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## Are cardiovascular diseases bad for economic growth?

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# ARE CARDIOVASCULAR DISEASES BAD FOR ECONOMIC GROWTH?

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# ARE CARDIOVASCULAR DISEASES BAD FOR ECONOMIC GROWTH?

## Abstract

We assess the impact of cardiovascular disease (CVD) mortality on economic growth, using a dynamic panel growth regression framework taking into account potential endogeneity problems. We start from a worldwide sample of countries for which data was available and detect a non-linearity in the influence of working age CVD mortality rates on growth across the per capita income scale. We then split the sample (according to the resulting income threshold) into low- and middle-income countries on one hand, and high-income countries on the other hand. In the latter sample we find a robust negative contribution of increasing CVD mortality rates on subsequent five-year growth rates. Not too surprisingly, we find no significant impact in the low- and middle-income country sample.

JEL Code: I10, O47.

Keywords: cardiovascular disease, growth empirics, dynamic panel data estimator.

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## 1 Introduction

There is abundant evidence that cardiovascular disease (CVD) and non-communicable disease (NCD) more generally are no longer mere “diseases of affluence”. CVD kills nearly three times as many people in developing countries each year as HIV, tuberculosis, and malaria combined, and it accounts by far for the predominant share of overall mortality in developed countries. The main question that this paper seeks to address is therefore: *are CVDs bad for economic growth?*

Theoretically, ill-health may not only reduce the supply and productivity of labor but can also impede human capital accumulation. Hence, a poor health status would negatively impact individual and social welfare both through the utility function and through a lower growth rate (and level) of national per capita income. The empirical literature on the role of health in explaining the variation in growth rates (or levels) of per capita incomes has grown substantially in recent years<sup>2</sup> – with the overall finding that health – when measured as life expectancy or adult mortality – enters as one of very few robust predictors of subsequent economic growth (Levine and Renelt 1992; Sala-i-Martin et al. 2004)<sup>3</sup>. However, research in this area has so far focused either on general health indicators (i.e., life expectancy, adult mortality) or on specific diseases most characteristic of developing countries. As for the latter, there is, for instance, evidence on the impact of malaria (Gallup and Sachs 2001), HIV/AIDS (Dixon et al. 2001), malnutrition (Weil 2005), and tuberculosis (Delfino and Simmons 1999) on economic growth.

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<sup>2</sup> For extensive overviews see e.g. Commission on Macroeconomics and Health (2001) and López-Casasnovas et al. (2005).

<sup>3</sup> A recent qualification is by Acemoglu and Johnson (2006).

While the vast majority of studies examines a worldwide sample, it is not obvious whether the same relationship exists between health and economic growth for rich countries as it does for poor countries. Indeed, Rivera and Currais (1999a, b), for example, find that an increase in life expectancy reduces economic growth rates among OECD countries.<sup>4</sup> Therefore, it seems important to pay particular attention to whether there are sample breaks and, if so, where they occur. To the best of our knowledge, only Bhargava et al. (2001) determine endogenously the sample breakpoint. They establish a threshold in terms of per capita income below which a higher adult survival rate is positively affecting growth. Above the (relatively low) per capita income threshold the relationship switches signs.

To the best of our knowledge, no study has hitherto explored in some more depth the growth implications of the particular type of diseases that account for the largest disease burden in high-income countries, and for an already substantial and fast growing burden in developing countries, i.e., non-communicable disease (NCD)<sup>5</sup>. The present paper fills this gap by examining the growth impact of cardiovascular disease (CVD), which is the disease that accounts for the greatest share of the NCD burden<sup>6</sup>. We follow Bhargava et al. (2001) in taking into particular account the potential difference between developed and developing countries, regarding the growth impact of this specific health indicator.

A priori, there is a lot to suggest that CVD might “matter” in economic terms.

First, a series of recent reports has highlighted the substantial and growing public health importance of CVD (Jamison et al 2006; WHO 2005). A distinctive feature of CVDs (as well as some other non-communicable diseases) is that it causes morbidity and, hence, potential

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<sup>4</sup> However, Knowles and Owen (1995, 1997) find a positive correlation when replacing life expectancy by public health expenditure as a proxy for “health”.

<sup>5</sup> It has come to be widely accepted that the term “non-communicable disease” is a somewhat unfortunate choice. Increasingly, NCDs are being called “chronic disease” in light of long duration. (WHO 2005).

<sup>6</sup> According to latest WHO estimates CVDs accounted for 51% of overall mortality due to NCDs in low- and middle-income countries in 2005 (WHO 2006).

productivity decline years or even decades ahead of death<sup>7</sup>. Second, a recent review of the economic impact of chronic disease has synthesized a significant number of cost-of-illness studies highlighting the substantial economic magnitudes involved in CVDs and the related risk factors (Suhrcke et al. 2005)<sup>8</sup>. Third, the same review also summarized the microeconomic evidence on the labor market impact of chronic disease (and relevant risk factors).<sup>9</sup>

The question then imposes itself whether on top of the obvious epidemiological evidence, the cost of illness as well as the microeconomic evidence, there may also be a macroeconomic impact of CVDs.

