DEVDATTA RAY

Thesis analyzes the relationships between health status, health expenditures, health care technology, economic growth, and inequality on the global scale using econometric methods. Results show that at present poorest countries’ income gradient is still high, public health expenditures are more health promoting than private spending, the Kuznets’ hypothesis is valid in poor countries, and cancer mortality is less responsive than tuberculosis to global diffusion of health care technologies.
ESSAYS ON ECONOMIC GROWTH, HEALTH AND INEQUALITY IN DEVELOPED AND LESS DEVELOPED COUNTRIES
Devdatta Ray

ESSAYS ON ECONOMIC GROWTH, HEALTH AND INEQUALITY IN DEVELOPED AND LESS DEVELOPED COUNTRIES

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ABSTRACT

Dissertation highlights the interplay between income inequality, economic growth, and health. Health care technologies, health expenditures, and health statuses are considered along with other relevant ancillary variables in process of interaction. Unlike in developed countries, health and health care are underrated and poorly financed areas in the developing world. This dissertation, comparing the countries globally, gives a comparative picture of the global state of health based on chosen variables, years, countries, and methods.

In the first article, considering 148 countries for years 1970–2010 in the framework of a health-income relationship, the aggregation of individual concave income function on health is analyzed. Taking log of the mean incomes results in biased aggregate health effects. A new method is suggested that corrects the income effects in the right direction, i.e. they provide smaller parameter estimates than the biased approach. The results for income inequality, measured with the $\text{GINI}$ coefficient, indicate that the effects of inequality on health are still significant in the poorest countries but non-significant among rich countries after the year 2000.

In the second paper, effects of public and private health expenditures on life expectancy at birth and infant mortality are analyzed on a global scale with 195 countries in years 1995–2014. New dynamic panel model estimators show that public health expenditures are generally more health promoting than private expenditures. However, the health effects are not as large as primary education effects are.

The third paper analyses cancer and tuberculosis mortality rates as health status indicator with 144 and 196 countries respectively for the period 1970–2012. Methods of trend growth modeling and convergence analysis, found in economic growth empirics, are used to elucidate the effects of global catch-up of health care technologies through diffusion between more and less advanced countries. The results show that there is evidence of larger declining trend process in low income countries for both illnesses when compared to higher income countries. The speed of declining has been restrained in high income countries in recent decades. Both $\sigma$- and $\beta$-convergences are found to be present for tuberculosis rates. For cancer mortality, no clear evidence of $\sigma$-convergence is found. When technologies and socio-economic factors are added to the $\beta$-convergence analysis, the convergence rates are the largest in lower income countries for both illnesses.

In the fourth paper, a simultaneous three equation model is specified between GDP per capita ($\text{GDPc}$) level, infant mortality rate, and health expenditures for
194 countries in years 1990–2014. GMM-2SLS estimation results indicate that simultaneous decreasing infant mortality rate and increasing GDPc level effects are found in the sample with three income level country groups. Income effect on health expenditures has a unit elasticity value for all income groups. The Kuznets’ hypothesis is not rejected for poor countries with the proposed inverted U-shaped GDPc level function on GINI coefficients that also identifies negative income inequality effects on GDPc growth in all income groups. Thus, the low-income and high-inequality trap can still be present among the poorest countries.

Overall the thesis indicates that globally effects of income, inequality, and health care technology on health are still very significant in the poor countries. Public health expenditures are more health promoting than private expenditures. There exists a catch-up in health technology effects on health statuses like tuberculosis and cancer in low income countries. Even today the Kuznets’ hypothesis may be present for poor countries. Results indicate that the low-income and high-inequality spiral can be avoided by raising health expenditures-GDP ratio and with cost effective health care technologies.

**Keywords:** health status, income health relationship, health expenditures, gross national income per capita, income inequality, health care technology diffusion, Kuznets’ hypothesis.
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Esseitä taloudellisesta kasvusta, terveydestä ja eriarvoisuuudesta kehittyneissä ja kehittyvissä maissa.
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TIIVISTELMÄ
Väitöksessä korostetaan tulojen eriarvoisuuden, taloudellisen kasvun ja terveyden välistä riippuvuutta. Terveydenhuollon teknologia, terveysmenot, terveyden tila ja niihin liittyvät täydentävät tekijät huomioidaan tässä vuorovaikutuksessa. Toisin kuin kehittyneissä maissa terveys ja terveydenhuolto ovat aliarvostettuja ja heikosti rahoitettuja kehitysmaissa. Tutkielmassa vertaillaan valtioita maailmanlaajuisesti antamalla kuva globaalista terveydentilasta ja valituista muuttujista eri vuosien, valtioiden ja metodien kohdalla.


Neljännessä arikkelissa tarkastellaan kolmen yhtälön simultaanimallin avulla bruttokansantuote per capitan, vastasyntyneiden kuolleisuuden ja terveysmenojen


Avainsanat: terveydentila, tulojen ja terveyden suhde, terveysmenot, bruttokansantuote per capita, tuloeriarvoisuus, terveyden huollon teknologian leviäminen, Kuznetsin hypoteesi.
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Finally (and certainly not the least), I would like to thank my mother, who through all my hardships stood with me, pushing and prodding me to finish my PhD. I thank her deeply for her love, compassion and imbibing in me the spirit to fight on. Words cannot express my gratitude to her.

Kuopio, February 2019
Devdatta Ray
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1 INTRODUCTION

1.1 BACKGROUND

The World Health Organization (WHO) defines health in its broader sense in its 1948 constitution as a “state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity” (WHO 2017c). Health status is a holistic concept that is determined by more than the presence or absence of any disease summarized by life expectancy, infant mortality or self-assessed health status. The determinants of health (WHO 2017d) include the social and economic environment, the physical environment and the person’s individual characteristics and behaviors. Specifically, in this context, we refer to income and social status. Higher income and social status are linked to better health, and greater the gap between the richest and the poorest people, the greater are the differences in health. Low income levels, absence of an environment of safe water and clean air, unhealthy workplaces, unsafe houses, deplorable work environment, and archaic community values contribute to bad health. Generally, the social determinants of health, especially incomes and inequalities, are mostly responsible for health inequalities seen within and between countries. Equity in health implies that ideally everyone should have a fair opportunity to attain their full health potential and no one should be disadvantaged from achieving this potential if it can be avoided (WHO EURO 2014). In contrast to this, health inequalities typically refer to individual differences in health status or in the distribution of it between different population groups, e.g. differences in morbidity between elderly people and younger populations or differences in mortality rates between people from different social classes.

The European Parliament estimated that losses linked to health inequities cost around 1.4 % of GDP within EU (WHO 2017a, b). WHO (2015a, b) provides examples of health inequalities and inequities between countries. These include e.g. IM as being 2 per 1000 live births in Iceland and over 120 per 1000 live births in Mozambique, or the lifetime risk of maternal death during or shortly after pregnancy as being only 1 in 17400 in Sweden but 1 in 8 in Afghanistan. Another example is that life expectancy at birth among indigenous Australians is substantially lower (59.4 for males and 64.8 for females) than that of non-indigenous Australians (76.6 and 82.0 respectively). Note that 87 % of premature deaths due to non-communicable diseases occur in low- and middle-income countries. Bad health and health care costs drain out household resources, often driving families into poverty, preventing development. In absolute terms from 1960 till today, the absolute gap between the average incomes of people in the richest and the poorest countries has grown by 135 % (Hickel 2015, 2016). The World Bank figures show that since 1960 the gap for Latin America has grown by 206 %, the gap for sub-Saharan Africa has grown by 207 %, and the gap for South Asia has grown by 196 %. On average, income inequality increased by 11 per cent in developing countries between 1990 and 2010 (UN 2017).

With respect to population health and the level of GDPc, the Preston curve (Preston 1975, 1996) is unfortunately still present, as differences in incomes between the countries are not less than it was forty years ago. Further, there has been only some progress in population health. The figures 1a & 1b below give the relationship between
a health status (e.g. infant mortality per 1000 born child) and the log of gross national income per capita (lnGNIc) in years 1970–1990 and 1991–2014 for 194 countries. In both periods, more income means less infant mortality, but at a given low income level in the more recent period 1991–2014 infant mortality is less than in the former period 1970–1990. This is due to health effects of non-income factors. As the average income levels have increased between the periods, the Preston curve in period 1991–2014 is below and less steep, especially with high income levels, when compared to the period 1970–1990 curve. Thus, at the general level higher incomes globally means better population health, but the marginal income effects are largest at low income levels. In other words, income distribution and income inequality have also health effects.

The dissertation focuses on the intricate process between income inequality, population health status, gross domestic product per capita and health expenditures. As health technology accounts for a large portion of expenditures, it is relevant to highlight its role in this multi-dimensional process. The outcomes of these mutually influencing variables determine the health situation and the socio-economic development at global, regional, and country specific levels. Unlike most previous research in this area, this thesis is not based on micro but on macro data, inclusive of health and economic aggregates. In this context, we bring in a novel four-quadrant setting with the goal to capture the macroeconomic determination of gross domestic product per capita level, economic growth, health status, health expenditure and effects of health care technology with given income inequality (see page 15). We contrast with it the results between the rich and the poor countries.

In this four-quadrant setting we study income inequality, health status, health expenditure, and income relationships in the framework of a modern rendition of the Kuznets’ hypothesis, meaning that the causation is going from inequality to the level and the growth of gross domestic product per capita. There are today hardly any studies that use Kuznets’ hypothesis to analyze the income inequality, health status and income relationships. Our research fills this void. The original Kuznets’ hypothesis had also the opposite causation, with first the level of gross domestic product per capita determining the income and wealth inequalities that were large enough to start the income growth process with capital formation. Later this growth
was hampered if the inequalities remained too large. We argue that health status and health expenditure with health care technology effects have important and often neglected roles in this inequality-income transmission process.

