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Zusammenfassung / Abstract

This study examines the effects of adult and non-adult mortality on the long-run level of income in a heterogeneous dynamic and cross-sectionally dependent panel. Employing data for 20 countries between 1800 and 2010, it is found that (i) while non-adult mortality has no long-run effect on GDP per capita, reductions in adult mortality lead to statistically and economically significant increases in the long-run level of per capita income; (ii) there are no significant differences in the long-run effects of adult mortality and non-adult mortality on GDP per capita before and after the onset of the demographic transition; and (iii) mortality in middle adulthood has the greatest impact on economic development, whereas early adulthood mortality and mortality in later adulthood have little to no impact on the long-run level of per capita income.

JEL-Klassifikation / JEL-Classification: Life expectancy; Adult mortality; Non-adult mortality; Economic development; Heterogeneous panel data models, Cross-sectional dependence; Demographic transition

Schlagworte / Keywords: I15; O11; J11; C23

1. Introduction

One of the most controversial issues in the empirical macroeconomics literature concerns the role of mortality in determining economic development. Some studies, such as Knowles and Owen (1995) and Aghion et al. (2011), find a positive influence of improvements in life expectancy at birth on GDP per capita growth; others, including Bloom et al. (2014) and Hansen (2014), fail to uncover a robust causal effect of life expectancy at birth on GDP per capita growth; still other studies, such as Acemoglu and Johnson (2007, 2014) and Hansen and Lønstrup (2015), find a negative effect of life expectancy at birth on GDP per capita; finally, there is a study by Cervellati and Sunde (2011) suggesting that the effect of life expectancy at birth on economic growth is insignificant or negative before and positive after the demographic transition from high to low fertility and mortality.

In general, this literature focuses either on a summary measure for all age-specific mortality rates, such as life expectancy at birth, or on a measure of adult or infant/child mortality. Thus, the question of whether the effect of adult mortality on GDP per capita differs from that of non-adult mortality has received little attention in the empirical literature, despite its obvious economic importance.

In one of the few studies on this issue, Lorentzen et al. (2008) use cross-country annual data averaged over the period 1960–2000 for a sample of up to 94 countries and find in OLS and IV regressions a significant negative effect of both infant mortality (measured by the probability of dying before age 1 year) and adult morality (measured by the probability of surviving to age 60 years, conditional on surviving to age 15 years) on economic growth. However, when they estimate a simultaneous-equations system where the potential growth effects of infant and adult morality are modeled via possible indirect effects on physical capital investment, secondary schooling, and fertility, they find a statistically significant negative effect of adult mortality and an insignificant effect of infant mortality.

In the same paper, Lorentzen et al. (2008) also report fixed effects, random effects, and between estimates based on an unbalanced 10-year panel covering the period 1970–2000 for up to 19 Indian states. Consistent with the results from their simultaneous equations model, they find in these regressions that reductions in the adult mortality rate (of males aged 20–40) are significantly associated with increases in economic growth, whereas the infant mortality rate (per 1,000 live births) is not significantly associated with growth.

In a related study, Aghion et al. (2011) use the cross-sectional data from the study of Lorentzen et al. (2008) to estimate OLS regressions of time averaged growth rates on time averaged infant and adult mortality data as well as OLS regressions of time averaged growth rates on initial levels of infant and adult mortality. In all these regressions, which are based a sample of 94 countries for the 1960–2000 period, the coefficients of infant and adult mortality are negative. However, while the infant mortality variable is always statistically significant, they find in one regression that lower adult mortality is not significantly associated with higher growth.

Using 10-year panel regressions covering 28 OECD countries over the 1960-2000 period, Aghion et al. (2011) also find that life expectancy at birth is significantly positively related to growth in GDP per capita, whereas life expectancies at ages 40, 60, and 80 are insignificant when included together with life expectancy at birth. From this finding, Aghion et al. (2011, p. 21) conclude that "reducing mortality below age 40 is particularly growth-enhancing."

A final paper related to this issue is Acemoglu and Johnson (2007), who consider not only the impact of life expectancy at birth on GDP per capita but also that of life expectancy at age 20. Using long-difference specifications, where the change in the dependent variable between two time points is regressed on the change in the independent variables between the same two time points, they find in cross-sectional IV regressions for up to 45 countries for 1940–1980 and 1960–2000 that life expectancy at age 20 has a significant negative effect on GDP per capita. Interestingly, the magnitude of the coefficient on life expectancy at age 20 is, in absolute terms, much greater than the

magnitude of the coefficient on life expectancy at birth (–3.27 versus –1.32 in the base sample for 1940–1980). This finding implies that the growth-reducing effect of adult mortality is greater than the growth-reducing effect of infant mortality. The smaller estimated effect of life expectancy at birth is even consistent with the possibility that reductions in infant or child mortality promote growth.

Given the small number of studies examining how mortality at different age periods affects economic development, and the opposing findings of the current research, the purpose of this study is to provide additional evidence on the effects of mortality of adults and non-adults (including infants, children, and adolescents) on the long-run, or steady-state, level of GDP per capita.

Our study differs from the above studies in three ways. First, we use a panel dataset of annual observations for 20 countries between 1800 and 2010. This long sample period allows us to examine the long-run or full multiplier effects of adult and non-adult mortality on GDP per capita over time. In addition, since all countries in our sample went through their demographic transitions during the late 19th and early 20th centuries, our 211-year sample period allows us to analyze the mortality–income relationship during the demographic transition and to test whether the effects of adult and non-adult mortality differ before and after the onset of the demographic transition; this might be of particular interest to developing countries since many of these countries have not completed their demographic transitions.

Second, we not only present results for one measure of adult mortality (life expectancy at age 21) and one measure of non-adult mortality (the mortality rate up to age 21), but we also experiment with different age-specific mortality rates and life expectancies to improve our understanding of how changes in mortality at different ages contribute to economic development.

Third, we use recently developed panel time series methods that allow us to account for heterogeneous effects of adult and non-adult mortality on GDP per capita across countries. Suppose, for example, that increased life expectancy increases schooling since the returns to

investment in education increase over a longer working life. To the extent that the impact of schooling on economic growth varies across countries, depending on country-specific factors such as the quality of schooling and the ability of countries to use schooling productively,[1] we should expect that the impact of mortality on economic growth is heterogeneous as well. It is therefore important to account for cross-country heterogeneity; otherwise, the estimates of the long-run effects might be inconsistent (see, e.g., Pesaran and Smith, 1995).

In addition, the methods we use allow us to test whether adult and non-adult mortality are weakly exogenous or "long-run forcing" for GDP per capita and to obtain consistent parameter estimates even in the presence of dynamic feedback effects from GDP per capita. Thus, our empirical strategy has the advantage that we do not need to rely on instrumental variables, which are difficult, if not impossible, to find. Moreover, econometric theory suggests that instrumental variables (IV) estimators are generally inconsistent when slope coefficients differ across panel units (see, e.g., Pesaran and Smith, 1995).

Finally, the estimation methods we use allow us to adequately control for cross-sectional dependence due to common shocks or spillovers among countries at the same time. The conventional approach to account for common shocks is to include time dummies. However, the use of time dummies assumes that all panel units react identically to common shocks, and may therefore be ineffective in eliminating cross-sectional dependence when countries respond differently to common shocks. Our approach does not make this assumption, and thus allows for cross-country differences in the impact of unobserved common factors.

To preview our main results, we find that (i) while non-adult mortality has no long-run effect on GDP per capita, reductions in adult mortality lead to statistically and economically significant increases in the long-run level of per capita income; (ii) there are no significant

[1] Rogers (2008), for example, argues that countries vary greatly in their ability to use schooling productively and finds that countries with higher levels of corruption, black market premia, and out migration of educated workers (brain drain) exhibit a lower effect of schooling on growth.

differences in the long-run effects of adult mortality and non-adult mortality on GDP per capita before and after the onset of the demographic transition; and (iii) per capita income responds mainly to changes in mortality at middle adult ages, whereas early adulthood mortality and mortality in later adulthood have little to no effect on the long-run level of per capita income.

The rest of this paper is organized as follows. In Section 2, we discuss the potential theoretical effects of adult and non-adult mortality on the long-run level of income. Section 3 develops the basic empirical model, discusses the empirical strategy, and describes the data. The econometric implementation and the estimation results are presented in Section 4, and Section 5 concludes the paper.

2. Potential effects of adult and non-adult mortality on the long-run level of income

2.1. Basic theoretical framework

There are several mechanisms by which adult and non-adult mortality may affect the long-run level of income. We illustrate these mechanisms by considering a standard Cobb-Douglas production function of the form

$$Y = K^{\alpha} H^{\beta} (AL)^{1-\alpha-\beta}, \tag{1}$$

where Y is output, K and H are stocks of physical and human capital, A denotes the level of technology or total factor productivity (TFP), and L denotes labor. As in the standard Solow (1956) model, L and A are assumed to grow exogenously at rates n and g per period t:

$$L = L(0)e^{nt}, \tag{2}$$

$$A = A(0)e^{gt}, \tag{3}$$

where $L(0)$ and $A(0)$ are the initial labor force and the initial level of technology, respectively. Defining $\tilde{k}$ as the stock of capital per effective unit of labor, $\tilde{k} = K / AL$, and $\tilde{y}$ as the level of output per effective unit of labor, $\tilde{y} = Y / AL$, the evolution of $\tilde{k}$ is governed by

$$\dot{\tilde{k}} = s_k \tilde{y} - (n + g + \delta)\tilde{k} , \tag{4}$$

where s_k is the investment rate in physical capital and δ is the rate of depreciation. Following Mankiw et al. (1992), we assume that a similar equation holds for the stock of human capital per effective unit of labor:

$$\dot{\tilde{h}} = s_h \tilde{y} - (n + g + \delta)\tilde{h} , \tag{5}$$

where s_h is the investment rate in human capital. Solving equations (4) and (5) for the steady state, substituting them into the production function, and taking logs, gives the steady-state or long-run level of income per capita (see, e.g., Mankiw et al., 1992):

$$\ln(y) = \ln A(0) + gt - \frac{\alpha + \beta}{1 - \alpha - \beta} \ln(n + g + \delta) + \frac{\alpha}{1 - \alpha - \beta} \ln(s_k) + \frac{\beta}{1 - \alpha - \beta} \ln(s_h) . \tag{6}$$

This equation shows that the long-run level of income per capita depends negatively on the growth rate of the population, n, and positively on the growth rate of technology (or TFP), g, the investment rate in physical capital, s_k, and the investment rate in human capital, s_h. In the following, we discuss how mortality of adults and non-adults may affect these mechanisms.

2.2. Population growth

If we ignore migration, the population growth rate is equal to the birth rate minus the death rate. A decline in mortality (of both adults and young people) therefore has a direct positive effect on population growth, since more people survive at each point of time. In addition to this direct effect, mortality may also indirectly affect population growth through effects on the birth rate.

One possible indirect effect is that reduced mortality increases the number of women of childbearing age and thus increases the number of births (see, e.g., Acemoglu and Johnson, 2007). The condition for this effect to occur is that the mortality rate of women of childbearing age declines. The implication is that to the extent that reductions in adult mortality coincide with reductions in mortality of people of parental age,[2] reductions in adult mortality may lead to faster increases in population growth than equivalent reductions in non-adult mortality.

Other indirect effects may result from the influence of mortality on fertility behavior. This influence may occur through several mechanisms. First, if parents gain utility from the number of surviving children, and choose the optimal number of surviving children in the face of a constraint on the total amount of time that can be devoted to child-raising and labor-market activities, then declines in child mortality reduce the number of births required to produce a target number of children.[3] Thus, in the case that parents are motivated to "replace" a child's death with a new child, reductions in child mortality necessarily result in reductions in total fertility (births per woman), but have no effect on net fertility (the number of surviving children) and hence population growth (see, e.g., Galor, 2012; Herzer, et al., 2012). The point is that in this case, where it is assumed that the loss of a child leads to a subsequent replacement of that child, only declines in child mortality reduce total fertility and thus do not affect population growth; declines in adult mortality, in contrast, do not exert such an effect on fertility and thus tend to increase population growth.[4]

Second, if there is uncertainty about the number of surviving children, and if parents want to avoid the possibility of having too few surviving children at the end of their reproductive age, then a precautionary demand for children arises, an effect also known as "hoarding effect." That is, in a high mortality environment, parents insure ex ante against the possible death of a child by having

[2] According to the CIA World Factbook (available at https://www.cia.gov/library/publications/the-world-factbook), the mean age at first birth for women was, for example, 30.5 years in Australia in 2006, 28.1 years in England and Wales in 2012, 30.4 years in Switzerland in 2012, and 25.6 years in the United States in 2011.