The present paper assesses the impact of CVD mortality on economic growth, adopting a panel growth regression framework, taking into account the potential endogeneity of CVDs. We are thereby careful in proxying CVD disease by its mortality rate among the working age cohort to capture as close as possible the impact of the disease on worker productivity. As in Bhargava et al. (2001), we start from a worldwide sample of countries for which data was available and detect a non-linearity in the influence of CVD on growth across the per capita income scale while controlling at the same time for more general health indicators. Then, we split the sample (according to the resulting income threshold) into low- and middle-income countries on one hand, and high-income countries on the other hand. In the latter sample we find a fairly robust negative contribution of increasing CVD mortality rates on subsequent five-year growth rates. Not surprisingly, we find no significant impact in the low- and middle-income country sample. Taken literally, this implies that as countries grow richer they should seek to avoid that what used to be “diseases of affluence” does not end up as obstacles to (even greater) affluence.

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<sup>7</sup> This feature of CVDs also helps prevent allegations of reverse causality in our growth regression framework adopted below.

<sup>8</sup> See also Petersen et al (2005) for cost-of-illness estimates for all European Union member-states.

<sup>9</sup> On the economic consequences of CVDs see also Leeder et al. (2004).

The paper is structured as follows. Section 2 argues why CVD may be of particular importance in explaining income growth; section 3 describes the empirical methodology, section 4 presents and discusses the results. The final section concludes.

## 2 Cardiovascular disease

Why should cardiovascular disease matter for growth? Traditionally, just like non-communicable diseases (NCDs) in general, CVDs have been considered by many as a “disease of affluence” – a consequence rather than a determinant of economic development. Moreover, the economic importance of NCDs (or CVDs in particular) has typically been downplayed as it (allegedly) affected predominantly people beyond retirement age.

However, such notions of “conventional wisdom” fail to correctly characterize the contemporary epidemiological reality (Ezzati et al. 2005). First, NCDs already account for a larger share of mortality than communicable diseases and child and maternal disease taken together in all but the low income countries (see Figure 1). The richer the country, however, the greater the share of mortality accounted for by NCDs (and CVDs), which is why we expect stronger evidence on the impact of CVD on income growth for high-income countries than for low- and middle-income countries.<sup>10</sup>

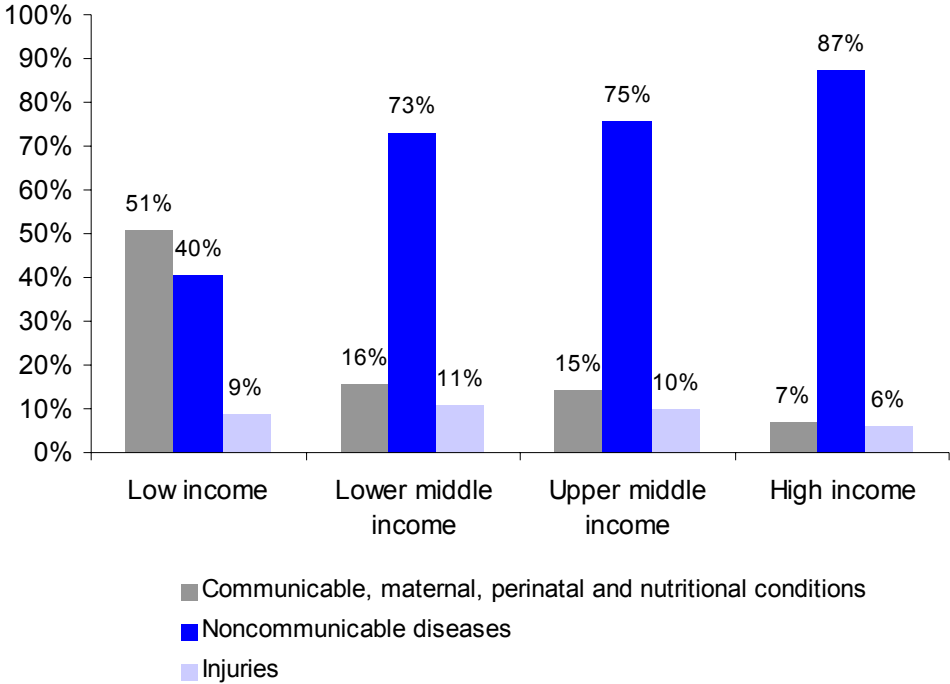
Second, a very large share of NCD and CVD mortality is actually occurring well before retirement age (see **Table 1**). For example, 31% of all CVD caused death falls into the age group younger than 65 years, even in low- and middle-income countries. When taking into

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<sup>10</sup> Figure 1 only gives a recent snapshot of the disease burden at one point in time. It is important to emphasise that recent trends unambiguously show the morbidity and mortality burden caused by CVDs (and NCDs) progressing at an increasing speed not only in high-income countries but also in developing countries (Jamison et al, 2006).

consideration that death is usually preceded by a period of morbidity, the disease burden falls even more into the working age group. Almost two thirds of the mortality and morbidity (measured in Disability Adjusted Life Years – DALYs) due to CVDs occurs before age 65.

**Figure 1: Causes of death by World Bank income group (2002)**



Source: WHO (2006)

**Table 1: Share of disease burden falling before age 65, low and middle income countries**

Disease category	Deaths	DALYs
All NCDs	39%	81%
Cardiovascular disease	31%	62%
<i>Communicable, maternal, perinatal and nutritional conditions</i>	89%	98%

Source: WHO (2006).