As the health-income relationship or the income gradient –hypothesis is at the heart of health economics, a proper testing approach at the aggregate level is conducted, unlike in the past, to avoid biased results. The thesis tackles this problem by introducing into the health-income relationship at aggregate level a correction term that filters out the artificial distribution effects from the health-income relationship estimation. The approach provides results on the income gradient –hypothesis that are less biased than the earlier ones seen in literature.

From the policy perspective, the relevance of this thesis is that it focuses on comparing countries with different health and development levels. The policy makers can get a view to alternative health outcomes and scenarios, thereby facilitating the fine tuning of their policies, depending on the state of their country’s economy. The results also help in forecasting outcomes for the future in poorer countries, when one would consider how the impacts of interest variables in the richer countries (e.g. health expenditure and health care technology) in the past have created the present positive health and development scenarios in these countries.

1.2 RESEARCH QUESTIONS

The main hypothesis of the thesis is that while income has positive health effects, income inequality has negative effects. In addition to these direct health effects income inequality has also indirect ones. As an economy’s income and its growth are conditioned by prevailing income distribution, there are indirect health effects from income distribution affecting GDPc growth. The dissertation focuses on the following questions:

(A) How increases in health expenditures can improve health status in developing nations with the presence of observed large income inequalities?

(B) How improved health status can also increase the developing country’s gross domestic product per capita on the one hand and reduce income inequality on the other?

In answering these questions, we note that public- and private health expenditures (HE’s) have different roles in relation to health status (HS). Different HE’s are responsible for improving health outcomes differently in the different country income groupings. Likewise, health care technologies (HCTs) can improve HS in poorer countries by diffusing the most efficient health care practices found in developed countries.

The main argument that is reinforced in the thesis is that in the long-run the positive outcomes in income-health relationship mean also less inequality (INEQ) and less inequalities mean better health and higher gross domestic product per capita (GDPc). The theoretical bridge that connects and enlightens the income distribution effect on income and its growth is the Kuznets’ hypothesis. The following figure sums up the hypotheses and the motivation of the thesis:
Quadrant I depicts the negative and monotone HS and income inequality (INEQ) relationship (HS–INEQ) with large given inequality. Empirically it is still valid for many poor and low-income countries (Deaton 2013). Quadrant II gives the Kuznets' hypothesis: At the high level of INEQ the GDPc level is low and less inequality does not sustain higher GDPc level. This is depicted with the non-linear Kuznets' curve that gives the poverty trap (point R1) with low health status. It shows that if a country is in this low-GDPc and high-INEQ state, it can escape from it by increasing INEQ that sustains higher GDPc, and after some high threshold level of INEQ, GDPc increases only if INEQ starts to decrease. Quadrants III and IV give the GDPc–HE and HE–HS relationships with shapes found in health economics literature. Now if the country increases its relative investment in health (i.e. HE/GDP-ratio increases: A moves to A*), and if the new level of health expenditures (HE) is utilized efficiently to raise the country's HS, then the country will escape from the low-GDPc high-inequality trap and will find herself in R2 with higher GDPc level and less INEQ than in R1.

Note that Kuznets' hypothesis is not necessary for our policy alternative (i.e., increases in HE/GDP and HS/HE ratios) to work-out successfully. The result with Kuznets' hypothesis speaks for a big jump in health policy with large exogenous productive investments in health services and technology (e.g. see Sachs 2004, Binagwaho 2014) with some short run equality costs to find the low-inequality but a higher GDPc-position.

Concerning the Quadrants III and IV the argument that income level and health care inputs – either at the personal or at the GDPc level – determines the health conditions of individuals and population is profound in the health economics literature (Grossman 1972). However, the heterogeneity of HS between both the individuals and the nations even at the same income levels asks for a more detailed relationship between health conditions and specific expenditures targeted to promote health care. The distinction between public and private expenditures here is important, since the former is mostly a policy variable determined by the political agenda by the state, while the latter reflects mostly the voluntary or individual choice-based demand for
health care. Both are determined in large extension by the general level of income in the country, but this does not rule out other factors affecting both the health conditions and health expenditures.

Our argument is that at least for poor countries, the resources devoted to public health provisions are more important for the population’s $HS$ than the private expenditures. The reason for this stems from the large $INEQ$ that prevails in most of the poor countries where there are sufficient incomes and private health expenditures only for a small fraction of the population. Note that globally health spending is also highly unequal. It is even more unequally distributed than national income of countries. Countries that spend little on health also have poorer health conditions. OECD countries have less than 20% of the world’s population, but account for over 85% of world’s spending on health today whereas the poorest three quarters of the world’s population account for only 7% of the world’s health expenditures. Looking across regions, at the other extreme, Africa contain about 12% of the world’s population, yet it uses 3% of the world’s health spending, while in Asia and the Pacific (including China) 25% of the world’s population account for only 2% of the world’s health spending (WHO Global Health Expenditure Atlas 2014).

Technology and technological innovation are crucial ingredients of health care. $HCT$s improve health and care delivery in many ways. Overall costs may rise with new $HCT$s, but we can improve health outcomes for a greater number of people. Long run benefits of using $HCT$s often outweigh short run costs. We defend the idea that the poorest countries can quite quickly adopt improvements in their $HS$s if they can afford and get access to technological advances found in rich countries. The social inequalities and the low level of health expenditures in poor countries however slower this important and urgently needed catch-up.

For example, cancers figure among the leading causes of morbidity and mortality worldwide, with approximately 14 million new cases and 8.2 million cancer related deaths in 2012 (WHO 2015a). At every stage of cancer diagnosis and treatment $HCT$s are used. Like cancer, $TB$ is a globally prevalent disease. In 2013, 9 million people fell ill with $TB$ and 1.5 million died from the disease (WHO 2015b). Between 2000 and 2013, an estimated 37 million lives were saved through $TB$ diagnosis and treatment with the help of $HCT$s (WHO 2015a). We argue that the convergence of $HS$ indicators like $TB$ or cancer mortality rates is an indication of global diffusion and efficient use of $HCT$s between the countries. In this sense different metrics of convergence are important to show the long run trends in disease mortality rates between nations.

The content of the dissertation is as follows. Chapter 1 introduces the dissertation, positions its background and explains the major research questions. Chapter 2 elucidates relevant literature with subsections on the interplay between inequality, economic growth, and health aggregates. Chapter 3 summarizes the four papers of the thesis while Chapter 4 provides the main conclusions of the thesis.
2 LITERATURE REVIEW

2.1 HEALTH, WEALTH AND ECONOMIC GROWTH

Good health leads to higher GDPc in the long run due to its impact on the population besides participation and productivity. The idea of health as a form of human capital has a long history (e.g. Mushkin 1962). Grossman (1972) developed a model in which illness prevented work so that the cost of ill health was lost labor time and worker productivity. However, a major difficulty in measuring the economic effect of health was the two-way causality between wealth and health (Smith 1999). Another difficulty was the lack of consensus on what was meant by “health”. Different studies used different health indicators. From the early 1990s, the role of human capital was almost universally regarded as being indispensable for economic growth. The groundbreaking analysis by Romer (1986) and Weil (2009) stressed also nutrition in a broader analysis of human capital. Fogel (1994), Barro and Sala-i-Martin (1995) were among the first in examining the relationship between economic growth and health rigorously. Without a labor force with some minimal levels of education (and health), a country was incapable of maintaining a state of continuous growth (Rivera and Curais 2003).

On a microeconomic level, many empirical studies have focused on the impact of health on productivity and wages. Different health indicators have been used that range from anthropometric measures such as weight, height and BMI to surveys that reported self-assessed HS (Rico et al. 2005). These lines of research are based on the idea that healthier workers are less susceptible to diseases, more alert, more energetic and consequently more productive, command higher earnings and life cycle consumption. On a macroeconomic level, both within country and cross-country analyses measure the effects of different inputs on total economic output. These inputs include human capital which is a combination of health and education. Today’s research analyzes health impacts on development to examine the channels through which health-related investments have a positive impact on economic growth and equity (Ruhm 2004).

Today, education affects economic outcomes and health affects education through two mechanisms. The first is the effect of better child health on school attendance, cognitive ability, and learning. The second mechanism is the effect of lower mortality and a longer prospective lifespan on increasing incentives to invest in human capital. Lower IM encourage parents to invest more resources in fewer children, leading to low fertility but high levels of human capital investment in each child (Kalemli-Ozcan et al. 2000).

The empirical literature on the effect of health on economic development (Bloom et al. 2004, Webber 2002, Acemoglu and Johnson 2007) focused mainly on the labor productivity effects of health on economic growth. On the other hand, the significance of the demographic variables in growth regressions had been asserted by many other authors (Bloom et al. 2004, Sala-i-Martin et al. 2004). The fertility equation was found e.g. by Schultz (1997), who considered the determinants of fertility to be education, income, employment, religion, nutrition, family planning, and child mortality. The research by Zhang and Zhang (2005) outlined a system of equations where in its simplest form education, investment, fertility and income were jointly determined, and LE was also featured as an explanatory variable in each of the system’s regressions.
Bloom et al. (2004) provided a summary of results of various studies that used LE as a proxy for health in the analysis of the direct effects of health on economic growth (e.g. Barro and Lee 1984, Bhargava et al. 2001, Barro and Sala-i-Martin 2004, Sachs and Warner 1997). In these studies, LE was shown to have a positive and significant effect on economic growth. Like Barro and Lee (2013), Bloom et al. (2004) controlled for workforce experience and showed that LE as a proxy for health had a significant positive effect on economic growth. Their results indicated that there was a real productivity effect of health on economic growth. The overlapping generation model (e.g. Chakraborty 2004, Kalemli-Ozcan et al. 2000) revealed that an increase in LE increased investment in education. The results were however affected by income distribution – both directly and indirectly via its health effects.