[3] In the influential model of Barro and Becker (1989), where parents are altruistic toward their children, declines in child mortality reduce the expected cost of producing each surviving child by reducing the average number of births needed to get a survivor.

[4] To the extent that parents fail to fully replace losses, reduced child mortality also tends to increase population growth.

more births than desired to ensure a certain number of surviving children (see, e.g., Kalemli-Ozcan, 2002). Given that this precautionary demand decreases with decreasing mortality (and hence with decreasing uncertainty), declining mortality can have a strong negative impact on net fertility, and thereby reduce population growth. Lorentzen et al. (2008) argue that because deceased young children can be more easily replaced by subsequent births than deceased adult children, the hoarding effect is more likely to occur when adult mortality (rather than child mortality) is high. In the presence of a hoarding motive, it could therefore be that a reduction in adult mortality exerts a negative effect on fertility and hence population growth.

Similarly, and third, if parents regard the number of children and the expected lifetime of each child as substitutes, as pointed out by Soares (2005), then rising adult longevity (or declining adult mortality) may reduce fertility. In a scenario where the utility of parents depends not only on the number of children, but also on the lifetime that each child will enjoy as an adult, both reductions in child mortality and increases in adult longevity may therefore lead to reductions in fertility.

Finally, and discussed in more detail below, gains in adult longevity raise the rate of return on investments in a child's human capital, which may induce parents to make trade-offs between quantity and quality of children. In this case, increases in adult longevity may increase educational attainment and reduce fertility (and hence population growth) (see, e.g., Soares, 2005).[5]

2.3. Human capital investment

Most theoretical models of the relationship between mortality and economic growth assume that human capital investment is a key channel through which mortality affects growth. Central to this literature is the idea, which dates back to Ben-Porath (1967), that gains in adult longevity increase the horizon over which investments in schooling will be paid off; an increase in the probability of

[5] Hazan and Zoabi (2006), however, argue that increases in longevity may also lead to an increase in the number of children because greater longevity of children increases not only the returns to quality but also the returns to quantity, which in turn may mitigate the incentive to invest more in the children's education.

surviving in the future therefore raises the incentive for individuals to invest in their own education or in the education of their children (see, e.g., Kalemli-Ozcan et al., 2000; Soares, 2005).[6] According to this idea, improvements in education are thus due to improvements in adult mortality rather than non-adult mortality.

Another central idea in this literature, which goes back to Becker (1960), is that parents make trade-offs between the quantity and the quality of their children within their given budget constraints. This implies that, by reducing parental costs of educating each surviving child, a decline in child mortality may lead to greater investments in children's human capital (provided that the decline in child mortality leads to a decline in births and the number of surviving children) (see, e.g., Kalemli-Ozcan, 2002). Similarly, as discussed in the above subsection, an increase in the adult longevity of children may in theory encourage parents to invest more resources in fewer children if parents derive utility from both the number of children they have and the longevity of their children (see, e.g., Soares, 2005).

A potentially important point is that these human capital investment decisions are made by parents. But if parents die, they cannot invest in their children. Thus, lower adult mortality also reduces the number of orphans, who receive less schooling than children with living parents (see, e.g., Case et al., 2004).

2.4. Physical capital investment

The standard Modigliani life-cycle model of savings and consumption (as described, for example, in Modigliani, 1971) suggests that an increased probability of surviving past the age of effective labor

[6] Hazan (2009) criticizes this idea by first documenting that for cohorts of American men born in 1840-1970, labor input declined despite the large gains in life expectancy. He then argues that because a rise in the lifetime labor supply is a necessary implication of the Ben-Porath type model that he examines, gains in life expectancy could not have caused human capital accumulation (and hence growth) via the Ben-Porath mechanism. However, more recent studies, such as those by Hansen and Lønstrup (2012), Cervellati and Sunde (2013), and Yasui (2016), show that an increase in adult longevity may theoretically increase education without an increase in lifetime labor supply.

force participation may increase savings rates in the long run. In other words, improvements in adult longevity create a greater need for people to save for their retirement.

A similar argument is that adults who expect to die early are unlikely to take actions that generate long-term benefits and short-term costs, such as investing in physical (and human) capital and saving for the future (see, e.g., Lorentzen et al., 2008). Lower adult mortality may thus increase the incentives to accumulate physical capital.

2.5. TFP growth

Bar and Leukhina (2010) develop an endogenous growth model in which decreases in adult mortality contribute to TFP growth by improving the transmission of knowledge across generations and encouraging innovation. Similarly, Lucas (2009, p.8) argues in his model of endogenous technological change that "a productive idea needs to be in use by a living person to be acquired by someone else, so what one person learns is available to others only as long as he remains alive. If lives are too short […], sustained growth at a positive rate is impossible."

Summarizing, it can be said that mortality may theoretically affect the long-run level of income through several channels, including population growth, human capital investment, physical capital investment, and TFP growth. While several theoretical arguments suggest that lower mortality tends to increase human capital accumulation, physical capital accumulation, and TFP growth, mortality can have both positive and negative effects on population growth (depending on whether and to what extent mortality reductions cause fertility reductions). Thus, the net long-run effect of mortality on economic development is theoretically ambiguous, and thus may be positive, negative or zero. Overall, however, the theoretical literature suggests that the net long-run effect of declining (young) adult mortality is more likely to be positive than the net long-run effect of declining non-adult mortality.

3. Model, strategy, and data

3.1. Basic estimating equation

This section presents the basic empirical model used to examine the long-run effects of non-adult mortality and adult mortality on the level of economic development. Given that static models are known to yield biased estimates when the underlying true model has a dynamic structure (see, e.g., Banerjee et al., 1986; Egger and Pfaffermayr, 2005), the basis of our analysis is an autoregressive distributed lag (ARDL) dynamic panel specification of the form

$$\ln(y_{it}) = \beta_{0i} \ln(y_{it-1}) + \beta_{1i} M_{it} + \beta_{2i} M_{it-1} + c_i + \mu_{it}, \qquad \mu_{it} = \lambda_i f_t + \varepsilon_{it}, \tag{7}$$

where i and t are country and time indices; $\ln(y_{it})$ is level of economic development (of country i in year t), measured by the log of real GDP per capita; and M_{it} represents two different measures of mortality:

(i) the non-adult mortality rate, $MORT\_21_{it}$, defined as the proportion of people not expected to survive to age 21, and

(ii) adult life expectancy, $LIFE\_21_{it}$, defined as the average number of years that a 21-year old individual is expected to live if current age-specific mortality rates continue to apply.

While the former depends on the mortality rates for every age below 21 (hence incorporating infant mortality and all other mortality rates up to age 21), the latter depends on the mortality rates for every age above 21 (hence incorporating life expectancies at all later ages). Thus, we define "adult" as age 21 or over,[7] but in robustness checks, we use alternative age cut-offs. More specifically, we experiment with different age-specific mortality rates and life expectancies, as discussed in the beginning of this paper.

It should be noted that we estimate the effects of non-adult mortality and adult mortality in separate and joint regressions for the two variables. We also note that the natural logarithms of

[7] We choose to use 21 as the age cut-off for adulthood because it was the legal age of adulthood (or majority) in most of our sample countries during most years of our sample period. Another reason for this choice is that 21 is a more realistic age at which individuals make economically relevant decisions (for example, about labor supply, savings, and family planning) than 18, the current legal age of majority in most countries.

$MORT\_21_{it}$ and $LIFE\_21_{it}$ are too collinear to include the log of non-adult mortality and the log of adult mortality in the same regression model. Therefore, following Lorentzen et al. (2008) and Hansen (2014) (and others), we do not log-transform the mortality variables.

Unlike some previous studies (see, e.g., Knowles and Owen, 1995; Caselli et al., 1996), we do not include variables for population growth, human capital, and/or physical capital in the regression, given the discussion in the previous section. If we included these variables, the estimates of the impact of adult and non-adult mortality on GDP per capita would leave out any effects operating through these three potential channels. Following (among others) Acemoglu and Johnson (2007, 2014) and Bloom et al. (2014), we thus use a parsimonious specification that aims to capture the net or total effect of (adult and non-adult) mortality on the long-run level of income.

While the coefficients β_{1i} and β_{2i} represent the short-run, immediate effects of changes in M_{it} in periods t–1 and t on $\ln(y_{it})$ in period t, the coefficient on the lagged dependent variable, β_{0i}, captures the extent to which short-run effects have long-run consequences—in our case, for the level of economic development. Our focus is on the long-run average (multiplier) effect of M_{it} on $\ln(y_{it})$, which can be calculated from the means of the individual country coefficients as follows (see, e.g., Pesaran and Smith, 1995):

$$\beta = \frac{\overline{\beta}_1 + \overline{\beta}_2}{1 - \overline{\beta}_0}, \tag{8}$$

where $\overline{\beta}_0$, $\overline{\beta}_1$, and $\overline{\beta}_2$ are the means of the coefficients β_{0i}, β_{1i}, and β_{2i}, respectively.[8]

The subscript i on these coefficients indicates that we allow all coefficients to vary across countries—by estimating separate time series regressions for each country and then averaging the individual country coefficients. The rationale for this approach is that standard (OLS, IV, and

[8] The conventional approach in panel studies is to calculate the long-run average coefficients by dividing the (sums of the) pooled short-run coefficients by one minus the pooled coefficient on the lagged dependent variable. To be consistent with this approach, we calculate the long-run average coefficients by dividing the (sums of the) average short-run coefficients by one minus the average coefficient on the lagged dependent variable. Alternatively, the average long-run coefficients can be calculated as the averages of the individual long-run coefficients, $\beta = N^{-1}\sum_{i=1}^{N}(\beta_{1i} + \beta_{2i})/(1 - \beta_{0i})$ (see, e.g., Pesaran and Smith, 1995). We present results based on this approach in Section 4.4.

GMM) dynamic panel estimators, which impose slope homogeneity restrictions across *i*, yield inconsistent and potentially misleading estimates of the average slope coefficients when the coefficients are heterogeneous (see, e.g., Pesaran and Smith, 1995).

As the above paragraph implies, the c_i are country-specific intercepts of individual time series regressions; these capture the effects of any country-specific omitted factors that are relatively stable over time, such as geography and culture.

The term $\lambda_i f_t$ denotes country-specific effects of unobserved common factors f_t that vary over time. Common factors can be a combination of "strong" factors representing global shocks and "weak" factors representing spatial spillovers (see, e.g., Chudik et al., 2011; Chudik and Pesaran, 2015a). Relevant examples of the former are climate change (and associated weather events), global financial crises, worldwide wars, global technological progress (and the associated discovery of medical knowledge), and global epidemics. Examples of the latter include the spread of diseases and cross-border pollution between (a limited number of) countries. These latent common factors induce cross-sectional error dependence and may lead to inconsistent regression coefficient estimates if they are correlated with the explanatory variables.[9]

Following the dynamic common correlated effects (CCE) mean group estimation approach of Chudik and Pesaran (2015b), we employ cross-sectional averages of current and lagged values of the variables in our dynamic model to proxy for the unobserved common factors. The cross-sectionally augmented ARDL (CS-ARDL) representation of equation (7) is given by

$$\ln(y_{it}) = \beta_{0i}\ln(y_{it-1}) + \beta_{1i}M_{it} + \beta_{2i}M_{it-1} + \sum_{\ell=0}^{p} g_{1i\ell}\overline{\ln(y_{t-\ell})} + \sum_{\ell=0}^{p} g_{2i\ell}\overline{M_{t-\ell}} + c_i + \varepsilon_{it}, \quad (9)$$

where $\overline{\ln(y_t)} = N^{-1}\sum_i^N \ln(y_{it})$ and $\overline{M_t} = N^{-1}\sum_i^N M_{it}$ are the cross-sectional averages of $\ln(y_{it})$ and M_{it}, and p is the number of lags of the cross-section averages. Monte Carlo simulations suggest that

[9] The presence of weak factors does not affect the consistency of conventional panel data estimators, but the standard errors are biased in the presence of weak factors (see, e.g., Chudik and Pesaran, 2015a).

the effects of both strong and weak factors tend to be eliminated by the use of (weighted) cross-sectional averages of the dependent and independent variables as additional regressors (see, e.g., Pesaran and Tosetti, 2011; Chudik et al, 2011), which is the basic idea of all (static, dynamic, homogeneous and heterogeneous) CCE estimators. Monte Carlo evidence also suggests that CCE estimators perform better under cross-sectional dependence than alternative estimators (see, e.g., Kapetanios et al., 2011). A specific and important advantage of the dynamic CCE or CS-ARDL estimator over the static CCE estimator is that it is robust to the presence of weakly exogenous regressors and thus to short-run feedback effects, in our case from GDP per capita to mortality.