In light of this purely epidemiological evidence, it is not hard to imagine that there could well be a significant macroeconomic impact on labor supply, productivity, population growth, physical and human capital accumulation, and income in levels and growth rates. Labor



supply is directly affected since morbidity usually precedes death. Affected workers do not only drop out of the workforce, but impose a health care burden on others reducing income and growth, for example, through distortionary taxation or health insurance premia to finance health care. Workers in ill-health may still work but at a reduced effort level, since the disease may affect their work ambition and durability, which in turn reduces average worker productivity. Worker productivity has not only an impact on the level of income but also on its growth rate, because worker productivity affects immediately the return on investment, capital accumulation and eventually income growth. Not only morbidity, but also life expectancy itself may have an impact on income in levels and growth. Since education is a fixed cost in the early stage of life that needs to be covered by education-wage premia over all later stages of working life, the return to human capital accumulation is negatively affected by a reduction in the expected working-age lifetime (Kalemli-Ozcan et al. 2000). A lower rate of human capital accumulation then again leads to reduced income growth. An increase of life expectancy also influences the fertility choice according to Soares (2005) which in turn affects the population growth rate and income growth (in a world with less than perfect international capital markets).

Figure 1 has suggested that despite its unquestionable global importance there may be significant differences between developing and developed countries in that communicable diseases are relatively more important for low-income countries and non-communicable diseases relatively more important for high-income countries. Since the study of Bhargava et al. (2001) pays particular attention to such structural breaks, we adopt its methodology and its control variables as a starting point.

**Table 2: Data-description**

Variable Abbreviation	Description	Source
GDP p.c.	GDP per capita in PPP	Penn World Tables 6.0 (rgdpl)
Openness	Sum of exports and imports divided by GDP	Penn World Tables 6.0. (openk)
Open 1965-90	Dummy with value one if country has granted access to its home market during 1965 until 1990	Gallup and Sachs (1998)
Investment rate	Net investment per GDP	Penn World Tables 6.0 (ki)
Adult mortality rate	Probability of a 15-year-old of dying before age 60.	World Develop- ment Indicators
Fertility rate	Number of children born to a woman that lives to the end of her child-bearing age	World Develop- ment Indicators
Secondary education	Share of working age population with secondary schooling or higher education	Barro and Lee (2000)
Density of doctors	Number of physicians per 1000 inhabitants	World Develop- ment Indicators
Injury mortality rate	Mortality rate by injury per 1000 inhabitants in working age	WHO mortality database
CVD	Cardiovascular Disease mortality rates per 1000 inhabitants in working age	WHO mortality database
Non-communicable disease mortality rate	Non-communicable disease mortality rates per 1000 inhabitants in working age	WHO mortality database
Old age dependency ratio	Old age (65 to over 95) on total population	World Development Indicators

We follow Bhargava et al. (2001) in applying panel data in five year intervals from the year 1960 until 2000 to avoid business cycle regularities to influence our results. The data are PPP-adjusted GDP per capita and its 5-year time lag, the average degree of openness in between

1965 and 1990 from Gallup and Sachs (1998), and 5-year lagged values of the investment rate, fertility rate, adult mortality rate and our variable of interest: CVD mortality rate of the working age population. We also use an interaction term of CVD mortality with lagged GDP per capita. Instead of the adult mortality rate, Bhargava et al. (2001) have used the adult survival rate, which is not publicly available. The data description, variable abbreviations, and sources are summarized in Table 2 that contains also additional variables that entered our analysis beneath.

Table A1 in the appendix gives summary statistics of each variable. Since we estimate a dynamic panel regression, and it is known that a standard OLS estimator is inconsistent in this case, we pay particular attention to the estimation methodology that is described next.

### 3 Empirical methodology

A panel growth regression model can be written in a general form as follows:

$$y_{it} = \sum_{l=1}^p \alpha_l \cdot y_{i,t-l} + \beta_0 + \beta_1 \cdot x_{i,t-1} + \beta_2 \cdot z_{i,t-1} + \eta_i + \varepsilon_{it}, \quad \text{for } t=1+p \dots T, \quad (1)$$

where  $y_{it}$  is GDP per capita of country  $i$  at time  $t=1..T$ ,  $x_{it}$  is a vector of pre-determined control variables,  $z_{it}$  is a vector of exogenous control variables,  $\eta_i$  an i.i.d. country-specific random effect,  $\varepsilon_{it}$  is the usual i.i.d. error term (possibly heteroscedastic but not autocorrelated), and

$\beta_j, j = 0,1,2$ , and  $\alpha_l, l = 1, \dots, p$ , are the regression coefficients with  $\left| \sum_{l=1}^p \alpha_l \right| \leq 1$ .<sup>11</sup> The initial

value of the dynamic process is assumed to be an i.i.d. random deviation from the steady state value. The time dimension of panel growth regressions was typically chosen to be 5 or 10

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<sup>11</sup> If a unit root exists, i.e.  $\left| \sum_{l=1}^p \alpha_l \right| = 1$ , then the Blundell and Bond (1998) estimator is still consistent but no longer efficient, as has been shown in Binder et al. (2003). The convergence rate can be obtained as  $\alpha_1 - 1$  if  $p=1$ .

year periods to avoid short term business cycle components to influence the convergence rate (see e.g. Islam, 1995, Caselli et al., 1996, and Bond et al. 2001).

A pre-determined (endogenous) covariate is defined as a random variable that is allowed to depend on past values of GDP per capita, but not on future GDP per capita. When allowing for pre-determined variables  $x_{it}$ , the reverse causality from past values of GDP to  $x_{it}$  is fully controlled for and the regression coefficient measures only the marginal effect from contemporary values of  $x_{it}$  to future values of GDP per capita. Hence, a regression coefficient of a pre-determined variable measures causality in a Granger sense.