In his pioneering work, Preston (1980) attributed about half of the gain in LE in developing countries from the 1930s to the late 1960s to the combined effects of changes in income, literacy, and the supply of calories. A number of authors followed Pritchett and Summers (1996), who argued from cross-country regressions that income was more important than any other factor and endorsed policies that downplayed the role of public action in health improvement. According to this view, if countries’ economies were growing, the health of their inhabitants took care of itself. Contrary to this, many countries showed improvements in health with little or no economic growth and vice versa. For the two populous countries, India and China, there was a negative correlation between rates of economic growth and progress in reducing infant- and child mortalities (Cutler et al. 2006, Dreze and Sen 2002).

A new family of theories emerged in the 1980s that were better equipped to explain long-term economic growth (Romer 1986, Lucas 1988). Since technology determined growth in an endogenous way, these were known as endogenous growth models. In the neo-classical growth model, the notion of growth as increased stocks of capital goods was codified in the Solow-Swan growth model. In contrast to these Lucas (1988) and Romer (1986) considered technology as endogenous and incorporated a new concept of human capital which had increasing rates of return. The focus shifted on what increased human capital (mainly education, learning, and level of R&D activity). Thus, in an endogenous growth model output was produced by combining physical and human capital inputs, where agents could invest in health and education, and increase their health status (Van Zon and Muysken 2001, Galor 2011). However, one saw how large variations in health between rich and poor countries contributed to income differences.

The “health view” assumed that income differences between the countries were mainly caused by different health environments. The “income view”, on the other hand, assumed that most differences between the countries had their roots in aspects of production that were unrelated to health, e.g. in physical capital accumulation or technology. This school of thought believed that if poor countries were to raise their level of GDPc to the level of rich countries, they would also have the same level of health as rich countries (Weil 2009).

For the poorer countries investing in health often provided a means of escaping from the poverty trap. In the developing world, investing in health was synonymous with higher labor productivity and income. In more advanced economies fighting against obesity, alcohol abuse, smoking and drug addiction improved industrial output, lowered absenteeism and reduced losses of human capital (and social investment opportunities) for the economy (Bohr 2006). Resources that would otherwise be spent on chronic health conditions could be spent on other aspects of community welfare.
Figure 3. below shows the concave relationship between health and income (i.e. the absolute income hypothesis, AIH), meaning each additional unit of income improves an individual’s health, but by smaller amounts. As income increases because of economic growth, income and HE go up and so does HS but at a decreasing rate. Increase in income and HE mean movement along the curve while usages of better HCT shifts the curve upwards. Note that income can also be a function of health that is $y_i = g(h_i)$ where $g$ is a convex function by which health is transformed to income. Bad HS means reduced job participation and labor productivity, and these decrease further earnings, thereby obstructing the nation’s economic growth.

Bloom and Canning (2008) discussed mechanisms through which health could affect income, focusing on labor productivity, savings and demographic structure. The first was the role of health in labor productivity. The second was the effect of health on education. The third was the effect of health on savings. The fourth was the effect of population health on population numbers, and age structure. The major force behind health improvements were HCT improvements and public health measures. Growth regressions showed that the initial levels of population health were a significant predictor of future economic growth (Bloom et al. 2004).

Improvements in health and decreases in mortality rates could catalyze a transition from high to low rates of fertility and mortality – the demographic transition (Lee 2003). High birth and low death rates both generated population growth but had quite different effects on economic growth (Bloom and Freeman 1988, Kelley and Schmidt 1995), because they affected the age structure quite differently. Bloom e al. (2004) found that the demographic dividend increased the potential labor supply but its effect on economic growth depended on the policy environment. The following Table 1 gives the sum-up of relevant recent studies.
Table 1. Recent studies of health impact on economic growth

<table>
<thead>
<tr>
<th>Study</th>
<th>Some important components</th>
<th>Relevant results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acemoglu &amp; Johnson (2007)</td>
<td>From 1940–2000 etc. LE, GDP, GDPc, population data for 75 countries; also predicted mortality data constructs using pre-intervention mortality rates for various diseases and dates of global interventions.</td>
<td>Object: health affects economic growth when health was instrumented using initial disease burden and worldwide technological progress in disease-specific interventions; found no evidence that the large increase in LE raised income per capita as health improvements increased longevity and spurred population growth which strained other factors.</td>
</tr>
<tr>
<td>Acemoglu, Johnson &amp; Robinson (2003)</td>
<td>Health conditions and disease environment data for varying years and countries.</td>
<td>Health differences are not large enough to account for cross-country difference in incomes; variations in political, economic and social institutions are central factors; health does not have a direct effect on growth.</td>
</tr>
<tr>
<td>Akach &amp; Canning (2010)</td>
<td>Adult height, nutrition and IM data for 39 countries 1960–2004.</td>
<td>In Sub-Saharan Africa, despite declining IM, because of improved nutrition, reduced childhood exposure to diseases etc.; adult heights have not increased, and region has not experienced health and human capital increases.</td>
</tr>
<tr>
<td>Bhargava et al. (2001)</td>
<td>Health indicator’s (e.g. adult survival rate or ASR) effect on economic growth rates at 5-year interval panel data 1965-90 in developed and developing countries.</td>
<td>Effect of health on economic growth is larger in developing than in developed countries; ASR in poor countries reflect nutrition levels, smoking prevalence, health infrastructure etc.; differences in ASR in middle and high-income countries influenced by genetic factors, access to and costs of preventive/curative health care.</td>
</tr>
<tr>
<td>Crimmins &amp; Finch (2006)</td>
<td>Historical mortality and height data from cohorts born before the 20th century in four northern European countries.</td>
<td>Cohorts that underwent substantial improvements in IM in developed countries in the late nineteenth century were the same cohorts experiencing gains in adult height and increased productivity.</td>
</tr>
<tr>
<td>Deaton (2007)</td>
<td>Environmental determinants of height data for 43 developing countries 1993–2004.</td>
<td>Cross country average height not a good indicator of the country’s HS; could still be the case that changes in population height over time reflect changes in HS.</td>
</tr>
<tr>
<td>Finlay (2007)</td>
<td>Role of health in development analyzed through direct labor productivity effect and indirect incentive effect for 64 nations 1960–2000.</td>
<td>Accounting for simultaneous determination of growth, education, fertility etc.; labor productivity hypothesis asserts that healthier individuals have higher returns to labor input; incentive effect says that healthier individuals with longer LE have incentive to invest in education as time of returns is extended; education drives economic growth and health has an indirect role. Results show that indirect effect of health is positive and significant.</td>
</tr>
</tbody>
</table>
### 2.2 INCOME INEQUALITY’S IMPACT ON ECONOMIC GROWTH

Rising income inequality ($INEQ$) is a concern today. In advanced economies, the gap between the rich and poor is at its highest level in decades (Stiglitz 2013). Inequality trends have been more mixed in emerging markets and developing countries with some countries experiencing declining inequality, but pervasive inequities exist with reference to access to education, health care, and health financing. Countries with higher levels of $INEQ$ tend to have lower levels of mobility between generations with parent’s earnings being a more important determinant of children’s earnings (Corak 2013). Inequality goes hand in hand with economic, financial, and political instability. Extreme inequality may damage trust and social cohesion causing conflicts. It can lead to a backlash against growth-enhancing economic liberalization and fuel protectionist pressures against globalization and market-oriented reforms (Claessens and Perotti 2007). Empirical research has shown that at present income gains rapidly decrease after the 50th percentile and become stagnant around the 80th–90th global percentiles before shooting up for the global top 1% (Krugman 2014).

Although trade has been an engine for growth in many countries by promoting competitiveness and enhancing efficiency, high volumes of trade and financial flows between countries partly enabled by technological advances have driven $INEQ$ (Dabla-Norris et al. 2015). In advanced economies, the ability of firms to adopt labor saving technologies and undertaking offshoring have been cited as an important driver of the decline in manufacturing and rising skill premium (Feenstra and Hanson 2003). Also decline in trade union membership has reduced the relative bargaining power of labor exacerbating wage inequality (Frederiksen and Poulser 2010).

An increase in $INEQ$ can have both growth-promoting and growth-dampening effects. In highly developed economies studies indicate that increasing $INEQ$ has reached a level that is becoming a brake on growth. For this reason, there is no fundamental contradiction between state-led income redistribution and economic growth. This picture is compatible with the relationship of $GDPc$ as a function of the society’s $INEQ$ measured by the income $GINI$ coefficient. The $GINI$ coefficient possesses four basic qualities of a good $INEQ$ measure: anonymity, scale independence, population independence, and transfer principle (Cowell 2013). With an increase in $INEQ$, growth-promoting incentives predominate and $GDPc$ increases. However, if income is unequally distributed, people have no great incentive to work. In this case,
an increase in GDPc can be expected from a reduction in INEQ. So, the relationship between economic performance measured on the basis of real GDPc and the degree of INEQ assumes an inverted U-trajectory in INEQ–GDPc space. There is no clear empirical evidence regarding the question of when INEQ has shifting growth effects. An increasing level of INEQ dampens future economic growth, weakening both the supply, i.e. human capital and real capital, and the demand. The question as to when this weakening, particularly the lack of demand for goods, leads to stagnation, depends largely on the economy’s GDPc level (Voitchovsky 2009, Petersen and Schoof 2015). Note that supply side effects from wealth inequalities can be substantial for growth when capital markets are imperfect and heterogeneous agents are loan constrained (Aghion et al. 1999).

Halter et al. (2014) investigated the effect of inequality on economic growth for different time horizons. Their results showed that the effect of inequality on economic growth was positive in the short-run (i.e. following five years). However, on the contrary, in the medium to long-run, the effect became negative. Kolev and Niehues (2016) found evidence for a non-linear relationship between inequality and growth when considering a sample of developed and developing economies. Thus, the effect of net INEQ on growth seemed to be negative only for less developed countries and for countries with high levels of inequality and non-significant or even positive otherwise. The negative effect diminished and became positive for high income levels as well as for low levels of initial inequality. Dabla-Norris et al. (2015) stressed the need to focus on the poor and the middle class as income distribution itself mattered for growth also. Thus, if the income shares of the top 20 percent (the rich) increased, the GDP growth declined over the medium term, suggesting that the benefits did not trickle down. In contrast, an increase in the income share of the bottom 20 percent (the poor) was associated with higher GDP growth. Technological progress, e.g., a resulting rise in the skill premium, and the decline of some labor market institutions had contributed to inequality in both advanced economies and emerging markets and developing countries. De Gregorio and Lee (2004) argued that, in addition to direct effects, INEQ affected economic growth indirectly by influencing other determinants of growth. In particular, they found that more inequality tended to raise fertility, lower secondary school enrollment, and the rule of law. Through these channels, greater income inequality lowered economic growth by more than the direct effect.