It should be noted that Chudik and Pesaran (2015b) prove the consistency of the dynamic CCE mean group estimator under the assumption of stationarity of the data. However, Kapetanios et al. (2011) show that the static CCE (pooled and mean group) estimators are consistent and produce correctly sized tests when the data are non-stationary and cointegrated. Coakley et al.'s (2005, 2006) results even suggest that the static CCE mean group estimator produces correct standard errors and consistent estimates of the average coefficients when the variables are non-stationary and not cointegrated, i.e. when the residuals (ε_{it}) are non-stationary. Similarly, Chudik et al. (2016) find that even if the residuals have unit roots, the dynamic CCE mean group estimator is consistent and the empirical size of the *t*-test based on the dynamic CCE mean group estimator is close to the nominal 5% level. And finally, Pesaran and Shin (1999) show that the conventional time series ARDL estimator is valid even if the variables are $I(1)$ (i.e. non-stationary in levels but stationary in first differences), provided that they are cointegrated.

The basic idea behind cointegration is that two or more non-stationary variables may be regarded as defining a long-run equilibrium relationship if they move together in the long run, even though they may drift apart in the short run. More specifically, two or more non-stationary variables are said to be cointegrated if some linear combination of them is stationary (see, e.g., Engle and

Granger, 1987).[10] Thus, the cointegration property is invariant to model extensions, which is in contrast to regression analysis where one new variable can alter the existing estimates dramatically (see, e.g., Juselius, 2006; Lütkepohl, 2007). The implication from this is that if two or more variables are cointegrated, no additional variables are required to obtain unbiased estimates of the long-run parameters. Another important implication of cointegration is the existence of long-run Granger causality, as discussed in more detail below.

To test for cointegration, the CS-ARDL representation above can be rewritten as a cross-sectionally augmented conditional error correction model (ECM) as follows:[11]

$$\begin{aligned}\Delta \ln(y_{it}) = {} & b_{1i} \ln(y_{it-1}) + b_{2i} M_{it-1} + b_{3i} \Delta M_{it} \\ & + \phi_{1i} \overline{\ln(y_{t-1})} + \phi_{2i} \overline{M_{t-1}} \\ & + \sum_{\ell=0}^{p-1} \phi_{3i\ell} \overline{\Delta \ln(y_{t-\ell})} + \sum_{\ell=0}^{p-1} \phi_{4i\ell} \overline{\Delta M_{t-\ell}} + c_i + \varepsilon_{it}\end{aligned} \quad , \tag{10}$$

where b_{1i} is the error correction coefficient, which represents the speed of adjustment to the long-run equilibrium relationship between the level of economic development and the level of mortality (of adults and younger persons), and should be significantly negative if there is a long-run or cointegrating relationship between $\ln(y_{it})$ and M_{it}; Δ is the first difference operator (e.g. $M_{it} - M_{it\text{-}1}$); and b_{3i} ($= \beta_{1i}$) represents the short-run (immediate) effect of a change in M_{it} on GDP per capita.[12]

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[10] As defined by Engle and Granger (1987), two variables are cointegrated [of order (1, l)] if each variable individually is stationary in first differences (integrated of order l), but some linear combination of the variables is stationary in levels (integrated of order 0). The conventional concept of cointegration between variables with stochastic trends is thus defined as the existence of a *linear* relationship between these variables over time that produces stationary residuals. Evidence of cointegration (in the usual linear sense) therefore implies the absence of significant nonlinearities in the estimated relationships, whereas a failure to find (linear) cointegration does not necessarily mean that there is no (nonlinear) long-run relationship among the variables (see, e.g., Kanas, 2005).

[11] To see this, consider a simple ARDL model of the form

$$\ln(y_{it}) = \beta_{0i} \ln(y_{it-1}) + \beta_{1i} M_{it} + \beta_{2i} M_{it-1} + c_i + \varepsilon_{it} .$$

To reformulate the model as a conditional ECM, take the difference of $\ln(y_{it})$,

$$\Delta \ln(y_{it}) = (\beta_{0i} - 1) \ln(y_{it-1}) + \beta_{1i} M_{it} + \beta_{2i} M_{it-1} + c_i + \varepsilon_{it} ,$$

and add and subtract $\beta_1 M_{it\text{-}1}$ from the right hand side,

$$\Delta \ln(y_{it}) = (\beta_{0i} - 1) \ln(y_{it-1}) + \beta_{1i} \Delta M_{it} + (\beta_{1i} + \beta_{2i}) M_{it-1} + c_i + \varepsilon_{it} .$$

Regrouping gives

$$\Delta \ln(y_{it}) = b_{1i} \ln(y_{it-1}) + b_{2i} M_{it-1} + b_{3i} \Delta M_{it} + c_i + \varepsilon_{it} ,$$

where $b_{1i} = (\beta_{0i} - 1)$, $b_{2i} = (\beta_{1i} + \beta_{2i})$, and $b_{3i} = \beta_{1i}$.

[12] It should be noted here that equation (10) implies the following: If there is a long-run (cointegrating) relationship between the level variables, then pooled cross-country, time series growth regressions that omit the lagged levels of the variables (or the lagged residual from the cointegrating equation) are misspecified.

Given that equation (10) is a reparameterization of equation (9), the long-run average effect of the different measures of mortality on GDP per capita can also be calculated from equation (10). The formula is:

$$\beta = \frac{\bar{b}_2}{|\bar{b}_1|}, \tag{11}$$

where $\bar{b}_2$ and $\bar{b}_1$ are, respectively, the means of the coefficients b_{2i} and b_{1i}.

Following Eberhardt and Presbitero (2015) and Cavatorta and Smith (2017), we use the CS-ARDL model in error correction form for our analysis. As mentioned above, the advantage of using equation (10) rather than (9) to estimate the long-run average effect of M_{it} on $\ln(y_{it})$ is that equation (10) can also be used to test for cointegration—by testing the significance of the error correction coefficient, H_0: $b_{1i} = 0$.

A second and independent reason for using equation (10) is that recent studies, such as Aghion et al. (2011), Bloom et al. (2014) and Hansen and Lønstrup (2015), use a similar specification (but do not allow for slope heterogeneity and country-specific effects of unobserved common factors and also do not use panel data with a long time series dimension) to estimate the effect of life expectancy at birth on economic growth. Thus, the use of equation (10) allows a direct comparison of our results with other recent studies.

3.2. Empirical strategy

The above discussion implies that our analysis involves several steps. In the following subsections, we discuss these steps in detail.

3.2.1. Step 1

Cointegration implies the existence of a meaningful long-run relationship between two or more non-stationary variables. In a first step, we test for non-stationarity using the cross-sectionally augmented panel unit root test (CIPS) proposed by Pesaran (2007). This test, which is based on an

average of individual country augmented Dickey Fuller (ADF) tests, follows the CCE approach by augmenting the ADF regressions with cross-sectional averages of the variables to eliminate the cross-sectional dependence.

As part of this first step, we also test for the presence of cross-sectional dependence in our data by applying the cross-sectional dependence (CD) test of Pesaran (2004) to each of the variables in our model ($\ln(y_{it})$, $MORT\_21_{it}$, and $LIFE\_21_{it}$).

In addition, we test for cointegration. As discussed above, some studies suggest that hypothesis tests based on CCE mean group procedures have rejection frequencies that are close to the nominal size even if the variables are non-stationary and not cointegrated. If inference based on the CCE mean group estimator is valid in the presence of non-stationarity, then, following Eberhardt and Presbitero (2015), we can apply the CCE mean group estimator to equation (10) to test the null hypothesis of no cointegration between $\ln(y_{it})$ and M_{it} using the standard error (or *t*-value) of the CCE mean group estimate of the error correction coefficient. Given, however, that the validity of this test has not been formally established, it is used here—in Step 2—to informally test for cointegration (when we estimate equation (10)).

In this first step of our analysis, we formally test for cointegration between y_{it} and M_{it} using the four panel cointegration tests of Westerlund (2007), denoted P_τ, P_α, G_τ, and G_α. The former two are pooled tests and the latter two are group mean tests. All these tests test the significance of the error correction coefficient in a conditional ECM similar to that in equation (10). The main difference between the model on which the Westerlund tests are based and the cross-sectionally augmented ECM given by equation (10) is that the former does not include proxies for unobserved common factors in the form of cross-sectional averages. To account for cross-sectional dependence in the residuals, we conduct the cointegration tests using Westerlund's cross-sectional dependence robust (bootstrapped) *p*-values.[13]

[13] An alternative would be the use of the ECM-based panel cointegration tests of Gengenbach et al. (2016). However, these tests are designed for balanced panels and therefore cannot be applied to our unbalanced panel data set.

3.2.2. Step 2

The second step is to estimate the average *b* coefficients in equation (10) and the long-run average effect of the different measures of mortality on the level of development using the CS-ARDL mean group estimator. In this step, we also test whether the average error correction coefficient in equation (10) is significantly different from zero, based on the standard error of the mean group estimator. As discussed above, this serves as an additional, informal test for cointegration.

In this context, it should be noted that for the dynamic CCE mean group estimator to be valid, the number of lags of the cross-section averages must be sufficiently large (without being too large).[14] Chudik and Pesaran (2015b) suggest as a rule of thumb that the number of lags should be equal to the integer part of $T^{1/3}$. Given this rule, and given that our maximum number of observation per unit is 211, we set $p = 5$ ($p - 1 = 4$) in equation (10). To check the robustness of our results, we also estimate specifications with $p = 4$.

We also note here that equation (10) is based on an ARDL model with one lag of each variable and that this model can be extended to include further lags of the variables, so that the ECM version includes lagged differences of the variables. The point is that ARDL estimates of the long run coefficients may be sensitive to the lag structure chosen. Therefore, we also estimate specifications of equation (10) with different lag structures.

In addition to the CS-ARDL approach represented by equation (10), we adopt the cross-sectionally augmented distributed lag (CS-DL) approach of Chudik et al. (2016) to check the robustness of the results. The CS-DL mean group estimator is in our case based on the following regression:

$$\ln(y_{it}) = \beta_i M_{it} + \sum_{\ell=0}^{p-1} \delta_{i\ell} \Delta M_{it-\ell} + \omega_{1i} \overline{\ln(y_t)} + \sum_{\ell=0}^{p} \omega_{2i\ell} \overline{M_{t-\ell}} + c_i + e_{it}. \quad (12)$$

[14] The dynamic CCE approach yields inconsistent estimates if the number of lags of the cross-sectional is too large (Chudik and Pesaran, 2015b).

where we use $p = 4$ and $p = 5$, as in the CS-ARDL regressions.

Chudik et al. (2016) show through Monte Carlo simulations that, unlike the CS-ARDL estimator, the CS-DL estimator is robust to the specification of the number of lags and serial correlation in the errors. However, while the CS-ARDL approach is robust to feedback effects from lagged values of the dependent variable to current values of the regressors, the CS-DL estimator of the long-run effect is biased in the presence of such feedback effects. Overall, the Monte Carlo evidence presented in Chudik et al. (2016) suggests that in large T panel datasets, such as the panel dataset used for our study, the CS-ARDL mean group estimator performs better than the CS-DL mean group estimator. Therefore, and because recent studies use a similar specification as in equation (10) (as discussed above), we prefer the former over the latter.

We note here that, in this step, we not only test the sensitivity of our long-run estimates to the use of different lags and the use of an alternative estimator (the CS-DL mean group estimator), but we also conduct several other robustness checks, including exploring potential outliers, a possible structural change in the slope coefficients, different samples, and different age-specific mortality rates and life expectancies. We discuss these robustness checks in more detail in Section 4.3.

Finally, it should be mentioned that we apply the CD test of Pesaran (2004) to the residuals of all models to test whether our results are contaminated by cross-section dependence.

3.2.3. Step 3

Cointegration implies long-run causality of at least one of the variables in the long-run relationship (see, e.g., Granger, 1988; Granger and Lin, 1995).[15] So far we have assumed that mortality is a long-run cause of economic development in the sense that changes in M_{it} have a direct effect on the

[15] Our definition of causality is based on the assumption is that the cause occurs before the effect, so that the "arrow of time" can be used to help distinguish between cause and effect. Of course, this assumption rules out the possibility that (correct) expectations regarding future levels of GDP per capita affect current levels of mortality. However, we consider this possibility unlikely because individual mortality depends mainly on current and past, rather than expected, economic conditions, besides the fact that it is difficult to predict in advance (and with accuracy) the level of GDP per capita.

long-run level of ln(y_{it}), whereas changes in ln(y_{it}) do not affect the long-run levels of non-adult mortality and adult mortality (controlling for the influence of unobserved common factors). In other words, non-adult mortality and adult mortality are assumed to be weakly exogenous.