For example, when assuming our variable of interest CVD to be pre-determined, we take into consideration that the population in richer societies has a higher probability of CVD. The regression coefficient, however, filters this reverse causality, and measures only the impact that CVD today has on future economic growth assuming that today's occurrence of CVD is not depending on the prospect of future growth. However, the last assumption is not a binding constraint for our estimations, since it is not perceivable that life style adjusts before a change of income.

Nickel (1981) has shown that an FE-estimator on (1) is inconsistent, when the time dimension is small, because there is a correlation of the group mean of the error term with the lagged dependent variable. Moreover, Trognon (1978) has shown that an OLS-estimator is also inconsistent, because the lagged dependent variable is correlated with the random effect. The direction of bias is generally not known without further information on the covariance matrix of all variables, although closed form solutions of the bias term exist.

Arellano and Bond (1991) recommend a one-step GMM-system estimator built on the following generalized moment conditions

$$E[W_i' \Delta \varepsilon_i] = 0 \quad (2)$$

with the instrument matrix

$$W_i = \begin{bmatrix} [y_{i1} \ x_{i1} \dots \ x_{ip} \ z_{i1} \dots \ z_{iT}] & 0 & \dots & 0 \\ 0 & [y_{i1} \ y_{i2} \ x_{i1} \dots \ x_{i,p+1} \ z_{i1} \dots \ z_{iT}] & \dots & \dots \\ \dots & \dots & \dots & 0 \\ 0 & \dots & 0 & [y_{i1} \dots \ y_{i,T-p-1} \ x_{i1} \dots \ x_{i,T-1} \ z_{i1} \dots \ z_{iT}] \end{bmatrix}$$

and  $\Delta \varepsilon_i$  denotes the  $(T-1-p)$ -dimensional vector of first-differenced error terms. All elements of the matrix  $W_i$  are valid instruments, because the lagged values of 2<sup>nd</sup> and higher order of the dependent variable are not correlated with the first differenced error term, where first-differencing whipes out the random effect.

Blundell and Bond (1998) point out a weak-instrument problem (see Staiger and Stock, 1997, and Hahn and Hausman, 2002) that is most severe, whenever the dependent variable follows a near-unit root process or whenever the variance of the random effect is large relative to the variance of the error term, and suggest the additional moment conditions

$$E[\Delta y_{i,t-1}' (\eta_i + \varepsilon_{it})] = 0, \quad (3)$$

for  $t=p+1, \dots, T$ . The moment conditions in (3) hold, because lagged first differences of the dependent variable have differenced out the random effect and are not correlated with the contemporary error term.

As usual in GMM estimation, the generalized moments (2) and (3) are replaced by their sample estimates and the GMM criterion function over all moment conditions is minimized with respect to all regression coefficients. Because the moment conditions (2) and (3) imply that observations are taken twice (in level and first differences), the applied solution is

identical to a system GMM estimator on a regression system of variables in levels and first differences.

The covariance matrix of the GMM criterion function depends on the regression estimates. Hence, a heteroscedasticity consistent one-step estimator replaces the estimated covariance matrix with an approximation (see Roodman, 2004, for details). A heteroscedasticity consistent two-step estimator uses the estimates of the second step to obtain an estimated moment covariance that is used to minimize the GMM criterion function again and obtain the more efficient two-step estimator. However, Arellano and Bond (1991) point out that there is a severe small sample bias of the covariance matrix of the regression coefficients, when applying the two-step GMM system estimator and recommend to use the one-step estimator. However, Windmeijer (2000) has developed a small sample correction to the regression coefficient covariance matrix. Hence, we will apply both the one-step system GMM estimator and the two-step system GMM estimator with small sample correction.

Bhargava et al. (2001) apply the Bhargava and Sargan (1983) dynamic panel estimator. While this estimator has similar statistical large sample properties and we are not aware of small sample Monte Carlo study comparisons, we prefer the Blundell and Bond (1998) estimator both, because its software is available for free<sup>12</sup> and a larger number of applications exist such that more experience has been gained in applications.

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<sup>12</sup> We thank David Roodman for his STATA 8.0 module xtabond2, which is used in this study. Of course, we take sole responsibility for any possible remaining errors in the program.

## 4 Results

We begin with a GMM one-step system estimation of the baseline specification of Bhargava et al. (2001), i.e. we regress GDP per capita on its 5-year time lag<sup>13</sup>, the average degree of openness in between 1965 and 1990 from Gallup and Sachs (1998), and 5-year lagged values of the investment rate, fertility rate, adult mortality rate and our variables of interest: CVD mortality rate of the working age population and its interaction term with lagged GDP per capita. Instead of the adult mortality rate Bhargava et al. (2001) have used the adult survival rate, which is not publicly available. A dummy variable for tropic countries as well as an interaction term of the survival rate and the lagged income level is left out, too, because both variables turned out to be insignificant for our sample, which is due to smaller coverage of observations caused by missing observations on CVD. There are few countries in our sample that are below the income threshold reported in Bhargava et al. (2001) which splits the sample into one group of countries with a positive and another with a negative impact of the survival rate on growth. Moreover, there are few countries in tropic regions that report CVD mortality rates.