Income redistribution policies imply negative effects on economic growth, by reducing performance incentives for taxpayers (which affects labor and capital supply), and through the welfare and growth losses associated with tax collection. It is therefore important to see that the negative growth effects of redistribution are not larger than the positive growth effects of the income redistribution (Berg and Ostry 2011a, b). Redistribution has played an important role in reducing INEQ in advanced economies, but the largest driver has been the increasing share of middle skilled occupations relative to low- and high-skilled occupations (Goos et al. 2009). In emerging countries, the middle-class squeeze in some countries reflects income polarization (Duclos et al. 2004, Zhang and Kanbur 2011). New information technology has not only led to improvements in productivity, but it has also played a central role in driving up the skill premium, resulting in increased labor INEQ.

Income and wealth distribution can be systematically, albeit in non-linear fashion, affected by the level of economic development. Kanbur and Summer (2012) gave a lucid review of the “Kuznets school” (see also Piketty 2014, Kanbur 2000, Deininger and Squire 1998). At the low level of GDPc, income and wealth distributions are wide,
but they narrow down when the economy reaches higher level of GDPc. The modern version of this hypothesis says that if the income or wealth distribution is unequal, the rate of economic growth is low. However, this version abstracts from the fact that the relationship suggested by Kuznets is path dependent. Kuznets (1955, 1966) used pre-World War II time series data for US, UK and Germany and argued that the level of development from agricultural to industrial society was the starting point. That is, the level of GDPc determined when the inequality-growth relationship was positive and when negative. Typically, at the low level of GDPc one observed positive relationship between inequality and growth, and the negative prevailed with higher levels of GDPc.

In addition to Kuznets’ hypotheses the human capital accumulation theory motivated non-linear inequality effects on economic growth. The access to a certain minimum level of education was limited for the population in less developed economies and depended on economic conditions, e.g. inequality. In developed countries, on the contrary, primary and even secondary education was mostly affordable also for the lower income classes. Therefore, the effect of inequality on economic growth was negative in less developed countries, decreasing in absolute terms with the level of development and becoming positive in high-income nations. Thus, it was possible to have a nonlinear relationship between inequality and economic growth depending both on the level of GDPc and inequality. The following Table 2 sums-up the main results in literature:

<table>
<thead>
<tr>
<th>GDPc level / growth – INEQ relationship</th>
<th>Authors</th>
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Voitchovsky (2009) gives a review on the relevant GDPc–INEQ literature most recent time. She concludes that many studies have neglected the fact that income or wealth inequality means different things to persons in different positions within the distributions. Thus, single estimates, e.g. on GINI coefficient in linear model for growth, do not tell much of theory or model implications that are based on behavior of people in different positions in income or in wealth distributions. The empirical results depend on the data and the method used. A negative effect of inequality is usually obtained from cross-sectional data with OLS estimation for growth rates (for similar results for 1980’s and 1990’s, see Benabou 1996 and Perotti 1996). Short growth spells with panel data methods like FE and the first difference GMM tend to report positive effects of inequality on growth. However, the selected sample (i.e. poor or
rich countries) matters much as inequality varies greatly between different samples of countries, and it is expected that in poor economies – or amongst the poor – inequality with all its dimensions has more adverse effect on growth than among more affluent and equal economies. Thus, different levels of inequality may be conducive to growth at different levels of development (Voitchovsky 2009).

Dominica et al. (2008) and Neves et al. (2016) conducted a meta-analytic reassessment of the effects of inequality on growth. The former pointed out that the magnitude of the estimated effect of inequality on growth in the literature depended crucially on the estimation method, data quality, and sample coverage. Studies using panel fixed effects estimators seemed to report stronger effect of inequality on economic growth than cross sectional results. Neves et al. (2016) extended the meta-analytic re-assessment to more recent studies and showed that the empirical literature on the inequality-growth nexus was biased towards statistically significant results. As the authors stressed, this made the empirical effect of inequality on economic growth larger in absolute terms than what it actually was. They also showed that the direction of effects followed a certain time pattern: in the 1990s, most of the published studies found negative effects, while at the beginning of this century this tendency got reversed and empirical studies increasingly documented positive results. Thus, the vast literature on inequality effects on growth points still to no general well-determined results.

2.3 IMPACT OF INCOME INEQUALITY ON HEALTH

Pickett and Wilkinson (2015) conducted a literature review within an epidemiological framework and inferred the likelihood of a causal relationship between income inequality and health by considering the evidence holistically. The body of evidence strongly suggested that income inequality affected population health. The evidence that large income differences had damaging health and social consequences was strong. Generally low social status and the quality of the social environment were both known to affect health (Berkman and Kawachi 2000, Marmot and Wilkinson 1999). The recent reviews of the literature on inequality and health had ranged in tone from critical (Deaton 2003) through skeptical (Lynch et al. 2004a, b) to enthusiastic (Wilkinson and Pickett 2006).

The policy debate in less developed countries (LDC) holds three main positions: pro-market liberalizers, the psycho-social school, and the pro-poor position. Pro-market liberalizers argue that raising average incomes through economic liberalization is the most effective way to improve public health. The seminal works by Preston (1975) and Pritchett and Summers (1996) show that the relationship between average income and health is curvilinear and concave, and that the causal direction is from wealth to health. The argument is based on reducing material deprivation: higher average incomes allow public investment in health infrastructure at the societal-level and sufficient expenditure on diet and medicine at the individual-level to protect health (Anand and Ravallion 1993, Dollar and Kraay 2002). The psycho-social school accepts these materialist pathways and the important role of average income levels, but also introduces non-materialist factors and income inequality. For individuals with relatively low incomes, inequality generates stress that damages health directly and indirectly by behaviors associated with stress like smoking and alcohol abuse (Rajan et al. 2013). Socially, these feelings manifest as reduced civic participation and anti-social behavior affecting the health of others, including those higher up the
income range (Lynch et al. 2000, Marmot 2002, Murali and Oyebode 2004, Wilkinson 1997). This view is closely related to the “social capital” paradigm in which inequality reduces “civic engagement” and “levels of mutual trust” (Kawachi and Kennedy 1999, Kawachi et al. 1997). Frayed social bonds manifest in poorer public health.

Subjective measures of well-being such as “life satisfaction” and self-reported health have received increasing attention following work by Stiglitz et al. (2008) advocating more holistic measures of development, including public health. Variables like average income and poverty may be the chief determinants of objective measures of public health like IM, but even in developing countries, inequality, among other factors, undermines such life satisfaction and self-reported health, and thereby undercuts the gains made by increasing income levels (Brockmann et al. 2009, Easterlin 2003, Easterlin et al. 2012, Knight and Gunatilaka 2011).

In the pro-poor position predictive power of literacy has been well-established in both developed and developing countries. Literacy mediates the investment in the infrastructure pathway by enabling a population to engage with the healthcare infrastructure available and respond to public health campaigns. In poor countries female illiteracy is associated with child mortality (Caldwell 1986, Sen 2002). At the individual level, it is associated with better personal protection of health, including healthier behaviors such as not smoking and improved diets (Kabir 2008). Table 3 sums-up some most relevant studies:

