1.675	0.951
Z_A^{LA}	-1.020	0.841	Z_A^{LA}	-0.837	0.791
<i>f_over</i>			<i>t_49</i>		
Z_A^{SPC}	1.310	0.094	Z_A^{SPC}	-0.556	0.711
Z_A^{LA}	1.280	0.100	Z_A^{LA}	-0.771	0.771
<i>m_infant</i>			<i>t_over</i>		
Z_A^{SPC}	-1.314	0.905	Z_A^{SPC}	0.387	0.341
Z_A^{LA}	-1.576	0.942	Z_A^{LA}	1.221	0.111
<i>m_14</i>			<i>d(gini)</i>		
Z_A^{SPC}	-1.359	0.913	Z_A^{SPC}	1.043	0.149
Z_A^{LA}	-0.445	0.671	Z_A^{LA}	1.513*	0.065

***, **, * indicate rejection of the null hypothesis at the 1%, 5%, 10% levels of significance respectively. d denotes the first difference of the variable.

Source: Author' estimations.

In the framework of the findings from the cross-sectional dependency tests, the homogeneity tests and second generation panel unit root tests the panel causality approach proposed by Dumitrescu and Hurlin (2012) seems to be an appropriate method.

3.3 Dumitrescu and Hurlin (2012)'s panel non-causality test

It is examined whether there exists a bilateral causality relationship between the population mortality and relative poverty through the Dumitrescu and Hurlin (2012)'s non-causality test. This test is a simple version of the Granger (1969) non-causality test for heterogeneous panel data models with fixed coefficients. Dumitrescu and Hurlin (2012) consider the following model:

$$y_{i,t} = \alpha_i + \sum_{k=1}^K \gamma_i^{(k)} y_{i,t-k} + \sum_{k=1}^K \beta_i^{(k)} x_{i,t-k} + \varepsilon_{i,t} \quad i=1,\dots,N, t=1,\dots,T$$

where $x_i = (x_{i1}, \dots, x_{iT})'$ and $y_i = (y_{i1}, \dots, y_{iT})'$ are stationary variables in T periods. $\beta_i = (\beta_i^{(1)}, \dots, \beta_i^{(k)})'$. It is assumed that lag orders K are identical for all cross-section units of the panel and the panel is balanced. Besides, it is allowed that autoregressive parameters $\gamma_i^{(k)}$ and the regression coefficients slopes $\beta_i^{(k)}$ are constant in time and they vary across groups. The hypotheses of the Dumitrescu and Hurlin (2012) test are formulated as follows:

$$H_0 : \beta_i = 0 \quad \forall i = 1, \dots, N$$

$$H_1 : \beta_i = 0 \quad \forall i = 1, \dots, N_1$$

$$\beta_i \neq 0 \quad \forall i = N_1 + 1, \dots, N$$

Under the null hypothesis, it is assumed that there is no individual causality relationship from x to y exists. This hypothesis is denoted the Homogeneous Non Causality (HNC) hypothesis. Thus under the null hypothesis of HNC, there is no causal relationship for any of the cross-section units of the panel. The alternative hypothesis is denoted the Heterogeneous Non Causality (HENC) hypothesis. Under the alternative hypothesis, we assume that there is a causal relationship from x to y for a subgroup of individuals and β_i may differ across groups. Besides, under the alternative hypothesis, we assume that there are $N_1 < N$ individual processes with no

causality from x to y and N_1 is unknown but provides the condition $0 \leq N_1/N < 1$. We propose the average statistic $W_{N,T}^{HnC}$ associated with the null HNC hypothesis, as follows:

$$W_{N,T}^{HnC} = 1/N \cdot \sum_{i=1}^N W_{i,T}, \text{ here } W_{i,T} \text{ denotes the individual Wald statistics for the } i^{\text{th}} \text{ cross-section unit}$$

corresponding to the individual test $H_0: \beta_i = 0$

Let denote Z_i the $(T, 2K+1)$ matrix $Z_i = [e: Y_i: X_i]$ where e denotes a $(T, 1)$ unit vector and by

$\theta_i = (\alpha_i \gamma_i' \beta_i)'$ the vector of parameters of the model. Let the test for the HNC hypothesis be $R\theta_i = 0$ where R is a $(K, 2K+1)$ matrix with $R = [0: I_k]$. For each $i=1, \dots, N$, the Wald statistic $W_{i,T}$ corresponding to the individual test $H_0: \beta_i = 0$ is defined as $W_{i,T} = \hat{\theta}_i' \cdot R' \cdot [\hat{\sigma}_i^2 R(Z_i' Z_i)^{-1} R'] \cdot R \cdot \hat{\theta}_i$

where $\hat{\theta}_i$, is the estimate of parameter θ_i obtained under the alternative hypothesis, and $\hat{\sigma}_i^2$ is the estimate of the variance of the residuals. Under the null hypothesis of non-causality, each individual Wald statistic converges to a chi-squared distribution with K degrees of freedom:

$$W_{i,T} \rightarrow \chi^2(K), \forall i = 1, \dots, N$$

The standardized $Z_{N,T}^{HnC}$ for $T, N \rightarrow \infty$ denotes the fact that $T \rightarrow \infty$ first and then $N \rightarrow \infty$ is as follows:

$$Z_{N,T}^{HnC} = \sqrt{N/2K} \cdot (W_{N,T}^{HnC} - K) \rightarrow N(0,1)$$

The standardized average statistic \tilde{Z}_N^{HnC} for a fixed T dimension with $T > 5 + 2K$ is as follows:

$$\tilde{Z}_N^{HnC} = \sqrt{\frac{N}{2K}} \cdot \sqrt{\frac{T-2K-5}{T-K-3}} \cdot \left[\left(\frac{T-2K-3}{T-2K-1} \right) \cdot W_{N,T}^{HnC} - K \right] \rightarrow N(0,1)$$

In this study, we investigated if there exists a causal relationship between population mortality and relative poverty using the Granger non-causality test for heterogeneous panel data developed by Dumitrescu and Hurlin (2012). The Dumitrescu and Hurlin (2012) panel non-causality test results are given in Table 6. According to the results illustrated in Table 6, a unidirectional causal relationship from gini coefficient, used as a proxy variable of relative poverty, to almost all population mortality (except for the ages above 50). No causal relationships were found between the population mortality rate for the ages above 50 and relative poverty.