If this assumption is correct, there is no need to use instrumental variables, as mentioned in the beginning of this paper. This point is worth emphasizing because it is well known that IV regressions may lead to spurious results when the instruments are weak or invalid and it is also well known that it is difficult (and sometimes even impossible) to find appropriate instruments for macroeconomic variables (see, e.g., Clemens et al., 2012). In addition, it is established, although less well known, that consistent IV estimation is not possible when the slope coefficients differ across cross-section units (see, e.g., Pesaran and Smith, 1995).

In order to test whether our mortality variables can be treated as weakly exogenous (or "forcing"), we employ a two-step procedure, as is standard practice in the non-stationary panel literature (see, e.g., Canning and Pedroni, 2008; Herzer, 2013; Eberhardt and Teal, 2013). In the first step, we estimate the long-run coefficients for each country (using equation (10)) to construct the error correction term $ec_{it} = \ln(y_{it}) - (\hat{c}_i + \hat{\beta}_{it} M_{it})$. In the second step, we include the lagged error correction term in a panel vector error correction model (VECM) of the form

$$\begin{aligned} \Delta \ln(y_{it}) &= c_{1i} + \alpha_{1i} ec_{it-1} + \sum_{j=1}^{k} \varphi_{11ij} \Delta \ln(y_{it-j}) + \sum_{j=1}^{k} \varphi_{12ij} \Delta M_{it-j} + e_{it}^{y} \\ \Delta M_{it} &= c_{2i} + \alpha_{2i} ec_{it-1} + \sum_{j=1}^{k} \varphi_{21ij} \Delta \ln(y_{it-j}) + \sum_{j=1}^{k} \varphi_{22ij} \Delta M_{it-j} + e_{it}^{M} \end{aligned}, \tag{13}$$

where k is the number of lags of the differenced series, ec_{it-1} represents the deviation from the equilibrium, and the adjustment coefficients α_{1i} and α_{2i} capture how ln(y_{it}) and M_{it} respond to deviations from the equilibrium. The Granger Representation Theorem (Engle and Granger, 1987) implies that for a long-run equilibrium relationship to exist between ln(y_{it}) and M_{it} at least one of the adjustment coefficients must be nonzero. If the adjustment coefficient in the Δln(y_{it}) equation is nonzero, $\alpha_{1i} \neq 0$, then the null hypothesis of weak exogeneity is rejected for ln(y_{it}). If the

adjustment coefficient in the ΔM_{it} equation is nonzero, $\alpha_{2i} \neq 0$, then the null hypothesis of weak exogeneity is rejected for M_{it}. Hall and Milne (1994) show that weak exogeneity in a cointegrated system is equivalent to the notion of long-run non-causality (see also Granger, 1988).[16] Thus, if (and only if) $\alpha_{1i} \neq 0$, then M_{it} has a causal impact on $\ln(y_{it})$ in the long run; if (and only if) $\alpha_{2i} \neq 0$, then M_{it} has a long-run causal impact on $\ln(y_{it})$; if both α_{1i} and α_{2i} are nonzero, then long-run Granger causality runs in both directions (see also Canning and Pedroni, 2008).

Following Canning and Pedroni (2008), we estimate the VECM separately for each country and then use the lambda-Pearson statistic to test the null hypotheses that $\alpha_{1i} = 0$ and $\alpha_{2i} = 0$ for all countries. The lambda-Pearson statistic, also commonly referred to as the Fisher statistic, is defined as

$$P_{\lambda} = -2\sum_{i}^{N} \log(p_i), \tag{14}$$

where p_i is the p-value of the t-test of the null hypothesis of no long-run causal effect for country i. The lambda-Pearson (Fisher) statistic is distributed as χ^2 with $2 \times N$ degrees of freedom.

Following (among others) Eberhardt and Teal (2013) and Eberhardt and Presbitero (2015), we test for weak exogeneity using the CCE approach. We thus augment equation (13) with cross-sectional averages of the dependent variables and the regressors, including ec_{it-1}, to control for any causal effects between $\ln(y_{it})$ and M_{it} that are due common factors. It should be noted that the application of the CS-ARDL estimation procedure in the first-step regression allows us to account for unobserved common factors in both steps.

3.3. Data

[16] The concept of long-run (Granger) causality was introduced by Granger (1988) and further developed by Granger and Lin (1995). It is to be distinguished from the more familiar notion of "Granger causality," which (in the usual sense) refers to short-run forecastability and does not account for long-run causality through the error correction term in an error correction model.

We now describe the data. Real GDP per capita is measured in millions of 1990 international Geary-Khamis dollars. The source is the Maddison Project Database of Bolt and van Zanden (2014) (available at http://www.ggdc.net/maddison/maddison-project/data.htm), which is an updated version of the original Maddison (2004) database and contains complete annual time series between 1800 and 2010 of real GDP per capita for over 100 countries.

Our mortality rates are constructed by subtracting survival rates (i.e., the percentage of each birth cohort expected to survive to age x) from the Human Mortality Database (available at http://www.mortality.org) from 100%. Thus, we define the mortality rate up to age x in year t as the expected value of the proportion of the birth cohort that will not survive to age x. In our main analysis, we set $x = 21$, and in our robustness checks, we experiment with several other values for x (as discussed above).

Life expectancy at age x is defined in the usual sense as the expected number of years that a person aged x in the given calendar year will live if current age-specific mortality rates continue to apply.[17] We use life expectancy at age 21 as our main measure of adult mortality; other age-specific life expectancies are used in the robustness checks. Data on life expectancies are also from the Human Mortality Database. This database covers 38 (or areas) countries over different time periods.

Based on these sources, we identify 22 countries with sufficient time series data to estimate equation (10).[18] However, for two of these countries—Japan and Taiwan—, the data are so highly cross-sectionally correlated that their inclusion induces cross-sectional dependence in the residuals of equation (10). In other words, even the dynamic CCE estimator is unable to filter out the cross-sectional dependence when Japan and Taiwan are included. Therefore, we exclude these countries

[17] The inverse of life expectancy at age x is the annual death rate for the population aged x and above in a stationary population.

[18] The dynamic CCE mean group estimators requires sufficient time series data to estimate the country-specific model parameters, including the intercept and the coefficients on the cross-sectional averages of the lagged levels and the current and lagged first differences of the individual series (i.e., the number of observations per country must be greater than the number of parameters to be estimated).

from our main analysis. As we show in our robustness checks, our main results do not change substantially if we include Japan and Taiwan.

Our main dataset is an unbalanced panel data spanning 20 countries over up to 211 years from 1800 to 2010; the minimum number of observations per country is 53; the average number of observations per country is 113.8, and the total number of observations is 2276. Table A1 in Appendix A lists the 20 countries in our main sample along with the period averages for $\ln(y_{it})$, $MORT\_21_{it}$, and $LIFE\_21_{it}$; Figures A1-A3 in Appendix B show the data for each country in each year.

Before closing this section, we present a first pass at the long-run relationship between non-adult mortality, adult mortality, and the level of economic development by plotting the average log of GDP per capita versus the average mortality rate up to age 21 and the average log of GDP per capita versus life expectancy at age 21 for the 20 countries in our main sample. These scatter plots are shown in Figure 1. They indicate a strong negative association between GDP per capita and the non-adult mortality rate and a strong positive association between GDP per capita and adult life expectancy. Of course, such cross-country scatter plots are unable to control or account for the influence of other factors.

[Figure 1 about here]

In fact, the two mortality variables have no significant association with GDP per capita in simple country and time fixed effects regressions of $\ln(y_{it})$ on $MORT\_21_{it}$ and $LIFE\_21_{it}$, as shown in Table 1. However, the table also shows that these estimates potentially suffer from bias due to error cross-sectional dependence (as indicated by the significant CD statistics), implying that results from conventional fixed effects models of the relationship between mortality and economic development should generally be viewed with some caution. In the next section, we examine the long-run relationship between y_{it}, $LIFE\_21_{it}$, and $MORT\_21_{it}$ in more detail using the methods described above.

[Table 1 about here]

4. Empirical analysis

In the empirical analysis, we first test whether $\ln(y_{it})$, $MORT\_21_{it}$, and $LIFE\_21_{it}$ are integrated (of order 1) and cointegrated. We then estimate the effects of non-adult mortality and adult mortality on economic development using the CS-ARDL mean group estimator in error correction form and test the robustness of the estimates. Finally, we test for weak exogeneity.

4.1. Panel unit root and cointegration tests

A number of panel unit root tests have been developed in recent years. The most commonly used tests are so-called first generation panel unit root tests. However, first generation panel unit root tests, which assume cross-sectional independence, may suffer from size distortions in the presence of cross-sectional dependence due to common factors (viewed as a source of strong cross-sectional dependence) or spatial spillovers or (viewed as a source of weak cross-sectional dependence). Indeed, the CD test reported in Table 2 strongly rejects the null hypothesis of no cross-sectional dependence in the data for the variables in levels and in first differences. Therefore, we employ a second generation panel unit root test to account for cross-sectional dependence: the CIPS test proposed by Pesaran (2007), as discussed above. The results of this test for the levels and first differences of the variable are also reported in Table 2. As can be seen, the test statistics do not reject the null hypothesis that $\ln(y_{it})$, $MORT\_21_{it}$, and $LIFE\_21_{it}$ have a unit root in levels, whereas the unit root hypothesis for the first differences is rejected. Therefore, we treat the variables as $I(1)$ and proceed to test for cointegration.

[Table 2 about here]

Table 3 reports the bootstrapped p-values for the Westerlund (2007) tests of the null hypothesis of no cointegration between $\ln(y_{it})$ and $MORT\_21_{it}$ (row (1)), between $\ln(y_{it})$ and

*LIFE*_21$_{it}$ (row (2)), and between ln(y_{it}), *MORT*_21$_{it}$, and *LIFE*_21$_{it}$ (row (3)). The results in row (1) show that all tests fail to reject the null of no cointegration between the log of GDP per capita and the non-adult mortality rate. In contrast, the results in row (2) show that all four tests reject the null of no cointegration between the log of GDP per capita and adult life expectancy. Similarly, we find evidence of cointegration when all three variables are included; three of the four test statistics in row (3) are significant at standard confidence levels.

[Table 3 about here]

4.2. Effects of non-adult mortality and adult mortality on economic development

We now estimate equation (10). The results of including the variables separately and jointly in the model are presented in Table 4. Before discussing the coefficient estimates, it is worth mentioning that the CD test does not reject the null hypothesis of no error cross-sectional independence for all regressions, from which it can be concluded that the results in Table 4 are not biased by the presence of error cross-sectional independence.

Turning to the coefficients, we find that the parameter for the lagged level of the dependent variable (ln(y_{it-1})) is significant and negative in all three regressions. Thus, the error correction coefficients provide no evidence against cointegration.

However, both the coefficient on *MORT*_21$_{it-1}$ and the long-run coefficient on *MORT*_21$_{it}$ are statistically insignificant. Similarly, we find in columns (1) and (3) that the change in the non-adult mortality rate is not significantly associated with growth. These results are very similar to the instrumental variable results on the effect of life expectancy at birth on growth in Bloom et al. (2014).

[Table 4 about here]

In contrast, we find in columns (2) and (3) that the coefficient on *LIFE*_21$_{it-1}$ is highly significant and positive. While this is inconsistent with the results of Hansen and Lønstrup (2015),

who find that the lagged level of life expectancy at birth is negatively correlated with income growth (in both OLS and IV regressions), it is consistent with the results of Aghion et al. (2011), who report significant positive coefficients on initial life expectancy at birth (in both OLS and IV specifications).

Not surprisingly, as columns (2) and (3) also show, the long-run effect of life expectancy at age 21 on GDP per capita is positive and highly significant. In our preferred specification, which, following Lorentzen et al. (2008) and Aghion et al. (2011), includes both measures of mortality, the estimate of the long-run semi-elasticity of output per capita with respect to life expectancy at age 21 is 0.074. Accordingly, an increase in life expectancy at age 21 by one year raises the long-run level of output per capita by 7.4%.

To evaluate the magnitude of this effect, we multiply the estimated long-run coefficient by the average change in life expectancy at age 21 (0.138) in our sample. The resulting value is 0.010, implying that the increase in life expectancy at 21 between 1800 and 2010 has, on average, increased GDP per capita by about 1% per year. Given that GDP per capita increased on average by 1.95%, this means that the increase in years of life expectancy at age 21 has been responsible for about 50% of the increase in GDP per capita in our sample.