Table 3. Studies of impact of inequality on health status

<table>
<thead>
<tr>
<th>Study</th>
<th>Some important components</th>
<th>Relevant results</th>
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<tbody>
<tr>
<td>Asafu-Adjaye (2004)</td>
<td>Panel data for 44 countries covering six time-periods.</td>
<td>INEQ (measured by GINI coefficient) has significant effect on HS when levels of income, savings and education are controlled; relationship consistent, regardless of HS and income specification; empirically support income inequality hypothesis (IIH).</td>
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<td>Avendano (2012)</td>
<td>34 OECD countries; 1960–2008 using panel FE models.</td>
<td>Finds IM and INEQ in cross sectional (CS) setting a positive correlation, but in panel model with FE, results disappear.</td>
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<td>Babones (2008)</td>
<td>Focuses on ecological correlation between INEQ and indicators of population health in broad panel of countries using CS data to demonstrate that relationships are largely non-artifact; tests whether relationships might be causal.</td>
<td>Finds correlation and temporal precedence of INEQ over health; unambiguous correlation between INEQ and population health at country level; in CS analyses, INEQ is significantly correlated with LE, IM and (inconsistently) with the murder rate; correlations not primarily due to the non-linear relation between individual income and individual health; change in INEQ in 1970–1995 significantly related to change in LE and IM, suggesting causal relationship; strong, consistent, statistically significant, and non-artifact correlation between national INEQ and population health; relative stability of INEQ over time in countries makes causality difficult to test.</td>
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<tr>
<td>Biggs et al. (2010)</td>
<td>Data from 22 Latin American countries 1960–2007.</td>
<td>Provide evidence that aggregate HS is dependent on GDPc level, but positive correlations are affected by the poverty and INEQ levels; during time of decreasing or constant poverty, the positive GDPc effects are strongest; INEQ and poverty found to exert independent, substantial positive effects between national income level and health.</td>
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<tr>
<td>Study</td>
<td>Some important components</td>
<td>Relevant results</td>
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<tr>
<td>Christiansen et al. (2017)</td>
<td>Compare Nordic countries to UK and Germany with data from European Social Survey and OECD Statistics 2002 and 2012.</td>
<td>Health measured by self-assessed health in five categories and transformed to a cardinal scale using Swedish time trade off weights; results show income related inequalities in health in Nordic countries are similar or lower than in UK and Germany.</td>
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<td>Clarkwest (2008)</td>
<td>State-level U.S. data 1970–2000 used to model effects of initial levels and changes in INEQ on 10-year LE changes; neo-materialist perspective with theoretical predictions of temporal ties between INEQ and change in HS focusing on adoption of longevity enhancing innovations.</td>
<td>States with higher levels of INEQ experience less subsequent improvement in LE; strong negative association between change in INEQ and change in longevity once initial levels of INEQ and other state characteristics are controlled; relation between INEQ and adoption of innovations in quality of medical care indicates that they are highly related and that differences in the average quality of care can account for the negative association between INEQ and LE.</td>
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<tr>
<td>Deaton (2003)</td>
<td>Analyzes theoretical basis and empirical evidence for a connection between INEQ and health; review cross-country studies on adult mortality for rich countries and child mortality for poor ones.</td>
<td>Strong evidence that before &quot;epidemiological transition&quot; INEQ determines mortality; i.e. in poor countries, INEQ means poor sanitation, unhealthy working and living environments, poor nutrition, and a plethora of infectious diseases, while in rich nations no robust correlation between LE and INEQ when one controls variables e.g., education and indicators of social capital.</td>
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<td>Herzer &amp; Nunnenkamp (2011)</td>
<td>Address INEQ and HS issues in context of panel data co-integration and Granger causality test for 35 countries 1970–1995; argue that studies on relationship between INEQ and health suffered from biases due to omitted country specific factors, endogeneity and heterogeneity.</td>
<td>INEQ has, on average, a small, but robust and statistically significant positive impact on HS; there is evidence that inequality is endogenous in the sense that poor health leads to increased INEQ; there are large cross-country differences in the effect of INEQ on health (in 35 percent of the cases effect is negative).</td>
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<td>Hildebrand &amp; Van Kerm (2009)</td>
<td>Pooled sample of 11 EU nations, using longitudinal and cross-national data from European Community Household Panel 1994–2001; self-reported health information.</td>
<td>Find consistent evidence that INEQ is negatively related to self-rated HS in EU for both gender particularly when measured at national levels; magnitude of the impact of INEQ on health is small; when controlling for potential reporting bias statistically significant effects of INEQ for both gender in the pooled sample.</td>
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<tr>
<td>Karlsson et al. (2010)</td>
<td>CS dataset from 2006 on 16000 individuals aged 40–79 in 21 nations; examine whether individual and relative income in a reference group and INEQ are related to HS.</td>
<td>Strong support for relative income hypothesis (RIH): average income within a peer-age group is negatively related to health; income inequality hypothesis (IIH) in high-income countries; finds evidence of heterogeneity, depending on the level of income development and how INEQ affected health in different countries.</td>
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<td>Leigh &amp; Jencks (2007)</td>
<td>Data on top income shares from Australia, Canada, France, Ireland, the Netherlands, New Zealand, Switzerland, UK, and US for 1905–2002; investigated relation between INEQ, LE and IM.</td>
<td>Finds in absence of country and year fixed effects (FE), positive relationship between inequality and mortality; in preferred FE specification, relation became small and statistically insignificant; results were less favorable for long-run negative INEQ effects on LE for 12 countries; RIH effects were present but IIH effects were doubtful in rich nations.</td>
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<tr>
<td>Study</td>
<td>Some important components</td>
<td>Relevant results</td>
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<td>Lorgelly &amp; Lindley (2007)</td>
<td>Estimate relationship between INEQ and HS using different countries’ panel data.</td>
<td>Found support for absolute income hypothesis on health (AIH), no support for IIH or RIH; limited evidence of an HS effect of INEQ; no gender differences.</td>
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<tr>
<td>Lynch et al. (2004a)</td>
<td>Review of 98 aggregate and multilevel public health studies examining the associations between INEQ and HS in years 1980–2000.</td>
<td>Find for OECD countries that aggregate and multilevel evidence suggest little or no effect of INEQ on HS indicators; overall, less support for a strong psychosocial version of the IIH and empirical support for a weaker version of IIH, i.e. in some contexts, INEQ contribute to HS; note that negative findings in no way contradict evidence that at the individual level people with higher incomes are healthier.</td>
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<tr>
<td>Rajan et al. (2013)</td>
<td>Using state-, district- and individual-level data from India for varying years investigate relationship between IM, under-five mortality, average income, poverty, INEQ and literacy; tests the inverse relation between economic status and ill health and the hypothesis that INEQ becomes a significant predictor of public health only after “epidemiological transition”</td>
<td>Low poverty and high literacy rather than wealth per se improve public health; IM negatively associated with average income levels and positively associated with poverty at both state and district-levels; INEQ not a predictor of infant or under-five mortality rates at state- or district-levels, but has strong effect on self-reported health at the individual-level; policies alleviating poverty are more effective than raising average income levels; non-income goods like literacy make important contributions to public health; policies need to be based on a broader understanding of societal well-being and the factors that promote it.</td>
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<td>Ram (2006)</td>
<td>Cross section data of 108 countries with variables pertaining to 2000 or late 1990s; lag of about 5 years allowed between health indicators: regressors econometrically pre-determined.</td>
<td>INEQ significant even after index of ethnic heterogeneity included, which itself has a negative association with population health; INEQ retains significance in presence of a measure of social capital that has weak link with HS; income level relatively more important for health in less developed countries and role of INEQ is stronger in developed economies.</td>
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<tr>
<td>Wagstaff &amp; van Doorslaer (2000)</td>
<td>Review of literature on negative effects of INEQ on health; studies various hypotheses (e.g. AIH, IIH, RIH, etc.) described explaining empirically observed association between INEQ and HS measures; question the relevance population and community-based studies.</td>
<td>Support strong evidence for AIH, some evidence for IIH, and nothing to support RIH; individual-level studies have potential to contrast between hypotheses; some evidence consistent with IIH at state level: individual’s health is a decreasing function of INEQ in his or her area, but that the strength of the effect depends on how well one controls for other influences on health, especially the individual’s income; INEQ does not capture hypothesized effects of social capital or psycho-social factors but rather effects of state-level policies towards poor that are correlated with INEQ.</td>
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<td>Zheng (2012)</td>
<td>U.S. National Health Interview Survey data (1986–2004) with mortality follow-up data (1986–2006); studies lagged effects of national level INEQ on individual mortality risk.</td>
<td>INEQ does not have an instantaneous detrimental effect on individual mortality risk but starts to exert its influence 5 years later; effect peaks at 7 years, and then diminishes after 12 years; this pattern holds for three measures of INEQ: GINI coefficient, Atkinson index, and Theil entropy index.</td>
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2.4 HEALTH EXPENDITURES AND HEALTH OUTCOMES

Research has shown that among high income OECD countries high health expenditure (HE) do not necessarily lead to better average health when measured by health adjusted LE (OECD 2014, Potrafke 2010). The public fraction of HE is highest in rich countries, which also have the highest total health expenditures. Private financing dominates in low-income countries, where direct out of pocket payments are more important than private insurance (Gerdtham and Johnsson 2000). During most of the second half and especially the last decades of the 20th century, public health expenditures (HEPUB) have been growing at a faster rate than national income (Maisonneuve and Martins 2006). According to Clements et al. (2012), HEPUB in advanced countries have been characterized by short periods of accelerated growth followed by periods of cost containment (see also, Docteur and Oxley 2003, Potrafke 2010). Cost containment policies have been implemented mainly through macroeconomic mechanisms, such as wage moderation, price controls, postponement of investments, and privatization.

Private health expenditures (HEPRIV) are overwhelmingly out of pocket payments. Private insurance tends to be a luxury good in high income countries or among high income households in low income countries. The importance of private insurance in total health spending depends significantly on the finance structure of health care system. In some countries private insurance is viewed as an integral part of the health system, subject to regulation. In other countries, private insurance is viewed as a complementary good and either tolerated or encouraged. However, in most countries, private insurance is simply one more segment of a fragmented health care finance system. The importance of private insurance and expenditures depends on the country specific factors, distribution of income as well as on public policy.

International comparisons of HE are fraught with many problems. The first one is the weak theoretical base for the determinants of aggregate HE, which provides little guidance as to the possible explanatory variables and the causal mechanisms involved (Gerdtham and Johnsson 2000). Here Parkin et al. (1987) stress the “atheoretical basis” of macroeconomic analysis of HE. The second problem is that rigorous assessment of the quality (i.e. accuracy and reliability) of the cross-national data is difficult due to differential classifications. The third problem of international comparisons is often the small available sample size for certain countries which causes restrictions on model size and inference. The fourth issue is that when doing cross-country comparisons such comparisons implicitly imposes the assumption of homogeneous relationships across countries which may appear unrealistic for many reasons, e.g. heterogeneous preferences, production functions etc. A fifth problem is that country comparisons are static, while the observed differences in HE and income are the result of both real and transitory differences when countries are in different stages of different growth adjustment processes (Gerdtham and Johnsson 2000).

A priori, it is unclear whether HE are normal or luxury goods, i.e. are the income elasticities of health demand lower or higher than one (Medeiros and Schwierz 2013). Maisonneuve and Martins (2006) suggest that high income elasticities (above one) often found in macro studies may result from the failure to control for price and quality effects in econometric analysis. Hall and Jones (2007) showed that one could generate the growth in health spending and the resulting change in LE solely as a result of growth in income using a model of endogenous health spending. Instead of modeling other changes simultaneously they showed that with reasonable parameter estimates and implied value of longevity gains, one could generate rise in HE and
The result was based on their theory that income elasticity for health is larger than one. Contrary to this, Acemoglu et al. (2013) showed that the elasticity is much lower than one. Research has shown that income increases are unlikely to be the primary drivers of the increase in the \( HE \) share of \( GDP \) (Gerdtham and Johnsson 2000). Rising incomes are also unlikely to be the major driver of medical innovation. An interesting possibility is that institutional factors such as the spread and level of insurance coverage have not only directly encouraged spending but also have induced the adoption and diffusion of new medical technologies (Acemoglu and Finkelstein 2008).