The first specification in Table 3 uses the 1-step GMM-system estimator with the first and second lag of the instrumented covariates as instruments. When choosing the lag number of instruments, a trade-off was taken into consideration between increased efficiency of additional instruments and an aggravation of the weak-instrument problem, if additional time lagged instrumental variables are only weakly correlated with the instrumented covariate.<sup>14</sup>

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<sup>13</sup> This is equivalent to estimating the growth rate of per capita income on its initial level. One just needs to subtract one from the coefficient on the initial income level, while all standard errors remain unaffected.

<sup>14</sup> See Hahn and Hausman (2002) and Staiger and Stock (1997) for discussions of the weak-instrument problem and Arellano and Bond (1991) and Blundell and Bond (1998) for a discussion of increased efficiency of additional instruments.

Apart from the lagged dependent variable, we also use GMM instruments for the investment rate, which is suspected by Bhargava et al. (2001) to be endogenous.

**Table 3: Full Sample**

Dep. income p.c.	GMM CVD exogenous (1)	GMM CVD endogenous (2)	GMM-small sample correction (3)	OLS (4)	FE (5)
Lagged income p.c.	1.71*** (5.27)	1.26*** (6.35)	1.34*** (5.96)	1.06*** (11.42)	1.06*** (7.41)
Open 1965-90	0.02* (1.69)	0.01** (2.20)	0.01 (1.59)	0.14*** (5.99)	-
Lagged Investment rate	0.12** (2.39)	0.09*** (2.78)	0.10*** (2.99)	0.06** (2.44)	0.06 (0.93)
Lagged fertility rate	-0.15 (-1.37)	-0.09 (-1.46)	-0.10* (-1.86)	-0.05* (-1.90)	-0.26*** (-4.60)
Lagged adult mortality	-0.25** (-2.52)	-0.20*** (-2.73)	-0.20*** (-2.70)	-0.06 (-1.46)	0.04 (0.73)
Lagged CVD	2.02*** (3.31)	0.92** (2.46)	1.05** (2.48)	0.41** (2.20)	0.83*** (2.77)
Interaction	-0.22*** (-3.28)	-0.09** (-2.27)	-0.11** (-2.35)	-0.04** (-2.08)	-0.10*** (-2.98)
Income threshold	9719	16178	14475	14408	4578
Hansen-Test	0.19	0.96	0.96	-	-
AR1	0.06*	0.08*	0.07*	-	-
AR2	0.05*	0.10*	0.09*	-	-
# observations	286	320	320	341	341

Remarks: z-values applying Arellano (1987) covariance matrix in parenthesis; \*, \*\*, \*\*\* denotes significance level at the 10, 5, and 1 % level respectively; AR1 test on first order autocorrelation – p-value; AR2 test on second order autocorrelation p-value; heteroscedasticity consistent Hansen-test on overidentifying restrictions p-value; Constant term not reported;

The estimated coefficients have all the expected signs. However, some control variables are not or only weakly significant. The coefficient on lagged income appears fairly large with



large standard error. However, one needs to be careful not to mistake this coefficient for the convergence rate, since lagged income also appears in the interaction term.

Turning to our variable of interest, the CVD variable and its interaction term are both significant at the 1 % level. At a threshold level of income below 9719 PPP US \$, there is a positive impact of CVD mortality rates on growth, and above this threshold there is a negative relation. Such an income threshold is also found in Bhargava et al. (2001) with respect to adult survival rates. However, the income threshold for adult survival rates is much lower separating poor from middle and high income countries, while the threshold for CVD separates rich from middle and poor income countries in the classification of the World Bank.

One needs to be careful in interpreting the relation of CVD and growth for three reasons. First, the test of second order autocorrelation of the estimated error term is weakly significant. This may render the covariate coefficient estimates inconsistent. Second, there may be reverse causality. Higher income growth may induce more luxury good consumption, which in turn could increase CVD mortality rates. Third, it may still be the case that there is no significant relation in one of the two sample halves, because the inclusion of the interaction term allows for a limited non-linearity only. In what follows we address all of these three possibilities.

First, we allow for endogeneity of the CVD variable and its interaction term in specification (2). This excludes any inconsistency from reversed causality as long as there is no correlation of future values of CVD on today's error term in estimating growth. The income elasticity of CVD mortality rates is reduced by half. Hence, reversed causality is likely to exist, but cannot explain the correlation of CVD and economic growth alone.

So far, the estimates rely on large sample theory. In specification (3), we apply the more efficient two stage GMM estimator with small sample correction from Windmeijer (2000). Again, we do not observe any significant changes in results and obtain very similar estimates in magnitude.

To finally have a comparison, we also apply in specification (4) an inconsistent OLS estimator and in specification (5) an inconsistent FE estimator. The t-values in parenthesis are calculated in both cases using an autocorrelation and heteroscedasticity consistent covariance matrix estimate of Arellano (1987). Since the openness variable from Gallup and Sachs (1998) has no time variation, it drops out in the FE specification. While coefficients are different in magnitude indicating the presence of a bias, the CVD variable and its interaction term remain significant in all specifications.

When considering the implied income threshold at which there is a positive/negative correlation of CVD and income growth, the GMM estimators are in between the OLS and FE estimator. The implied threshold of the GMM estimators is roughly in the range that divides rich OECD and non-OECD from middle income countries according to the World Bank classification. For this reason, we continue analysing the rich-country sub-sample.