Returning our attention to the estimated error correction coefficient in column (3), we may also note that the level of in GDP per capita adjusts relatively slowly to changes in life expectancy at 21. Given that the half-life of a shock to $\ln(y_{it})$ is approximatively $-\ln(2)/\ln(1+ b_{1i})$, the estimated error correction coefficient implies that it takes about 4 years for 50% of the full effect to be realized, and about 20 years for 97% of the impact to occur.

For completeness, we note that the coefficient on $\Delta LIFE\_21_{it}$ in column (3) is positive and statistically significant, suggesting that life expectancy at age 21 also has a short-run influence on GDP per capita (growth).

4.3. Robustness

We conduct several sensitivity checks to gauge the robustness of our main findings. For the sake of brevity and clarity, we report here only the estimated long-run coefficients. Moreover, unless otherwise indicated, we always estimate models in which the mortality rate (up to age x) and life expectancy (at age x) are included jointly.

4.3.1. Country outliers

The first robustness test investigates whether the insignificant long-run effect of non-adult mortality and the significant positive long-run effect of adult life expectancy are due to individual country outliers. To undertake such a test, we re-estimate the CS-ARDL model excluding one country at a time from the sample and present the z-statistics of the sequentially estimated long-run effects (calculated by dividing the long-run coefficients by their standard errors) in Figure 2. The horizontal axis represents the country that is dropped from the sample (following the order presented in Table A1); the vertical axes plot the z-statistics for the long-run coefficients of $MORT\_21_{it}$ (right axis) and $LIFE\_21_{it}$ (left axis) in the remaining sample. While the z-statistics for the non-adult mortality variable (thick line) are in absolute value always smaller than the 10% critical value of 1.645, the z-statistics of the sequentially estimated long-run effects of $LIFE\_21_{it}$ on $\ln(y_{it})$ (thin line) are always greater than the 5% critical value of 1.96. It thus can be concluded that our regression results are not driven by a single country.

[Figure 2 about here]

4.3.2. Alternative lag structures

In Table 5, we test the sensitivity of our results to alternative lag structures. In column (1), the baseline model (with four lags of the first differences of the cross-sectional averages) is augmented with one lag of the differenced dependent variable; in column (2), the baseline model also includes

one lag of the first difference of $MORT\_21_{it}$ and $LIFE\_21_{it}$. Columns (3), (4), and (5) report results based on three lags of the first differences of the cross-sectional averages. In column (3), we use no lags of the first differences of the variables (as in our baseline specification); the results in column (4) are based on one lag of the differenced dependent variable (as in column (1)); and those in column (5) use one lag of the first difference of all variables (as in column (2)). As can be seen, the qualitative results remain the same regardless of which lag structure specification is used. The long-run coefficient on $MORT\_21_{it}$ is always insignificant while that on $LIFE\_21_{it}$ is always significant (at least at 10% level), and the sign of the latter is always positive. However, the CD test rejects the null hypothesis of no cross-sectional dependence in the residuals for the specifications in columns (2), (4), and (5) at the 10% significance level or less, implying that the estimates in these columns are likely to be biased.

Finally, it should be noted here that in the columns where the CD statistics indicate no evidence of error cross-sectional dependence (columns (1) and (3)), the estimated long-run coefficients of life expectancy at age 21 are smaller than their counterparts in Table 4, but still economically large: according to the long-run coefficient on $LIFE\_21_{it}$ in column (1), the increase life expectancy at age 21 between 1800 and 2010 has been responsible for about 38% of the increase in GDP per capita in our sample.

[Table 5 about here]

4.3.3. Alternative estimator

As a third robustness check, we use the CS-DL mean group estimator to estimate the long-run effects of non-adult mortality and adult mortality on the level of economic development. Table 6 presents the results. In column (1), the specification includes four lags of the cross-sectional averages of the independent variables and three lags of the first differences of $MORT\_21_{it}$ and $LIFE\_21_{it}$; the results in column (2) are based on a specification with five lags of the cross-sectional averages of the explanatory variables and four lags of the first differences of these variables. Using

these specifications, we again find insignificant long-run effects of $MORT\_21_{it}$, whereas the estimates of the long-run effect of $LIFE\_21_{it}$ on $\ln(y_{it})$ are positive and statistically significant (although only at the 10% level). However, the CS-DL results are contaminated by cross-section dependence, as indicated by the significant CD statistics, and should therefore be viewed with some caution. In addition, the CS-DL estimator is biased in the presence of feedback effects (as discussed above). Therefore, we prefer the CS-ARDL estimator, which is used in the remainder of this robustness analysis.

[Table 6 about here]

4.3.4. Allowing for a possible change in the slope coefficients and using an extended sample

Cervellati and Sunde (2011) find evidence that the effect of life expectancy at birth on growth is negative or insignificant before the onset of the demographic transition and positive after the onset of the demographic transition. Their explanation for this finding is that in the early stages of the demographic transition, when fertility is high, reductions in mortality (at all ages) produce an acceleration of population growth and thereby exert a negative effect on per capita income growth, whereas in the later stages, reductions in mortality lead to a sufficient fertility decline to bring about a decrease in population growth. An alternative explanation for their results is that in the early stages of the demographic transition, increases in life expectancy at birth are mainly attributable to declines in infant and child mortality, rather than improvements in adult mortality. If the effects of infant and child mortality on growth differ from that of adult mortality, and advances in life expectancy at birth at the beginning of the demographic transition mainly reflect declines in infant and child mortality, then it is not surprising to find differences in the effects of life expectancy at birth before and after the onset of the demographic transition.

If the long-run effects of non-adult mortality and adult mortality on GDP per capita differ before and after the onset of the demographic transition, then the onset of the demographic

transition should produce a significant change in the slope coefficients of the mortality variables. To investigate this, we estimate a modified specification of the baseline model in which we include a (lagged) dummy for the period after the onset of the demographic transition, DU_{it}, and an interaction term between this dummy variable and our two measures of mortality. DU_{it} equals 1 for the period after the onset of the demographic transition and 0 otherwise. Following Cervellati and Sunde (2011), we define the onset of the demographic transition as the date when life expectancy at birth exceeds 50 years. The estimates of the long-run coefficients from this specification are shown in column (1) of Table 7. As can be seen, only the long-run coefficient on $LIFE\_21_{it}$ is significant.

Thus, we find no evidence of a change in the long-run coefficients of the mortality variables due to the onset of the demographic transition (as the insignificant interaction terms indicate). In other words, the results in column (1) of Table 7 suggest that the long-run effects of non-adult mortality and adult mortality on GDP per capita are independent of the onset of the demographic transition.

[Table 7 about here]

In column (2) of Table 7, we present estimates using the original sample plus Japan and Taiwan. The point estimates and significance levels of the long-run coefficients on $MORT\_21_{it}$ and $LIFE\_21_{it}$ are similar to those in column (3) of Table 4. However, as discussed in Section 3.3, the inclusion of Japan and Taiwan induces cross-sectional dependence in the residuals, as the CD statistic indicates. Therefore, we prefer the sample without these two countries.

4.3.5. Different age-specific mortality rates and life expectancies

As a final robustness check, we experiment with different age-specific mortality rates and life expectancies. More specifically, we replace the mortality rate up to age 21 with the mortality rate up to age 1, labelled $MORT\_1_{it}$, and estimate the long-run effect of this measure on GDP per capita together with the long-run effect on GDP per capita of life expectancy at a particular age x, labelled

$LIFE\_x_{it}$. Table 7 shows the results of this exercise using age-specific life expectancies at ages 1, 18, 21, 30, 50, and 60. As can be seen, the long-run coefficient on $MORT\_1_{it}$ is always insignificant. The long-run coefficient on $LIFE\_x_{it}$, in contrast, is positive and significant in columns (1)-(6), from which it can be concluded that increases life expectancy at ages ≤ 50 are associated, in the long run, with increases in GDP per capita. In this context, it is interesting to note that life expectancy at age 30 has the largest long-run coefficient (see column (4)), followed by life expectancy at age 21 (see column (3)), whereas the coefficient on life expectancy at age 60 is smallest and not significant (see column (7)). Overall, the pattern of the coefficients in Table 7 suggests that gains in life expectancy of middle-aged adults have the largest impact on GDP per capita, whereas infant and old-age mortality appear to have no statistically significant effect on economic development. More specifically, given that the long-run coefficients on life expectancy at ages 1, 18, and 21 in columns (1) – (3) are not larger than the long-run coefficient on life expectancy at age 30 in column (4) (although life expectancies at ages 1, 18, and 21 incorporate the mortality rates at all later ages, whereas life expectancy at age 30 includes "only" the mortality experience at older ages), it can be cautiously concluded from the results in Table 8 that mortality up to age 30 has little to no effect on GDP per capita in the long run.

[Table 8 about here]

While age-specific life expectancy at age x depends on the mortality rates for every age above x, age-specific mortality up to age x depends on the mortality rates for every age below x. Therefore, if a decline in adult mortality has a positive long-run effect on output per capita, we should find a significant negative long-run coefficient for our mortality rate at a certain adult age. To investigate this, we estimate the long-run coefficient on the mortality rate of people aged $0 - x$ for all values of x up to 90 years (without including life expectancy). The results of these estimations are shown in Figure 3. The horizontal axis represents the age x in the age-specific mortality rate, $MORT\_x_{it}$, and the vertical axes represent the estimated long-run coefficients (—)

and their *z*-statistics (▬). From the figure it can be seen that the *z*-statistics of the long-run effects of $MORT\_x_{it}$ on y_{it} are in absolute value smaller than the 10% critical value of 1.645 only for values of *x* between 33 and 85 and that the mortality rate up to age 43 has the largest long-run coefficient (in absolute value). This again suggests that long-run increases in income are mainly due to decreases in mortality in middle adulthood, whereas mortality at young (and old) ages has little to no effect on income per capita.

[Figure 3 about here]

4.4. Weak exogeneity tests

The significant error correction coefficients in the conditional ECMs (with and without non-adult mortality) already suggest that GDP per capita can be treated as endogenous in the long-run relationship. In this section, we provide a formal test of the weak exogeneity of $MORT\_21_{it}$ and $LIFE\_21_{it}$ for the regression parameters in the conditional ECM for $\Delta\ln(y_{it})$.

As discussed in Section 3.2.3, this test involves first estimating the long-run coefficients on $MORT\_21_{it}$ and $LIFE\_21_{it}$ for each country separately. The country-specific long-run coefficients are then used to construct the error correction term

$$ec_{it} = \ln(y_{it}) - (\hat{c}_i + \hat{\beta}_{it}^{M} MORT\_21_{it} + \hat{\beta}_{it}^{L} LIFE\_21_{it}), \tag{15}$$

which is then included in a panel VECM of the form (7) to test whether it is significantly different from zero (using the lambda-Pearson statistic).

A point worth mentioning is that the country-specific long-run coefficients are in part very sensitive to the number of lagged cross-sectional averages used in equation (10), as can be seen in Figures A4 and A4 in Appendix C. Specifically, the long-run coefficients for Ireland (country 10) change dramatically depending on the lag length and appear as outliers in the distribution of the long-run coefficients when a four-lag model is used. Therefore, we use the individual long-run

coefficients from a cross-sectionally augmented ECM specification with two lags of the cross-section averages to construct the error correction term given by equation (15).

Before turning to the results of the weak exogeneity test based on the individual long-run coefficients from the two-lag model, we report the average long-run coefficients on $MORT\_21_{it}$ and $LIFE\_21_{it}$ from this model: While the CS-ARDL estimate of the average long-run coefficient on $MORT\_21_{it}$ is -0.053 with a standard error of 0.051, the average long-run coefficient on $LIFE\_21_{it}$ is estimated to be 0.070 with a standard error of 0.018.[19] As expected, these estimates were very similar to their counterparts in column (3) of Table 4 and, again, suggest that a decline in adult mortality, on average, has a positive long-run effect on GDP per capita, whereas a change in non-adult mortality, on average, has no impact on GDP per capita.

This interpretation of the estimation results is based on the assumption that $MORT\_21_{it}$ and $LIFE\_21_{it}$ can be treated as weakly exogenous. The lambda-Pearson statistics in Table 9 support this assumption, revealing that the null hypothesis of weak exogeneity of $\ln(y_{it})$ can be rejected at the 1% level, whereas the hypothesis of weak exogeneity cannot be rejected in the case of non-adult mortality and adult mortality.

3. Conclusions

In this study, we examined the effects of adult and non-adult mortality on the long-run level of income in a dynamic heterogeneous and cross-sectionally dependent panel. Our results, based on unbalanced panel data for 20 countries for the period 1800-2010, lead to three main conclusions:

(i) While non-adult mortality does not affect per capita income in the long run, a reduction in adult mortality has a large causal positive effect on the long-run level of income.