At the country level, Akinkugbe and Mohanoe (2009) performed time series analysis using the error correction model and found that in addition to \( HE_{PUB} \), the availability of physicians, female literacy, and child immunization significantly influenced \( HS \) in a developing country like Lesotho. At the regional level, Anyanwu and Erhijakpor (2007) with a panel data analysis and \( FE \) model, found that total \( HE \) were a significant contributor to health outcomes. Filmer and Pritchett (1997) had previously provided evidence that while \( HE \) impacted on \( IM \), it was not the dominant driver of this. Factors such as education, income, technological change, and cultural differences were identified by some researchers as major drivers of \( HS \) rather than \( HE \) (Filmer and Pritchett 1999). While some studies have shown significant positive or negative impact of \( HE \) on \( HS \), others have not found any significant relationship between the two (e.g. Burnside and Dollar 1998).

Musgrove’s (1996) summary on the few studies on determinants of child mortality suggested that income was always significant, but the public share in \( HE \), and the share of public spending on health in \( GDP \) were not so. Contrary to these results some studies found a significant impact of \( HE_{PUB} \) on \( HS \). Anand and Ravallion (1993) suggested the impact of \( HE_{PUB} \) on health was likely to vary widely from country to country and hence the results were sensitive to the sample used. One of the purposes of the study by Novignon et al. (2012a, b) was to investigate the impact of total \( HE \) on various \( HS \) after controlling for country-specific demographic structures and economic conditions. A differential analysis of public and private \( HE \) was performed. The results showed that both private and public sources of \( HE \) were significantly associated with improved \( HS \). The findings implied that \( HE \) were essential components in improving \( HS \) in impoverished countries. There was need for governments in the regions to increase amounts allocated to health care service delivery. In addition, establishing effective public-private partnerships in developing the health sector were to go a long way to improve population \( HS \).

Although one focuses on the economic implications of population health, there is clearly two-way causality as health depends on income and development level (Bloom and Canning 2008). Fogel (2004) also emphasizes the role of access to food while Deaton (2006) puts more weight on public health measures such as clean water and sanitation. Cutler and McClellan (2001) examine the increasing contribution of medical care to \( HS \). Easterly (1999) argues that, although income levels and population health are closely related, the effect of changes in income on population \( HS \) over a reasonable time span appears to be quite weak. By contrast, relatively inexpensive public health interventions and policies can have remarkable impacts on population health, even in very poor countries. The major force behind specific health improvements has been improvements in \( HCT \) and public \( HS \) measures that prevent the spread of infectious diseases (Cutler et al. 2006).
2.5 HEALTH CARE TECHNOLOGIES

Technological innovations have yielded remarkable advances in health care. Breakthroughs in a variety of areas have improved health care delivery and patient outcomes. The proliferation of HCT and its expanding uses have contributed to increasing health care costs and the former has often been cited as a culprit for the latter. However, this relationship is variable, complex, and evolving. Creation and development of new technologies are driven by both demand and supply side forces. Innovation in relation to medical devices has been defined in several ways, each focusing on specific traits of this multi-dimensional concept. Current definitions adopted are focused on therapeutic added value, including clinically significant benefits, large health gains, and favorable risk-benefit balance at an acceptable cost. Following innovation management and economics theory, innovation can be e.g. classified according to three broad dimensions: (i) the source of innovation, (ii) the degree of discontinuity introduced, and (iii) the impact or consequences of innovation (Ciani et al. 2016).

There are many benefits of HCT innovations (Cutler and McClellan 2001). The most important is the value of better health, i.e. a longer life as well as improved quality of life after early or proper diagnosis of diseases. A second benefit of HCT innovation is its effect on income formation. One part of this benefit is an increase in productivity that results from HCT allowing people to work and earn more. Offsetting this productivity benefit are the medical and nonmedical costs of additional years of life. The net value of medical technology change is the difference between the benefits and costs. A positive net value implies that technological change is worth it in sum.

Cutler and McClellan (2001) reported on a series of studies that examined the costs and benefits of medical technology changes. These studies showed that medical spending was worth the increased cost of care. HCT change affects treatments in two ways, namely, treatment substitution and expansion. If the new HCT supplements the existing instrumentation and its purpose is to expand the treatment into the conditions that have not been treated previously due to scientific or economic reasons, one can say that it could have a cost increasing effect (expansion effect). On the other hand, extra savings may be expected if a decrease in the relative price of a given type of new treatment reduces the use of the other, more expensive types of care (substitution effect). Sorenson et al. (2013) reviewed selected 86 articles and analyzed them for key cross-cutting themes, e.g. impact of technology on costs, factors influencing this relationship, methodological challenges in measuring such linkages etc. They argued that attention is needed to be focused on exploring whether investments in medical technology resulted in better values as measured by therapeutic benefits, cost effectiveness, and other important health outcomes. In addition, they asked under which conditions HCT causes the most effective and efficient use of available health care resources.

Past literature reviews imply that HCT diffusion is an important contributor to improved HS as measured by LE and IM. Preston (1996) and Soares (2005) argued that increases in LE have occurred independently of increases in per capita income. Preston suggested also that mortality changes in developing countries came about through the provision of public programs and the dissemination of health knowledge. While not denying the importance of other factors, Soares (2005) placed emphasis on changes in mortality determined from technological innovations in medical and biological sciences. Both authors gave evidence that the positive relationship between
LE and GDPc had shifted upwards steadily over time. Kremer (2002) emphasized the importance of modern HCT in allowing tremendous improvements in health at low income levels. Jamison et al. (2001) documented the importance of different rates of technological progress across countries for the declining cross-country variation in IM. Becker et al. (2005) argued that in the last 50 years, countries starting with modest longevity levels experienced LE gains significantly larger than countries starting with high longevity thresholds. They attributed the convergence in LE in large part to the global diffusion of existing knowledge that had helped reduce mortality from major diseases.

International medical diffusion occurs through two distinct channels. Firstly, imports of medical goods, such as drugs, vaccines, and medical equipment. Papageorgiou et al. (2007), Caselli and Wilson (2004), and Eaton and Kortum (2001) consider that production of goods embodying HCT is concentrated in a small number of R&D intensive countries while the rest of the world typically imports these goods. Secondly, there are direct flows of medical knowledge from a few frontier countries to the rest of the world, i.e. flows that are facilitated by information networks created by medical persons, drug firms, and students at international levels (Papageorgiou et al. 2007).

Baltagi et al. (2012) modeled differences across OECD countries in health productivity as a function of traditional factor inputs, life styles conditions, and technological progress. The authors first explored available data on HCT to explain health productivity in the OECD countries. Their results showed that spatial spill-overs in LE were significant. They pointed to the existence of interdependence across countries in technology adoption.

Skinner and Staiger (2016) developed a general model in which hospitals and physicians sought to maximize the health of their patients by adopting new technologies, including diffused ones, in the face of financial and knowledge-based barriers. Variations across hospitals in these barriers lead to differences in the diffusion rate of new technologies. The authors tested whether hospitals adopting effective treatments also adopted other, less cost-effective technologies. Like Eaton and Kortum’s (1999) study with aggregate productivity, they found substantial differences in the extent to which some hospitals lagged behind in the diffusion of highly effective technologies.

Although technological progress had led e.g. to dramatic improvements in survival for heart attack patients (Cutler 2004), these improvements are largely associated with the adoption of effective new technologies rather than more factor inputs per se (Chandra and Skinner 2012). Informational or organizational barriers explain the slow diffusion in both health and non-health sectors of the economy. Diffusion is influenced by organizational support and tactile learning from peers (Keller 2004). Parente and Prescott (2002) provide a ready explanation for why some countries lag so far behind “frontier” countries in technology adoption. Government restrictions and monopoly restraints interfere with the benefits of efficient technology adoption. If patients both know about the benefits of HCT and are sensitive to published and reliable information about hospital and care quality, physicians would be forced to respond rapidly to new innovations or face the loss of patients. But when quality measures are limited, patients are not well informed, and markets are distorted, large inefficiencies remain.

The global diffusion of medical innovations is closely related to the concepts of growth convergence found in the modern economic growth theory. In $\sigma$–convergence cross-section standard deviation (SD), the average variability around the mean,
diminishes during the sample years. In \textit{\(\beta\)-convergence} or regression to the mean (Baumol 1986, Barro and Sala-i-Martin 1992a, Sala-i-Martin 1994, Mankiw et al. 1992, Boyle and McCarthy 1997) convergence exists if a poor economy tends to grow at a faster rate than a rich one. Within this method two forms of convergence are distinguished: \textit{unconditional or absolute} and \textit{conditional} rates of growth. In the long run one would expect convergence of \textit{HE} and \textit{HS} indices across the countries due to the existence of their upper bounds and diminishing health returns of health capital inputs.

The convergence of \textit{HE} in developed countries has been examined by Hitiris (1997), Barros (1998), Nixon (2000a), Hitiris and Nixon (2001), Narayan (2007), Panopoulou and Pantelidis (2012), Lau et al. (2014), Pekkurnaz (2015), and Nghiem and Connelly (2017). The findings highlight the importance the non-linearity and dynamics in \textit{HE}. There is evidence of convergence among sub-groups of countries and specific \textit{HE}.

Clark (2011) offered new evidence on inequality trends in \textit{HS} indices and economic development. He examined whether \textit{LE} averages and \textit{IM} converged across 195 countries during the 1955–2005 period. Consistent with prior work, he found that cross-national inequality in \textit{LE} declined during the 1955–2005 period, but this convergence stalled during the post-1990 era. Moreover, contrary to previous work, he found that \textit{IM} diverged continuously across the sample period. In sum, Clark argued that economic development had contributed to both convergence in \textit{LE} and divergence in \textit{IM}.