When restricting the sample size, different control variables become relevant. Since the rich countries have unconstrained access to the international capital market, the fertility rate and the investment rate are no longer determinants of income growth. The fertility rate influences income growth, because, when entering the labor market, the newly born cohort needs to be endowed with capital to keep the capital intensity constant. This additional need for capital formation reduces the rate of net investment at a given savings rate in a closed economy. However, whenever there is access to an international capital markets, the additional capital

formation can be financed from abroad without being constrained by the domestic savings rate. In a similar vein, the investment rate is equal to the savings rate in a closed economy. Hence, economies with smaller savings rates invest less and grow less (Feldstein and Horioka, 1980). This relation breaks down, when there is access to the international capital market.

**Table 4: OECD Sample**

Dep. income p.c.	GMM CVD endogenous (1)	GMM CVD endogenous (2)	GMM CVD exogenous (3)	GMM CVD endogenous (4)	GMM small sample correction (5)	OLS (6)	FE (7)
Lag income p.c.	0.77*** (24.75)	0.70*** (5.05)	0.52*** (5.58)	0.67*** (5.77)	0.54*** (3.11)	0.83*** (8.73)	0.51*** (3.93)
2 <sup>nd</sup> lag income p.c.	-	0.03 (0.24)	0.75*** (3.83)	0.49*** (2.88)	0.52*** (3.53)	0.32*** (2.77)	0.31** (2.49)
3 <sup>rd</sup> lag income p.c.	-	-	-0.50*** (-3.07)	-0.37*** (-2.68)	-0.30*** (2.03)	- (-3.70)	-0.12 (-0.82)
Lagged Openness	0.03 (1.61)	0.04** (2.49)	0.05*** (2.96)	0.04** (2.38)	0.03 (1.47)	0.03*** (3.13)	0.08 (1.01)
Lag share of secondary schooling	0.00 (0.12)	0.10** (2.49)	0.11*** (3.61)	0.09*** (3.06)	0.09** (2.24)	0.04* (1.91)	0.02 (0.80)
Lagged CVD	-0.10*** (-3.08)	-0.07** (-2.41)	-0.07*** (2.57)	-0.08*** (3.31)	-0.09** (2.20)	-0.03 (-1.38)	-0.10*** (-2.60)
Hansen-Test	0.99	0.99	0.96	0.99	0.99	-	-
AR1	0.00***	0.00***	0.01	0.00	0.03	-	-
AR2	0.01***	0.01***	0.09*	0.99	0.98	-	-
# observations	164	164	141	143	143	143	143

Remarks: z-values applying Arellano (1987) covariance matrix in parenthesis; \*, \*\*, \*\*\* denotes significance level at the 10, 5, and 1 % level respectively; AR1 test on first order autocorrelation – p-value; AR2 test on second order autocorrelation p-value; heteroscedasticity consistent Hansen-test on overidentifying restrictions p-value; Constant term not reported;

Indeed, the investment rate and the fertility rate turn out to be insignificant on the sub-sample of rich countries. In addition, the openness variable of Gallup and Sachs (1998) is also insignificant, which may be due to the lack of time variation of this variable. Instead, we apply the openness variable from the Penn World Tables, which is defined as the sum of imports and exports divided by GDP. Since GDP is part of the construction of the openness variable from the Penn World Tables, we will treat this variable as endogenous in GMM estimations. In addition, the share of the working age population with secondary education becomes a significant determinant of income growth, because human capital - unlike physical capital - is typically not mobile internationally.<sup>15</sup>

Table 4 displays the results on the rich-country sub-sample. First, we note that in specification (1) the CVD variable is highly significant with negative sign, when applying a GMM-one step estimator with endogenous CVD variable. Unfortunately, the autocorrelation test of second order indicates severe problems of autocorrelation, which renders the estimator inconsistent. To avoid this misspecification problem, we add in specification (2) an additional time lag of the dependent variable as covariate, but the autocorrelation problem remains. Since there is an additional lag of the dependent variable, all instruments are lagged by one more time, too, to avoid correlation of the instruments with the error term.

Only, when we also add the third 5-year time lag of the dependent variable in specification (3), the problem of autocorrelation of the error term is substantially reduced. Still, when the CVD variable is assumed exogenous, there is autocorrelation at the 10% significance level. However, specification (4) endogenizes the CVD variable and the autocorrelation problem disappears. Hence, we consider this specification as our baseline, since it seems to be well specified: besides lack of autocorrelation of second order, the Hansen test of overidentifying

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<sup>15</sup> Bhargava et al. (2001) have pointed out that human capital measures are not significant in a panel data set with more countries.

restrictions is passed and the estimated coefficients are all significant with expected signs. For example, larger openness and a larger stock of human capital increase income growth.

Our variable of interest, CVD, is also highly significant at the 5% level. An increase in the mortality rate by 1% decreases the per capita income growth rate by about one promille point. This is a small amount in terms of growth rates, but a large amount in money terms when summed up over the lifetime of an economy.

Since the number of rich countries is rather small, it is important to apply the small sample correction of Windmeijer (2000) to the covariance estimates. Specification (5) displays the results. While openness becomes insignificant mainly due to a larger standard error, all other estimates remain significant.

Finally, we report for the sake of comparison the estimates of an inconsistent OLS and FE estimator in specifications (6) and (7), respectively. While the magnitude of coefficients varies at times substantially indicating a presence of bias, the qualitative results remain the same, although the CVD variable is insignificant in the OLS specification.