(ii) The long-run income effects of non-adult mortality and adult mortality do not differ before and after the onset of the demographic transition.

[19] The CD statistic is 1.43, suggesting that there is no error cross-sectional dependence.

(iii) Mortality in middle adulthood (after age 30) has the greatest impact, whereas early adulthood mortality and mortality in later adulthood (after age 60) have little to no impact on the long-run level of per capita income.

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Table 1. Static fixed effects estimates

	(1)	(2)	(3)
$MORT\_21_{it}$	-0.008		-0.007
	(0.010)		(0.012)
$LIFE\_21_{it}$		0.011	0.004
		(0.013)	(0.018)
CD	-8.47***	-8.31***	-8.47***
Number of observations	2276	2276	2276

Notes: The dependent variable is $\ln(y_{it})$. All regressions include country and time fixed effects (coefficients not reported). Robust standard errors are in parentheses. CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. *** indicates significance at the 1% level.

Table 2. Cross-sectional dependence and panel unit root tests

	$\ln(y_{it})$	$\Delta\ln(y_{it})$	$MORT\_21_{it}$	$\Delta MORT\_21_{it}$	$LIFE\_21_{it}$	$\Delta LIFE\_21_{it}$
CD	119.64***	28.02***	120.78***	39.80***	107.85***	36.81***

CIPS	-0.610 (*c*, *t*)	-9.997*** (*c*)	0.219 (*c*, *t*)	-12.492*** (*c*)	1.234 (*c*, *t*)	-13.479*** (*c*)

Notes: CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. CIPS is the cross-sectionally augmented IPS test proposed by Pesaran (2007); *c* (*t*) indicates that the test includes country-specific intercepts (and time trends). All panel unit root statistics reported are standardized so that they are distributed as standard normal under the null of a unit root; large negative values lead to rejection of a unit root in favor of (trend) stationarity. Given the large number of observations for each country, we used six lags in the CIPS test to adjust for autocorrelation. *** indicates significance at the 1% level.

Table 3. Panel cointegration tests

	P_τ	P_α	G_τ	G_α
(1) Bivariate cointegration tests between $\ln(y_{it})$ and $MORT\_21_{it}$	0.269	0.404	0.465	0.932
(2) Bivariate cointegration tests between $\ln(y_{it})$ and $LIFE\_21_{it}$	**0.001**	**0.000**	**0.015**	**0.053**
(3) Trivariate cointegration tests between $\ln(y_{it})$, $MORT\_21_{it}$, and $LIFE\_21_{it}$	**0.097**	**0.078**	**0.018**	0.109

Notes: The table reports cross-sectional dependence robust *p*-values (based on 1000 replications) from the four panel cointegration tests of Westerlund (2007). The dependent variable in the Westerlund tests is Δy_{it}. The null hypothesis is that the variables are not cointegrated. We used the Akaike Information Criterion to determine the optimal lead and lag length with a maximum number of two leads and lags. The kernel bandwidth is set according to the rule $4(T/100)^{2/9} = 4$. *p*-values in bold indicate that the null hypothesis of no cointegration can be rejected at standard significance levels.

Table 4. CS-ARDL estimates of equation (10)

	(1)	(2)	(3)
$\ln(y_{it-1})$	-0.139***	-0.104***	-0.165***
	(0.023)	(0.022)	(0.026)
$MORT\_21_{it-1}$	-0.011		0.006
	(0.009)		(0.010)
$LIFE\_21_{it-1}$		0.010***	0.012***
		(0.004)	(0.004)
$\Delta MORT\_21_{it}$	-0.011		-0.005
	(0.009)		(0.010)
$\Delta LIFE\_21_{it}$		0.005	0.010***
		(0.005)	(0.003)
Long-run coefficient on $MORT\_21_{it}$	-0.076		-0.038
	(0.060)		(0.060)
Long-run coefficient on $LIFE\_21_{it}$		0.096***	0.074***
		(0.042)	(0.028)
CD	1.20	0.76	1.14
Number of observations	2183	2183	2183

Notes: The dependent variable is $\Delta\ln(y_{it})$. The number of lags of the cross-section averages in the cross-sectionally augmented ECM regressions was set to $p - 1 = 4$. The long-run coefficients of the variables were calculated by dividing their lagged coefficients by the absolute value of the coefficients on y_{t-1}. Standard errors are in parentheses; the standard errors of the coefficients were constructed nonparametrically as described in Pesaran and Smith (1995); the standard errors of the long-run coefficients were computed using the delta method. CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. *** (**) [*] indicates significance at the 1% (5%) [10%] level.

Table 5. CS-ARDL estimates of the long-run effects of non-adult mortality and adult mortality on GDP per capita using different lag structures

	(1) $p-1=4$ $k^y=1, k^M=0$	(2) $p-1=4$ $k^y=1, k^M=1$	(3) $p-1=3$ $k^y=0, k^M=0$	(4) $p-1=3$ $k^y=1, k^M=0$	(5) $p-1=3$ $k^y=1, k^M=1$
Long-run coefficient on $MORT\_21_{it}$	-0.027	-0.067	-0.023	-0.021	-0.032
	(0.050)	(0.050)	(0.048)	(0.040)	(0.037)
Long-run coefficient on $LIFE\_21_{it}$	0.054**	0.044*	0.063**	0.046**	0.049**
	(0.021)	(0.023)	(0.026)	(0.020)	(0.023)
CD	1.52	1.96**	0.99	1.78*	1.79*
Number of observations	2182	2182	2202	2200	2200

Notes: The dependent variable is $\Delta\ln(y_{it})$. $p-1$ is the number of lags of the first differences of the cross-section averages included in the model; k^y is the number of lags of the (differenced) dependent variable; and k^M is the number of lags of the first differences of the independent variables. The long-run coefficients of the variables were calculated by dividing their lagged coefficients by the absolute value of the coefficients on y_{t-1}. The standard errors of the coefficients were constructed nonparametrically as described in Pesaran and Smith (1995). The values in parentheses are the standard errors of the estimated long-run coefficients, calculated by the Delta method. CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. ** (*) indicates significance at the 5% (10%) level.

Table 6. CS-DL estimates of the long-run effects of non-adult mortality and adult mortality on GDP per capita

	(1) $p=4$ $p-1=3$	(2) $p=5$ $p-1=4$
Long-run coefficient on $MORT\_21_{it}$	0.003	-0.001
	(0.033)	(0.036)
Long-run coefficient on $LIFE\_21_{it}$	0.044*	0.053*
	(0.025)	(0.030)
CD	3.69***	3.09***
Number of observations	2203	2183

Notes: The dependent variable is $\ln(y_{it})$. p is the number of lags of the first differences of the independent variables; $p-1$ is the number of lags of the cross-section averages of the independent variables. Standard errors are in parentheses; they were constructed nonparametrically as described in Pesaran and Smith (1995). CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. *** (*) indicates significance at the 1% (10%) level.

Table 7. CS-ARDL estimates of the long-run effects of non-adult mortality and adult mortality on GDP per capita allowing for a possible change in the slope coefficients and using a broader sample of countries

	(1)	(2)
Long-run coefficient on $MORT\_21_{it}$	-0.007	-0.066
	(0.031)	(0.062)
Long-run coefficient on $LIFE\_21_{it}$	0.068***	0.080***
	(0.025)	(0.022)
Long-run coefficient on $MORT\_21_{it} \times DU_{it}$	-0.037	
	(0.043)	
Long-run coefficient on $LIFE\_21_{it} \times DU_{it}$	-0.007	
	(0.011)	
Long-run coefficient on DU_{it}	0.559	

	(0.553)	
CD	0.38	4.55***
Number of countries	20	22
Number of observations	2183	2278

Notes: The dependent variable is $\Delta\ln(y_{it})$. DU_{it} is a dummy for the period after the onset of the demographic transition. The estimates in column (1) are based on a broader sample that includes our original sample as well as Japan and Taiwan. The number of lags of the cross-section averages in the cross-sectionally augmented ECM regressions was set to $p - 1 = 4$. The long-run coefficients of the variables were calculated by dividing their lagged coefficients by the absolute value of the coefficients on y_{t-1}. The standard errors of the coefficients were constructed nonparametrically as described in Pesaran and Smith (1995). The values in parentheses are the standard errors of the estimated long-run coefficients, calculated by the Delta method. CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. *** indicates significance at the 1% level.

Table 8. CS-ARDL estimates of the long-run coefficients on $MORT\_1_{it}$ and $LIFE\_x_{it}$ for different ages x

	(1) $x = 1$	(2) $x = 18$	(3) $x = 21$	(4) $x = 30$	(5) $x = 40$	(6) $x = 50$	(7) $x = 60$
Long-run coefficients on $MORT\_1_{it}$	0.006	-0.010	-0.009	-0.014	-0.017	-0.033	-0.049
	(0.095)	(0.099)	(0.099)	(0.09)	(0.081)	(0.073)	(0.069)
Long-run coefficient on $LIFE\_x_{it}$	0.088***	0.101***	0.107***	0.111***	0.094***	0.072**	0.047
	(0.030)	(0.031)	(0.033)	(0.034)	(0.03)	(0.028)	(0.037)
CD	1.18	0.74	1.14	0.41	0.33	0.24	0.03
Number of observations	2183	2183	2183	2183	2183	2183	2183

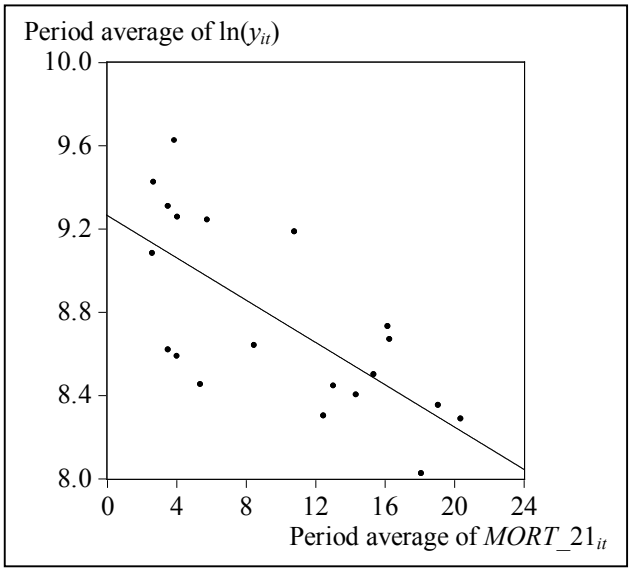
Notes: The dependent variable is $\Delta\ln(y_{it})$. x denotes age and $LIFE\_x_{it}$ is life expectancy at age x. The number of lags of the cross-section averages in the cross-sectionally augmented ECM regressions was set to $p - 1 = 4$. The long-run coefficients of the variables were calculated by dividing their lagged coefficients by the absolute value of the coefficients on y_{t-1}. The standard errors of the coefficients were constructed nonparametrically as described in Pesaran and Smith (1995). The values in parentheses are the standard errors of the estimated long-run coefficients, calculated by the Delta method. CD is the cross-sectional dependence test of Pesaran (2004); the CD statistic is normally distributed under the null hypothesis of no cross-sectional dependence. *** (**) indicates significance at the 1% (5%) level.

Table 9. Weak exogeneity tests

	(1) $\ln(y_{it})$	(2) $MORT\_21_{it}$	(3) $LIFE\_21_{it}$
Lambda-Pearson	88.78	49.96	44.32
	[0.000]	[0.135]	[0.294]

Notes: The lambda-Pearson (Fisher) test statistics are distributed as chi-squared with $2\times N$ (= 40) degrees of freedom. The *p*-values are in brackets. The results were obtained with four lags of the differenced regressors.