Moser et al. (2005) investigated to what extent worldwide improvements in mortality over the past 50 years have been accompanied by convergence in the mortality. Their analysis used UN data for 1950–2000 for 152 countries with populations of at least 1 million in 2000. They concluded that the shift from global convergence to divergence was being driven by reversals in adult mortality. Goli and Arokiasamy (2014) tested the convergence hypothesis for trends in maternal and child mortality indicators during 1990 to 2008 for 187 countries by using three different types of convergence metrics. They found some discrepancies in the progress achieved in child mortality and in maternal health. Graphical assessment indicated clear evidence of catch-up process for all the maternal- and \textit{IM} indicators, but surprisingly the \(\beta\)-convergence model estimates showed lack of convergence. The results of the \textit{absolute \(\beta\)-convergence} analysis suggested a divergence in the progress of the maternal mortality ratio across the countries for the entire period and convergence towards the end of the sample period.
3  SUMMARIES OF PAPERS

3.1  PAPER 1


The dependency of health on income is almost universal in health economics. The main hypothesis is that the absolute income hypothesis (AIH) stating a person’s health level is a concave function of his or her income level. Interestingly, quite often, this simple micro relation is also estimated with macro data as access to individual level data is not possible. However, this type of regression leads to different parameter values compared to micro data results. This is especially true when non-linear micro relations are estimated with aggregate data, such as mean values. Besides the involved aggregation problem when estimating the concave income function on HS with aggregate data, it surprisingly produces also a spurious negative correlation between average HS and spread of incomes. So, AIH is not a robust hypothesis at the aggregate level. This is because the aggregation is distribution free only when the relationship between health and income is linear. Thus, regressing average health on average income non-linearly and on some income distribution measure produces results that are artificial since all higher moments, not only mean income, exists for any non-linear function of the (random) income variable. This transfer or “concavity-induced income inequality” effect (Deaton and Muellbauer 1980, Stoker 1993) is also called a statistical artifact of aggregation (Gravelle 1998). The finding that there is a negative partial correlation between average health and a measure of spread of income may therefore not be considered as evidence that individual health is adversely affected by INEQ. It is simply the result of the curvilinear relationship between income and the health level which is operating at the individual level (Gravelle et al. 2002 p. 579, Lynch and Smith 2002 p. 61).

In this paper, after analyzing this aggregation problem in detail, we propose an alternative AIH model at macro level that reduces the aggregation biases when we use HS as a log function of income. This enables us to add elements of income inequality hypothesis (IIH) also to the model because we are able to control for aggregation artifacts in the model. In details, if we estimate the following model for some index of population health:

\[ HS_k = \alpha + \beta \ln \bar{Y}_k + d'X_k + \varepsilon_k, \]

where \( \bar{Y}_k = \frac{1}{n} \sum_{i=1}^{n} Y_{ik} \) is at aggregation level \( k \), then the OLS-estimate for \( \beta \) is biased, since the consistent aggregation is based on typically not known geometric mean of incomes \( \ln \bar{Y}^o_k = \frac{1}{n} \sum_{i=1}^{n} \ln Y_{ik} \) instead of the arithmetic mean \( \bar{Y}_k \). To overcome this problem, the following approximation method is used. Jensen’s inequality implies for concave function like ln-function in the context of the aggregate mean income the following:
The Taylor approximation around $Y$ for $\ln(Y - \theta)$ gives $\ln(Y - \theta) \approx \ln Y - \frac{\theta}{Y}$.

Applying this first-order result to our $k$ aggregate level health model above gives us the bias-correcting model:

$$HS_k = \alpha + \beta (\ln Y_k - \frac{\theta}{Y_k}) + d'X_k + \mu_k = \alpha + \beta \ln Y_k - \frac{\beta \theta}{Y_k} + d'X_k + \mu_k.$$ 

In practice, applying this model is not difficult. In order to analyze the size of suggested bias correction for parameter $\beta$ in OLS-estimation, the following three models with 148 countries in the years 1970–2010 ($k = 1,\ldots,148$; $t = 1,\ldots,41$; 6068 observations) are estimated:

**Model A**: $LE_{kt} = \alpha_1 + \beta_1 \ln GDP_{kt} + \gamma_1 / GDP_{kt} + \delta_1 GINI_{kt} + \epsilon_{kt,1}$

**Model B**: $LE_{kt} = \alpha_2 + \beta_2 \ln GDP_{kt} + \delta_2 GINI_{kt} + \epsilon_{kt,2}$

**Model C**: $LE_{kt} = \alpha_3 + \beta_3 \ln GDP_{kt}^* + \delta_3 GINI_{kt} + \epsilon_{kt,3}$

Here $LE$ is at birth in years, $GDP_c$ is at 2005 constant prices, and $INEQ$ is measured by $GINI$ coefficients. $\epsilon_{kt,i}$'s are the error terms of the models. Data is taken mainly from World Development Indicators of the World Bank, IBRD-IDA database of the World Bank, Penn World Tables, Socio-Economic Database for Latin America, World Income Distribution, UNU WIDER, and United Nations data sources.

The first equation (Model A) is based on micro foundations wherein a person’s incomes and the prevailing income distribution in his or her social environment has effects on his/her health. The variable $1 / GDP_{kt}$ is the correction term stemming from the inadequate aggregation of the log of income effect on health at the micro level. Model B is the reference model that includes the aggregation bias, and the coefficient $\beta_2$ is expected to be biased upward. The model lacks the microeconomic interpretation. Finally, Model C corrects for the aggregation bias with the estimation of the errors-in-variables bias, i.e. $\ln GDP_{kt}^*$ in Model C is equal to

$$\ln GDP_{kt}^* = \ln GDP_{kt} - \frac{\hat{\theta}}{GDP_{kt}},$$

with $\hat{\theta}$ coming from OLS regressions $\ln GDP_{kt}$ on $1 / GDP_{kt}$ for each of the sample countries separately with time series observations.

The estimation of three models above was first conducted in panel data setting. We considered the group means model, $FE$ model, $FE$ model with $AR(1)$ errors, and random coefficient model (RCM). To have as many results as possible for the suggested aggregation correction method, we also estimated models A, B, and C for 41 yearly cross-sections. This produced 41 different coefficient estimates for each
year in the sample, proving the broader picture as to how \( GDPc \) and \( GINI \) affect \( LE \) during the sample period of 1970–2010. Because of large discrepancies in \( LE \) and \( GDPc \) levels we estimated the models also with quantile regression approach as it estimated the conditional quantiles functions instead of mean conditional function like OLS-regressions.

Generally, the results with bias correcting model alternatives show that they correct the health income effects in the right direction, i.e. they provide smaller parameter estimates than biased models. The bias-correcting estimates also from quantile regression approach indicate that the poorest countries’ income gradient is still much higher than for the rich countries. However, the median effect of \( \ln GDPc \) across the countries decreases during the last sample decennials. The results for \( INEQ \) measured with \( GINI \) coefficient imply that in the poorest countries \( INEQ \) health effects are still significant but the effects are small or non-significant amongst the rich countries after year 2000. We argue that the proposed bias-correcting method retains the interest in macro health modeling and also offers a new model alternative for other fields.

3.2 PAPER 2


The target of the paper is to answer the question as to whether \( HEPUB \) and \( HE_{PRIV} \) can explain \( HS \) variables like \( LE \) and \( IM \). Our data consist of 195 countries in 21 years starting in 1995 and ending in 2014. In our models \( LE \) is determined by private and public expenditures and by exogenous variables of primary education rate (\( PCR \)) and level of R&D expenditures (\( RDE \)). In the model for \( IM \) we replace \( RDE \) for food supply (\( FS \)). We argue that \( HE’s \) are direct means and resources to achieve better health among the population in a country and not the income level of the country (\( GDPc \)) as such. Thus, in our estimated models, level of education, level of technology, and food supply per capita refer generally (among many other similar variables) to the country’s development level that sustains \( HS \) but are not directly targeted, like \( HE’s \), to improve the nation’s health level.

There are differences in \( HE \) amongst developing and developed nations. Countries and regions vary significantly along the \( HEPUB \) vs. \( HE_{PRIV} \) dimension. Since health care demand and supply are derived type variables depending on many driving factors, \( GDPc \) level is not considered as a direct major predictor of \( HS \) in our empirical analysis. Instead we focus directly on \( HEPUB \) and \( HE_{PRIV} \). We argue that in the short run the effects of policy or consumer choices for levels of \( HEPUB \) and \( HE_{PRIV} \) have more direct impacts on \( HS \) variables than \( GDPc \). Thus, the observed heterogeneity of \( HS \) between nations even at same income levels asks for more detailed relationship between health conditions and specific expenditures targeted to promote health care provisions.

Our main argument and test hypothesis is that, at least for the poor countries, the \( HEPUB \) is more important for the population’s \( HS \) than the \( HE_{PRIV} \). In terms of econometrics this means that we propose following FE dynamic panel data models to determine the levels of \( LE \) and \( IM \) depending on \( HEPUB \) and \( HE_{PRIV} \).
\[ \ln LE_t = \alpha_0 + \alpha_1 \ln LE_{t-1} + \alpha_2 \ln HE_{PRIV,t} + \alpha_3 \ln HE_{PUB,t} + \alpha_4 \ln PCR_t + \alpha_5 \ln RDE_t + \epsilon_{1,t} \]

\[ \ln IM_t = \beta_0 + \beta_1 \ln IM_{t-1} + \beta_2 \ln HE_{PRIV,t} + \beta_3 \ln HE_{PUB,t} + \beta_4 \ln PCR_t + \beta_5 \ln FS_t + \epsilon_{2,t} \]

In the models, one period lagged health variables \( LE \) and \( IM \) reflect the dynamics of \( HS \), i.e. past \( HS \) determines the current one. In general terms the models capture more directly the income driven health part of the simultaneous income-health relationship (Weil 2009, Chapter 6), i.e. \( HE \)'s and other indicators of the living standard determine the \( HS \) of the country’s population.