Since there seems to be a cyclical component in GDP growth data of a rather large wave length which seems puzzling at first glance, we also investigate the same data annually. To be able to do this, we need to interpolate the secondary schooling share variable first. Then, we find that autocorrelation disappears only if we include time lags of the dependent variable for the first four years, the 10<sup>th</sup> year and the 15<sup>th</sup> year both for the entire sample and the rich-country sub-sample<sup>16</sup>. Hence, the rather large cyclical pattern in the data is not an artefact of choosing five-year growth rates in the previous analysis. The first four year time lags

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<sup>16</sup> The results are not reported, but available from the authors upon request.

probably capture business cycle components of GDP growth. The larger lag length of 10 and 15 years may be explained by Kondratieff-type technology waves.

Next, we investigate the omitted variable problem. It may be that CVD mortality rates are correlated to an omitted variable that causes itself economic growth. For example, CVD mortality rates may be larger in societies with bad health systems which in turn may decrease the working moral, productivity, rate of return to investment, and eventually economic growth. Alternatively, it may be the case that the quality of the health system in general is itself a proxy variable for the quality of public intermediate goods supply. Finally, it may be a proxy for the innovative capability of an economy.

For this reason, we apply a number of alternative measures of the health system as control variables. Since an additional variable implies an additional regression coefficient that must be estimated, the number of moment restrictions will be insufficient to have several time lags of the instruments. In order to avoid inefficiency of the estimates, when using only one instrument, we refer to the specification, when the CVD variable is assumed exogenous and the two first valid lags of the instruments are applied. This procedure is justifiable, because we found in specifications (3) and (4) of Table 4 that the magnitude of the coefficient of the CVD variable is very similar in both cases.

In Table 5, specification (1), we add the control variable adult mortality rate that has been used before already in Table 3. Next, we add the logarithm of the number of doctors per population in specification (2). The density of doctors can be thought of as a control for the coverage of the health system. In specification (3), we add the logarithm of the lagged mortality rate caused by injuries to control alternatively for the quality of the health system.

Specifications (4) and (5) eventually control for the lagged logarithm of the old age

**Table 5: Control variables OECD sample**

Dep. income p.c.	GMM CVD exogenous (1)	GMM CVD exogenous (2)	GMM CVD exogenous (3)	GMM CVD exogenous (4)	GMM CVD exogenous (5)
Lagged income p.c.	0.52*** (5.23)	0.61*** (7.40)	0.54*** (5.08)	0.41*** (3.46)	0.44*** (4.81)
2 <sup>nd</sup> lag income p.c.	0.73*** (3.95)	0.60*** (3.56)	0.72*** (3.80)	0.77*** (4.22)	0.76*** (4.55)
3 <sup>rd</sup> lag income p.c.	-0.48** (2.95)	-0.40** (2.27)	-0.49*** (2.82)	-0.37* (1.92)	-0.43*** (-3.32)
Lagged openness	0.05 (2.75)	0.04 (2.32)	0.05*** (3.15)	0.03 (1.13)	0.07*** (3.17)
Lagged secondary schooling share	0.11** (3.69)	0.09** (2.55)	0.11*** (3.67)	0.10*** (2.94)	0.10*** (3.80)
Lagged CVD	-0.07** (1.96)	-0.07** (2.44)	-0.07** (2.03)	-0.07** (2.06)	-0.19*** (2.60)
Lagged mortality rate	-0.02 (-0.28)	-	-	-	-
Lagged density of doctors	-	-0.04 (0.99)	-	-	-
Lagged injury mortality rate	-	-	-0.01 (-0.13)	-	-
Lagged old age dependency ratio	-	-	-	-0.13* (1.76)	-
Lagged non- communicable disease mortality rate	-	-	-	-	0.23* (1.78)
Hansen-Test	0.97	0.97	0.95	0.98	0.98
AR1	0.02**	0.01***	0.01**	0.03	0.02
AR2	0.11	0.40	0.07*	0.10*	0.07
# observations	141	130	141	141	141

Remarks: z-values applying Arellano (1987) covariance matrix in parenthesis; \*, \*\*, \*\*\* denotes significance level at the 10, 5, and 1 % level respectively; AR1 test on first order autocorrelation – p-value; AR2 test on second order autocorrelation p-value; heteroscedasticity consistent Hansen-test on overidentifying restrictions p-value; Constant term not reported;

dependency rate, which may control for the innovative potential of an economy, and the lagged value of the mortality rate caused by non-communicable diseases to ensure that indeed cardiovascular disease is responsible for the relation with economic growth.

In all cases, the CVD variable remains significant at the 5% level, while most of the control variables are not. The only exception is the old age dependency rate. Aging economies tend to grow slower. Overall, weak problems of autocorrelation remain in some specifications. Since the estimated coefficient of CVD remains remarkably stable over all specifications (with and without weak autocorrelation problems), we do not observe any substantial bias of its estimates even in the presence of weak autocorrelation.

We also tried all estimates of Table 5 assuming the CVD variable to be endogenous<sup>17</sup>. The CVD variable was always highly significant, but some other control variables became insignificant occasionally and some signs of control variables were not as expected.

Finally, we investigate whether the CVD variable really does have the opposite sign in the sub-sample of middle and low-income countries (results not reported, but available on request). However, we find that no significant relationship exists in this sub-sample. Hence, any negative impact of CVD on economic growth is only significant if income levels are sufficiently high.