Figure 1. Scatter plots of the log of real GDP per capita against the non-adult mortality rate and the log of real GDP per capita against adult life expectancy



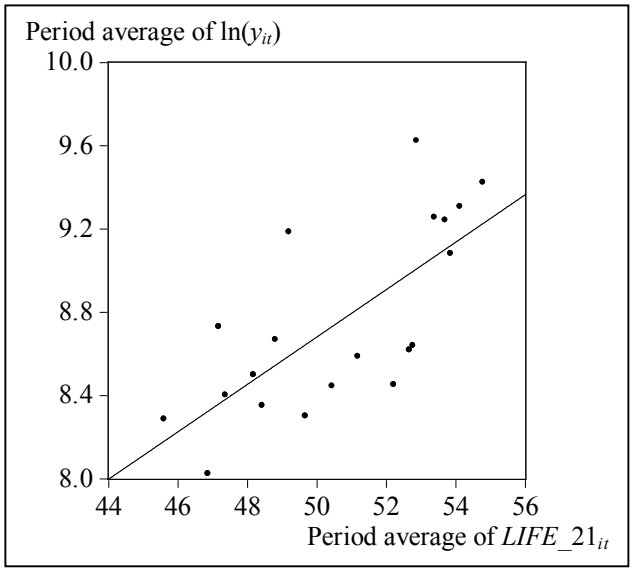


Figure 2. CS-ARDL estimation with single country excluded from the sample

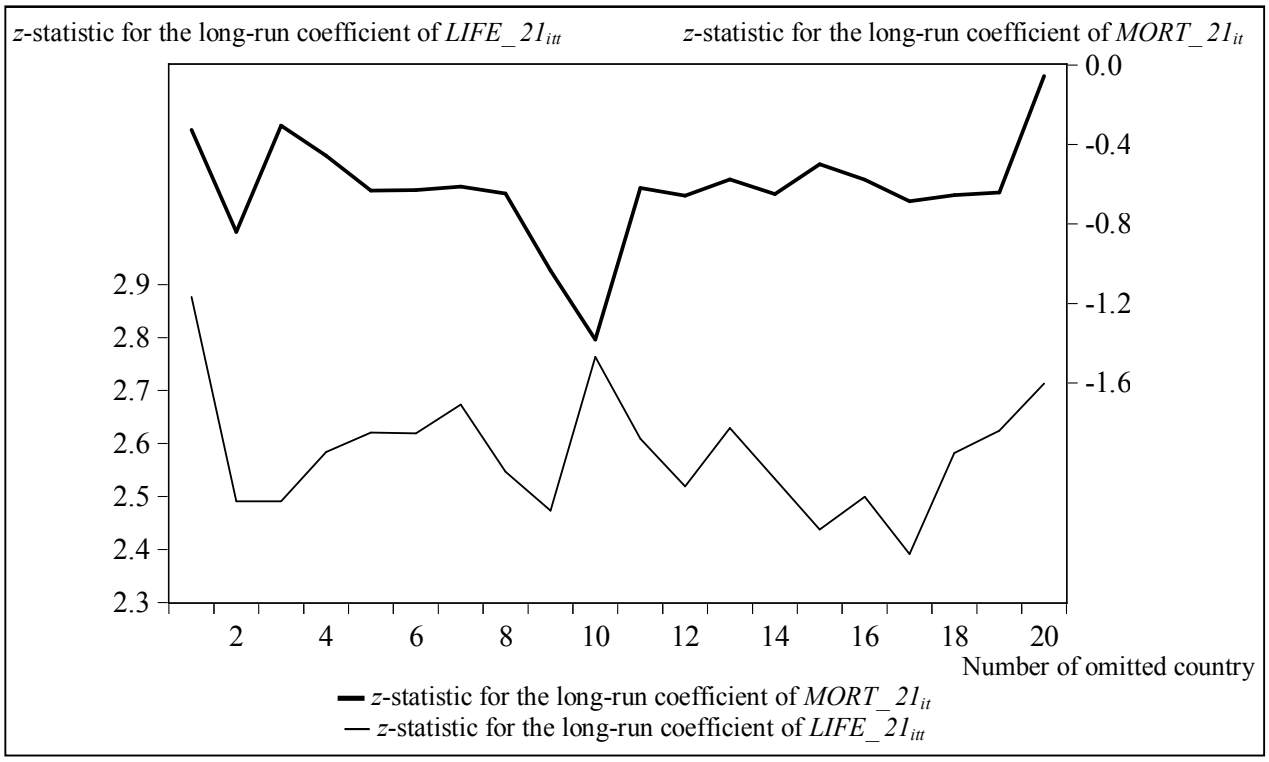
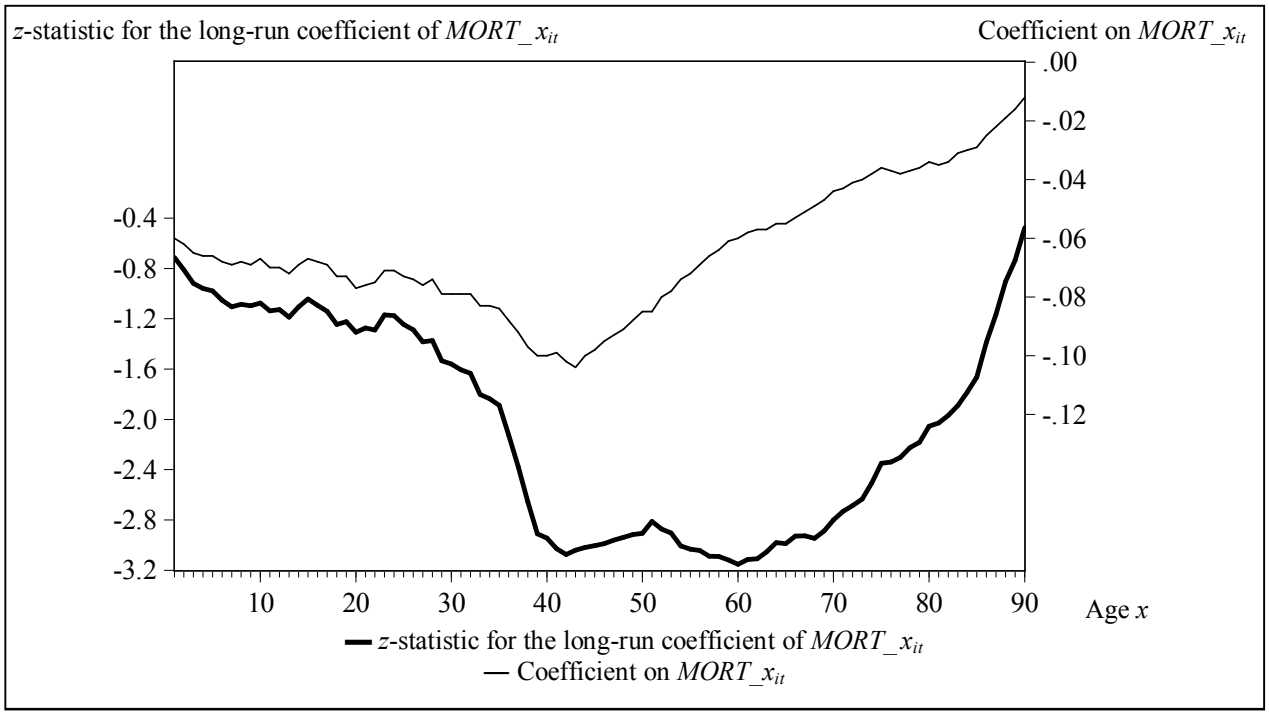


Figure 3. CS-ARDL estimates of the long-run coefficients on $MORT\_x_{it}$ for different ages x



Appendix A. Countries in the main sample, 1800-2010

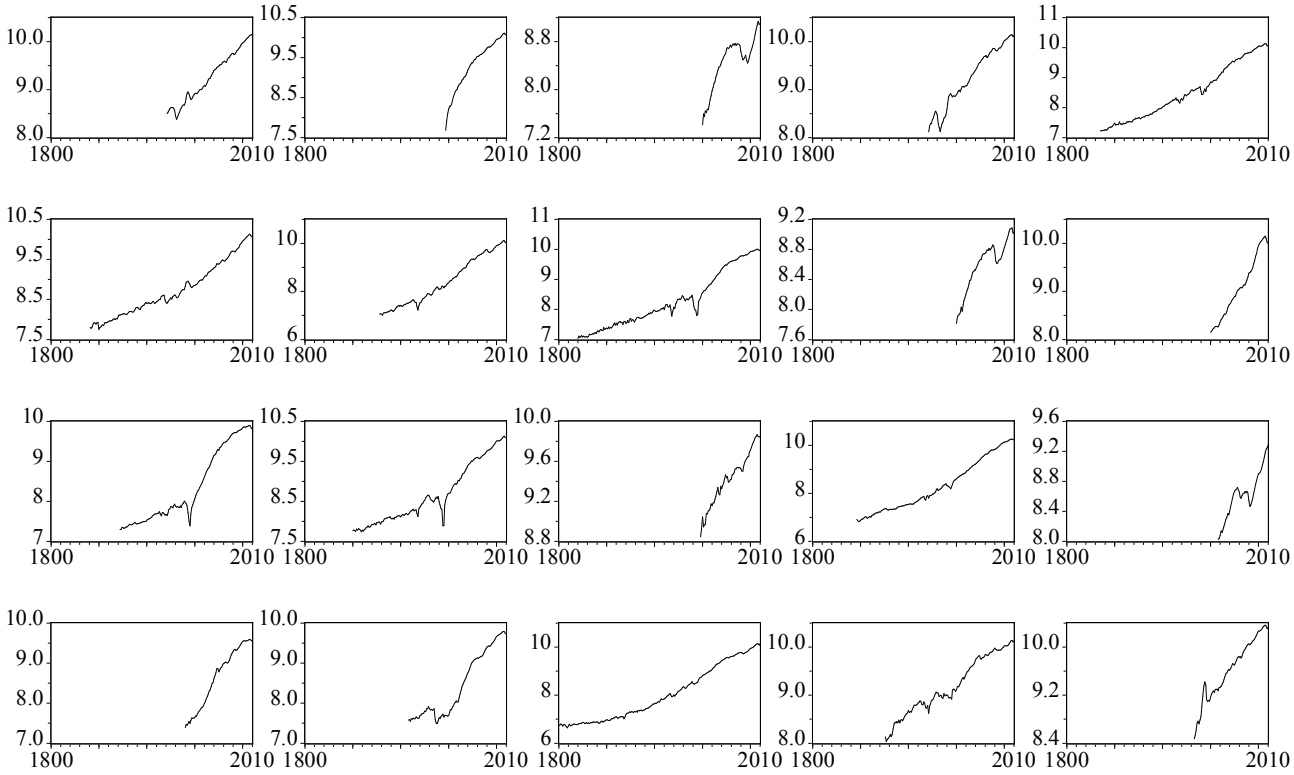
Table A1. Countries and summary statistics

	Average of $\ln(y_{it})$	Average of $MORT\_21_{it}$	Average of $LIFE\_21_{it}$
Australia	9.257	4.043	53.381
Austria	9.309	3.512	54.121
Bulgaria	8.453	5.366	52.211
Canada	9.243	5.769	53.689
Denmark	8.500	15.356	48.172
England and Wales	8.731	16.163	47.168
Finland	8.402	14.340	47.363
France	8.288	20.364	45.594
Hungary	8.588	4.019	51.182
Ireland	9.083	2.606	53.848
Italy	8.352	19.070	48.424
Netherlands	8.669	16.268	48.802
New Zealand	9.424	2.676	54.782
Norway	8.302	12.457	49.662
Poland	8.619	3.503	52.663
Portugal	8.641	8.466	52.765
Spain	8.446	13.037	50.437
Sweden	8.025	18.093	46.860

Switzerland	9.186	10.786	49.198
United States	9.625	3.867	52.866

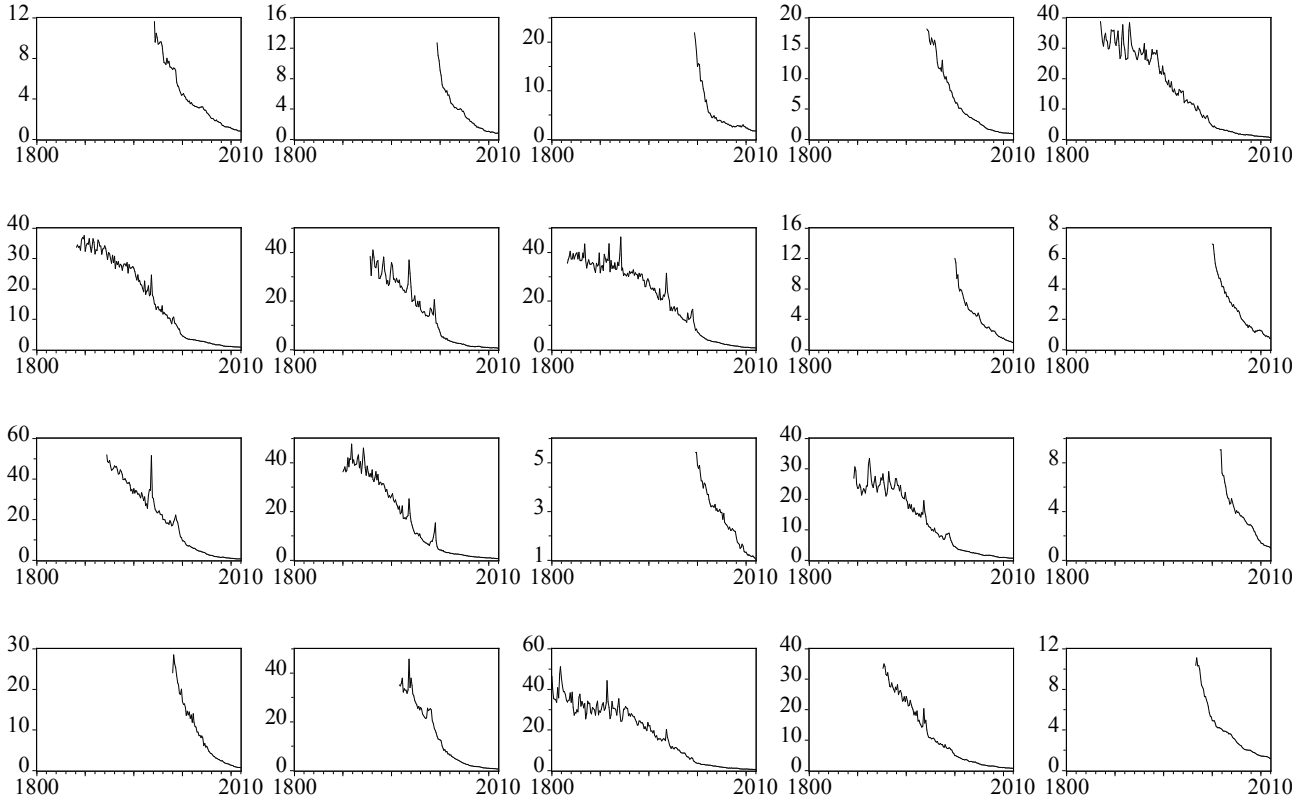
Appendix B. Main variables by country

Figure A1. Log of real GDP per capita by country over the period 1800-2010, ln(y_{it})