Table 6 The Dumitrescu-Hurlin Panel Granger Non-Causality Test Results

<i>Hypothesis</i>	<i>Asymptotic</i>	<i>Hypothesis</i>	<i>Asymptotic</i>	<i>Hypothesis</i>	<i>Asymptotic</i>
	$Z_{N,T}^{HnC}$		$Z_{N,T}^{HnC}$		$Z_{N,T}^{HnC}$

<i>f_infant</i> \Rightarrow <i>gini</i>	-1.212 (0.19)	<i>m_infant</i> \Rightarrow <i>gini</i>	-1.159 (0.20)	<i>t_infant</i> \Rightarrow <i>gini</i>	-1.182 (0.19)
<i>gini</i> \Rightarrow <i>f_infant</i>	17.227*** (1.42E-65)	<i>gini</i> \Rightarrow <i>m_infant</i>	15.645*** (2.81E-54)	<i>gini</i> \Rightarrow <i>t_infant</i>	17.157*** (4.75E-65)
<i>f_14</i> \Rightarrow <i>gini</i>	-1.038 (0.23)	<i>m_14</i> \Rightarrow <i>gini</i>	-1.116 (0.21)	<i>t_14</i> \Rightarrow <i>gini</i>	-1.084 (0.22)
<i>gini</i> \Rightarrow <i>f_14</i>	5.190*** (5.62E-07)	<i>gini</i> \Rightarrow <i>m_14</i>	2.002* (0.05)	<i>gini</i> \Rightarrow <i>t_14</i>	2.946*** (0.00)
<i>f_49</i> \Rightarrow <i>gini</i>	-0.949 (0.25)	<i>m_49</i> \Rightarrow <i>gini</i>	-0.886 (0.26)	<i>t_49</i> \Rightarrow <i>gini</i>	-0.912 (0.26)
<i>gini</i> \Rightarrow <i>f_49</i>	6.704*** (6.92E-11)	<i>gini</i> \Rightarrow <i>m_49</i>	6.393*** (5.32E-10)	<i>gini</i> \Rightarrow <i>t_49</i>	6.144*** (2.52E-09)
<i>f_over</i> \Rightarrow <i>gini</i>	-1.014 (0.23)	<i>m_over</i> \Rightarrow <i>gini</i>	0.660 (0.32)	<i>t_over</i> \Rightarrow <i>gini</i>	-0.632 (0.32)
<i>gini</i> \Rightarrow <i>f_over</i>	-1.22 (0.18)	<i>gini</i> \Rightarrow <i>m_over</i>	0.386 (0.37)	<i>gini</i> \Rightarrow <i>t_over</i>	-1.019 (0.23)

***, **, * indicate rejection of the null hypothesis at the 1%, 5%, 10% levels of significance respectively. The values in parentheses show probability values of the variables.

Source: Author' estimations.

4. Conclusion

Rapid increases in income inequality in developed and developing countries over the past three decades have raised numerous concerns among both policymakers and economists. This concern is based on the worrisome of the possibility that income inequality might be causally related to population mortality. The strongest advocate of the income inequality hypothesis has been Richard Wilkinson (1992, 1996, 2000), who has put forward a variety of evidence, from individual, area, cross-country, and time-series data. Wilkinson (1997) stated that income inequality is the cause for lack of social capital, cohesion, social trust, self-esteem and a cause for disinvestment in social capital. Perceived deprivation, hopelessness, depression, isolation, insecurity and anxiety are all results of relative poverty and can trigger worse health and higher mortality. Thus, more equal countries have been shown to be more cohesive and better integration is known to benefit health (Hoffmann 2008).

Consequently, rapid growth in income inequality goes hand in hand with underinvestment, which will reap poor health outcomes in the future. The relative and even absolute deterioration in social and biological assets that is occurring in increasingly unequal societies can be expected to produce poor health outcomes in the future.

Many studies have highlighted the importance of the income inequality-population mortality relationship. This study investigates whether there is a causal relationship between income inequality and population mortality rate by age and gender of G7 countries over the period 1970-2010. First, we analyzed whether the variables contain a unit root using the Hadri and Kurozumi (2012) test. As a result of the test, we found that the all population mortality variables in level and gini coefficient in its first difference were stationary. Finally, the study analyzed whether there was a causality relationship between income inequality and population mortality using the Granger non-causality test for heterogeneous panel data developed by Dumitrescu and Hurlin (2012). As a result of the test, a unidirectional causality relationship was found from income inequality to population mortality rate, except for the ages above 50. No causal relationship was found between population mortality rate for the ages above 50 and income inequality. Hence, this study pushes a step further with the finding that the Gini index, used as a measurement of the income inequality to be significant predictors of prospected the both genders mortality rate for at ages 0-1, ages 1-14, and ages 15-49.

This study, which have differentiated from the other studies by examining the age and gender specific mortality rate-income inequality relationship and using the time period of 1970-2010 concludes that given the importance effect of health on productivity, output and income as well as sustainability of an aging society, redistributive policies aimed at reducing income inequality might lead to improved population health and so, there might be positive spillover effects in the society.

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