## 5 Conclusion

The present paper has provided what is to the best of our knowledge the first attempt to assess the impact of cardiovascular disease on economic growth, using a growth regression framework. We used state-of-the-art methodology to address the most salient technical

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<sup>17</sup> The results are not reported but available from the authors upon request.



problems that could be addressed. There remains, however, a general concern about the application of the growth regression methodology (Durlauf, 2001). Data limitations matter, too: CVD mortality data, which we constructed on the basis of the WHO Mortality Database, is missing for many low-income countries, and where data is available, it is of significantly worse quality in the low- and middle-income countries compared to the high-income countries (WHO 2006). Hence our findings for the latter group of countries can be considered much more reliable than those for the low- and middle-income countries.

Bearing in mind those reservations, the results suggest that CVDs are bad for growth, but only once the countries have reached a fairly high level of per capita income. It is not entirely surprising to note that CVDs have no statistically significant role in explaining growth differences between rich and poor countries in the time period examined (1960-2000). Too many other important factors distinguish the two country groups, and CVDs have only been emerging quite strongly in the later part of this period in developing countries. The result should therefore not be misinterpreted as a justification for complacency in the developing countries facing the fast growing burden of CVDs. Concern as for the potential future economic impact of CVDs in developing countries is particularly warranted in light of the fairly reliable prediction that not only the speed of the CVD epidemic in developing countries is likely to exceed that previously experienced by high-income-countries, but also that its adverse impacts are likely to be felt more strongly in the developing world (Schmidhuber and Shetty, 2005).

Our results also contribute to the relatively small literature on the role of health as a determinant of growth in high-income countries. As briefly mentioned above, the empirical evidence on the role of health in economic growth in rich countries has produced rather unsatisfactory and mixed results, not least depending on the choice of health proxy. The very

few studies that used public health expenditure as a proxy for health did find a significant positive contribution to economic growth in high-income countries. However, in light of the poor empirical link between health expenditures and health outcomes (Anderson and Poullier, 1999), one may have reason to question the interpretation of the results (Rivera and Currais (1999a, b), Knowles and Owen (1995, 1997)). In contrast, studies that used life expectancy as a proxy for health generally failed to find a significant impact on growth, and where the impact was significant, it was commonly of a negative sign.

Our findings suggest that part of the ambiguous results may indeed be due to the choice of health proxy. As life expectancy varies very little between rich countries, it is not surprising to find that its explanatory power is highly limited. CVD-mortality (at working age) does vary more markedly between rich countries and therefore represents a more “appropriate” indicator to quantify existing health differences between rich countries. Nevertheless, the present study has only taken a first step towards an improved understanding of the “true” role of health in rich countries’ medium and long term growth performance. For certain, in order to better understand the contribution of health to economic development in rich countries, there is a critical need to go beyond the most general health indicators that have traditionally been applied in the developing country literature.

## Appendix

**Table A 1: Summary Statistics – Full sample and Rich Country Sample**

Variable name	# observations	Mean	Std. dev.	Min	Max
GDP p.c.	341	11365.32	6916.805	1369.589	33308.4
1 <sup>st</sup> lag GDP p.c.	341	10109.32	6229.086	1121.392	28409.62
Open 1965-90	341	0.5822242	.4552672	0	1
Investment rate	341	20.90187	8.594127	3.249018	68.34639
Fertility rate	341	2.968865	1.572043	1.18	7.4168
Adult mortality rate	341	.011316	.0044232	.0052631	.0375629
CVD mortality rate	341	134.7982	65.41324	32.61629	390.1721
Openness	143	60.18594	47.53438	10.86745	301.3148
Secondary education – total stock	143	36.58671	12.75781	6.8	69.6
Density of physicians	135	2.079481	.7873906	.6569	4.7
Injury mortality rate – working age	143	55.79993	16.82787	26.05	101.93
Old age dependency rate	143	.1191733	.0299058	.033502	.1777936
Non-communicable disease mortality rate	143	292.5629	64.62071	160.1588	470.0503

### Data coverage

#### Baseline Specification for Table 3 – Full sample.

Argentina, Armenia, Australia, Austria, Azerbaijan, Belgium, Bulgaria, Belarus, Belize, Brazil, Barbados, Canada, Switzerland, Chile, China, Colombia, Costa Rica, Cuba, Czech Republic, Germany (united after 1990), Denmark, Dominican Republic, Ecuador, Egypt, Spain, Estonia, Finland, France, UK, Greece, Guatemala, Hong Kong, Honduras, Croatia, Hungary, Ireland, Iceland, Israel, Italy, Jamaica, Japan, Kazakhstan, Kyrgyzstan, Republic Korea, Sri Lanka, Lithuania, Luxembourg, Latvia, Moldova, Mexico, Mauritius, Netherlands, Norway, New Zealand, Panama, Philippines, Poland, Puerto Rico, Portugal, Paraguay, Romania, Russia, Singapore, El Salvador, Slovenia, Sweden, Syria, Thailand, Trinidad and Tobago, Ukraine, Uruguay, USA, Venezuela.

#### Baseline specification for Table 4 and 5 – high income and higher-middle income countries according to World Bank classification.

**Australia, Austria, Belgium, Canada, Czech Republic, Germany (united from 1990),**

**Denmark, Spain, Finland, France, UK, Greece, Hong Kong, Ireland, Iceland, Israel, Italy,**

**Japan, Netherlands, Norway, New Zealand, Portugal, Singapore, Sweden, USA.**

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