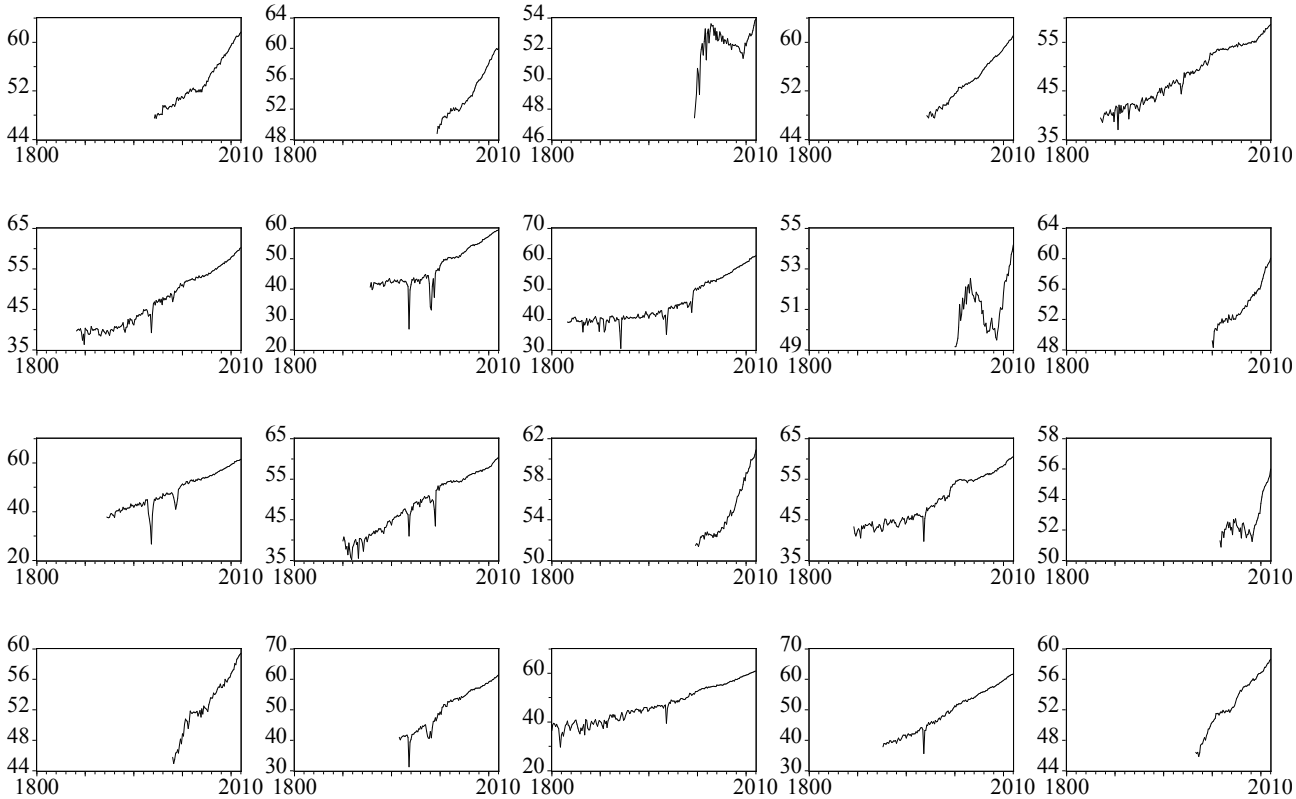
Notes: The countries from left to right are: Australia, Austria, Bulgaria, Canada, Denmark, England and Wales, Finland, France, Hungary, Ireland, Italy, the Netherlands, New Zealand, Norway, Poland, Portugal, Spain, Sweden, Switzerland, and the United States.

Figure A2. Non-adult mortality rate by country over the period 1800-2010, $MORT\_21_{it}$



Notes: The countries from left to right are: Australia, Austria, Bulgaria, Canada, Denmark, England and Wales, Finland, France, Hungary, Ireland, Italy, the Netherlands, New Zealand, Norway, Poland, Portugal, Spain, Sweden, Switzerland, and the United States.

Figure A3. Adult life expectancy by country over the period 1800-2010, $LIFE\_21_{it}$



Notes: The countries from left to right are: Australia, Austria, Bulgaria, Canada, Denmark, England and Wales, Finland, France, Hungary, Ireland, Italy, the Netherlands, New Zealand, Norway, Poland, Portugal, Spain, Sweden, Switzerland, and the United States.

Appendix C. Individual country CS-ARDL estimates of the long-run coefficients on *LIFE*_21$_{it}$ and *LIFE*_21$_{it}$

Figure A4. Individual country CS-ARDL estimates of the long-run coefficients on *MORT*_21$_{it}$ with two and four lags of the cross-section averages

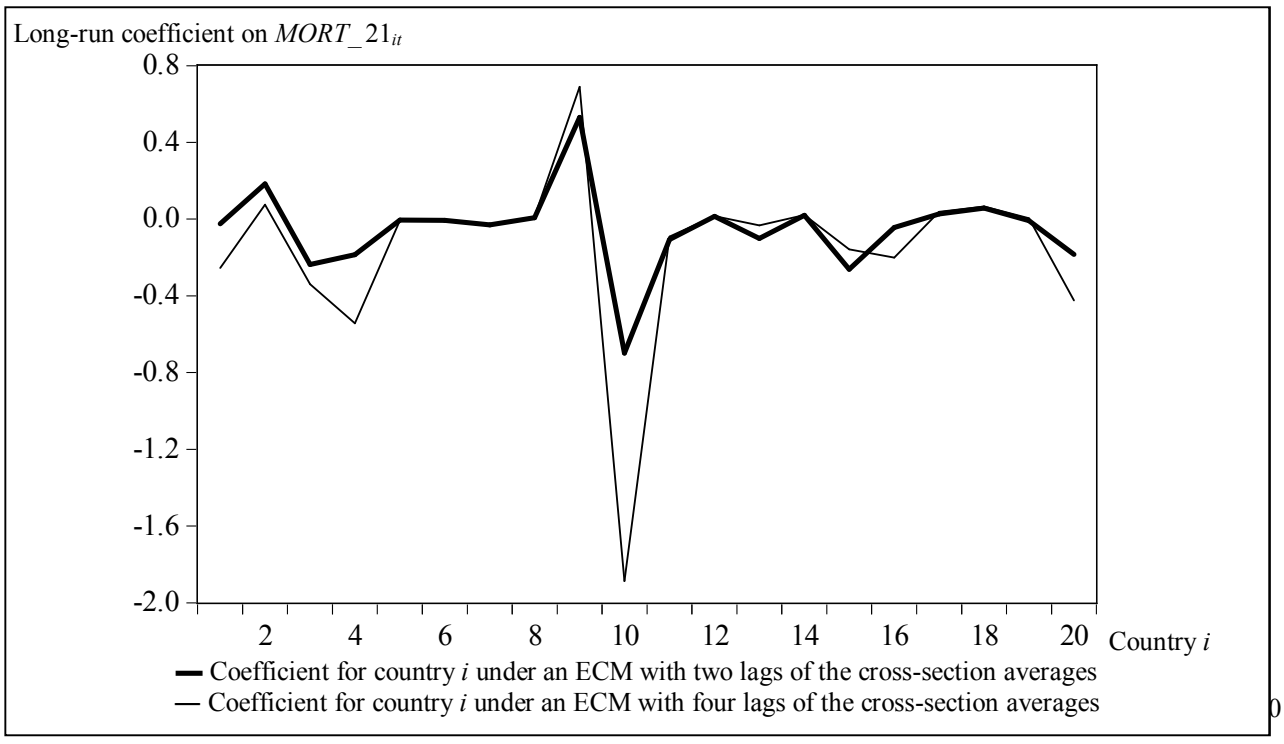
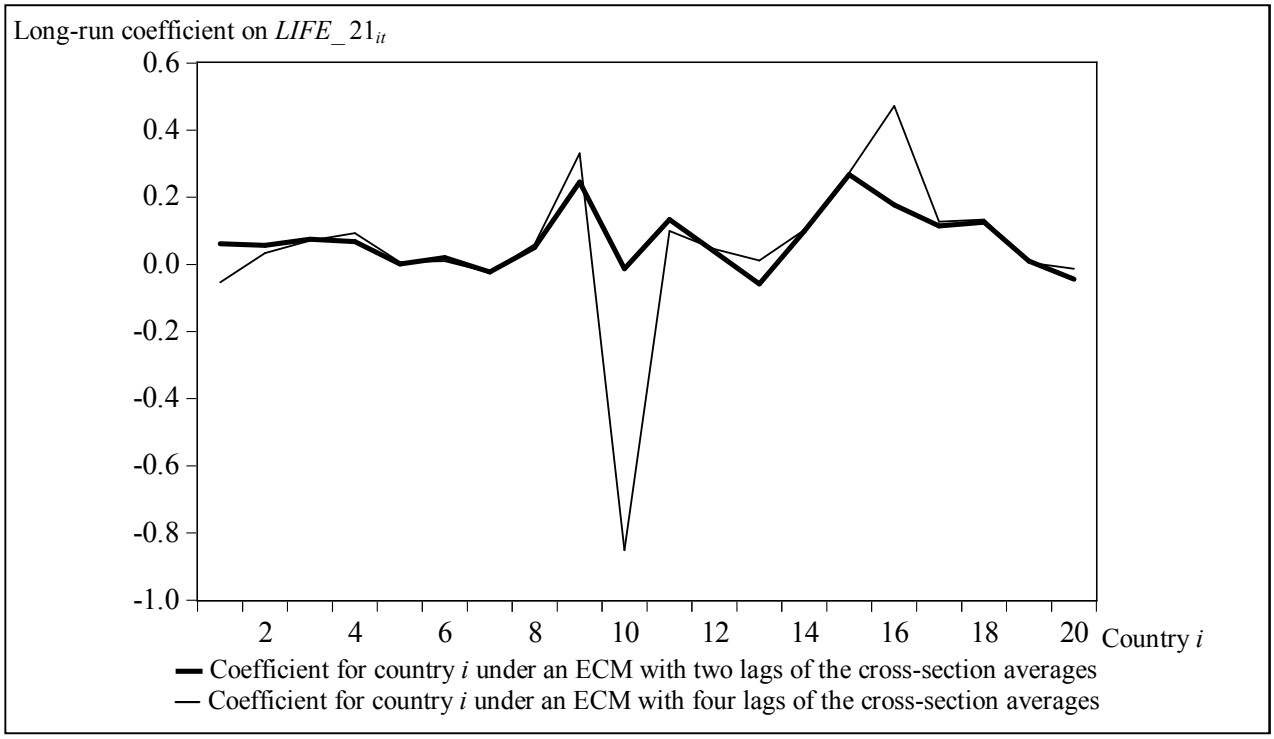


Figure A5. Individual country CS-ARDL estimates of the long-run coefficients on $LIFE\_21_{it}$ with two and four lags of the cross-section averages



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