The econometrical problems of dynamic panel data models are well-known, stemming from the fact that lagged dependent variables are not exogenous as they correlate with the unit-specific effects (\( \alpha_i \) and \( \beta_i \)) and with model errors. This causes short panel bias to \( FE \) and \( RE \) estimators and efficiency problem to \( IV \)-approach for the first difference model when solving the short panel bias. \( IV/GMM \) type estimators leading to consistent and more efficient \( GMM \) estimators, as suggested by Arellano and Bond (1991), Ahn and Schmidt (1995) and Blundell and Bond (1998), have been popular in recent times. Empirical drawbacks of complex \( IV/GMM \) agenda, i.e. the problems of close to unit root dynamics with not so short panels and weak or too many instruments, have led to quite large group of alternative estimators which have tried to correct for the \( 1/T \) time series bias in several different ways. In response to this we use estimation methods that are planned to be enough robust against near unit-root case and are allowed to avoid strict exogeneity assumptions. We use the long difference \( IV \) method, \( LD_{IV} \) proposed by Hahn, Hausman and Kuersteiner (2007), and the \( KR \) estimator (\( KR_{PRE} \)) by Keane and Runkle (1992) that allows for pre-determined variables to be used as instruments.

The annual data for model estimations are collected from different sources (e.g. World Bank 2015a-f, Gapminder 2015, WHO 2015a,b). The data consists of observations from 195 countries in the years 1995–2014. To analyse effectively the \( HE_{PUB} \) and \( HE_{PRIV} \) effects on \( LE \) and \( IM \), the \( K \)-means cluster method is used, which identifies clusters of countries with average country specific growth rates of \( LE \) and \( IM \) in sample period 1995 – 2014. We also divide the countries into two groups based on their average level of gross national income per capita (\( GNIc \)) during the sample period: group 1 with \( GNIc \) level below 2 440 US$ during the sample years (77 countries and 39.5 % of sample countries), and group 2 countries with \( GNIc \) level higher than 2 440 US$ (118 countries and 60.5 % of sample nations).

We observe that different methods provide quite varying results on point estimates, but qualitatively clear accordance is found in the signs of coefficient estimates across the estimation methods. Across the different data configurations and model estimations the mean of long run elasticity of \( HE_{PUB} \) coefficient estimates is 0.036 for \( LE \) and –0.023 for \( IM \). Thus, if public expenditures increase a 10 %, it will increase \( LE \) by 0.36 % and reduce \( IM \) by 2.31 %. For \( HE_{PRIV} \), we find 0.23 % and -0.83 % mean elasticities. These estimates imply that globally \( HE_{PUB} \) are more effective to improve \( HS \) than \( HE_{PRIV} \). However, we can’t say that this is also true for the poorest countries. Our results (Linden and Ray 2017) with a longer data set (34 OECD countries in years 1970–2012) and different methods show that public expenditure effects in non-poor countries dominate the private ones. We argue that as the expenditure co-movement (i.e. correlation between public and private expenditures) is smaller in poor countries compared to the rich nations, correctly targeted public health expenditures and their
marginal increases can matter much in low income countries. A general policy option is to develop and subsidize the use of public health care services so that the poorest can have access and resources for them.

3.3 PAPER 3


If the spread of mortality rates across the countries decreases in time, then some convergence in mortality rates happens between the countries. Likewise, if the absolute growth rates of declining mortality rates are larger in poor countries compared to rich ones, then the high levels of mortality rates in poor countries will eventually reach the levels of rich countries. The paper gives results on HS convergence as affected by the use of HCT globally and attempts to validate the global catch-up hypothesis caused by HCT diffusion. The paper’s target is to test for convergence and to show impact of HCT on tuberculosis (TB) and cancer mortality in different countries classified by per capita gross national income (GNIc) levels.

The theoretical basis of convergence is derived in the neoclassical growth models which support the result that in the long run countries move towards a common steady state level of income per capita because of decreasing marginal product of capital, i.e. the steady state is obtained under certain conditions. Convergence is connected to long-term growth. In case of long run mortality rates, convergence is due to the existence of upper bounds of health indicators and diminishing health returns of HCT capital inputs (Gächter and Theurl 2011, Grossman 1972).

The paper uses two methods to study the question of convergence besides trend growth models. In sigma (σ)-convergence, a cross-section standard deviation (SD) or coefficient of variation (CV) of a variable across a group of homogenous countries decreases over time. As SD is defined as a numerical measure of the average variability around the mean, and if its value diminishes with successive measures over time, this then supports the hypothesis of convergence (Nixon 2000b). The second approach limits the shortcomings of the above σ-convergence, positing that convergence exists if e.g. a poor economy tends to grow at a faster rate than a rich one and the poor country catches up with the rich one. This property corresponds to the concept known as beta (β)-convergence or regression to the mean (Barro and Sala-i-Martin 1992b, Sala-i-Martin 1996, Boyle and McCarthy 1997). Within this method one finds the unconditional or absolute and conditional convergences. Here we use also the convergence testing framework based on linear deterministic trend growth models with serially correlated errors (e.g. see Linden 2002). These model alternatives help us analyze if the growth process between the different income wise country groupings are different or not.

We analyze two illness related mortality rates (WHO 2015a, b) with 144 countries during the period of 1970–2012. We also analyze periods 1995–2012 separately with 196 countries, because in the years 1970–1995 the mortality rates in many countries are measured poorly. To get a more reliable picture of how TB and cancer mortality rates are affected by HCT, health care resources and relevant socio-economic variables are also added to the test models. The first study variable is $TBM_i$ (TB deaths per 100,000 persons including HIV positive cases) for 196 countries. The second is $CM_i$ (cancer deaths per 100,000 persons and includes summation of deaths caused by 24 types of
cancer) in 144 different countries. Data is obtained from different sources (e.g. World Bank 2015a-f, Gapminder 2015, WHO 2015a, b).

The differentiation of the sample countries into 4 groups is done by their income levels using the World Bank’s Atlas method (using current US$): Group 1 (30 low income economies) consists of countries which have a \( GNIc \) of $1045 or less. Group 2 (49 lower middle-income economies) refers to \( GNIc \) of $1046 – $4125. The 3rd group (55 upper middle-income economies) has a \( GNIc \) of $4126 – $12735. Group 4 (62 high income economies) has a \( GNIc \) of $12736 and above.

The paper uses following methods:

(i) \( \sigma \)-convergence: The sample variance of log of variable \( y \) in time \( t \) is given by

\[
\sigma_t^2 = \frac{1}{N} \sum_{i=1}^{N} (\ln y_{i,t} - \mu_t)^2 ,
\]

where \( \mu_t \) is the sample mean of (log) income at time \( t \).

(ii) Trend growth panel fixed effects model:

\[
\ln y_{i,t} = a_{i,0} + b_1 t + b_2 t^2 + \epsilon_{i,t} \quad (t = 1, \ldots, T)
\]

where \( a_{i,0} \) refers to the country specific starting value of growth process and coefficients \( b_1 \) and \( b_2 \) measure the growth rate and the acceleration of growth process during the sample period. When analyzing the time evolution of \( TBM \) and \( CM \) we expect growth rates to be negative with enforcing acceleration, i.e. \( b_1 < 0 \) and \( b_2 < 0 \), if mortality rates are decreasing rapidly. The estimates for model parameters are obtained more efficiently with models

\[
\ln y_{i,t} = a_{i,0} + b_1 t + \epsilon_{i,t}, \quad \text{and}
\]

\[
\Delta \ln y_{i,t} = b_{1,1} + b_{2,1} t + \epsilon_{i,t}^*,
\]

where \( b_{2,1} = 2 \times b_2 \) and \( \epsilon_{i,t}^* = \Delta \epsilon_{i,t} \). We allow also in the second equation country specific accelerations to be present in model. This model has standard properties as \( \epsilon_{i,t}^* \) are expected to be non-auto correlated. However, both heteroskedastic and clustered errors are still present. Thus, models are estimated with country specific weights and White’s HSCE -corrections.

(iii) Convergence models: \( \beta \)-convergence

\[
\ln \left( \frac{y_{i,t}}{y_{i,t-1}} \right) = a + \beta \ln y_{i,t-1} + u_{i,t}
\]
where $-1 < \beta < 0$. The error term $u_{it}$ is independent over $t$ and $i$ and has mean zero and finite variance $\sigma_u^2$. Because $a$ is assumed to be constant across economies, steady state growth rates are identical, but economies can have different growth paths to it, depending on their initial states. This is the case of unconditional or absolute $\beta$-convergence, i.e. average growth rates of poor economies are unambiguously greater than those of rich economies because of global catch-up effects (e.g. Sala-i-Martin 1996, Young et al. 2008). Allowing for heterogeneity across the economies ($a_i \neq a_j$), $-1 < \beta < 0$ would imply the case of conditional or relative $\beta$-convergence. The average growth rate of an economy is an increasing function of its distance from its own steady state growth level.

In practice we estimate following three initial-period cross-sectional $\beta$-convergence (or Barro) regressions with OLS and quantile (median) methods:

$$\ln\left(\frac{y_{iT}}{y_{i,t_0}}\right) = \alpha_i + \beta_1 \ln y_{i,t_0} + u_{i1}$$

$$\ln\left(\frac{y_{iT}}{y_{i,t_0}}\right) = \alpha_i + \beta_2 \ln y_{i,t_0} + \nu' \chi_{i,t_0}^{TECH} + u_{i2}$$

$$\ln\left(\frac{y_{iT}}{y_{i,t_0}}\right) = \alpha_i + \beta_3 \ln y_{i,t_0} + \nu' \chi_{i,t_0}^{TECH} + \delta' \chi_{i,t_0}^{SOC} + u_{i3}$$

Here $\chi_{i,t_0}^{TECH}$ and $\chi_{i,t_0}^{SOC}$ refer to the HCT and socioeconomic variables that condition the $\beta$-convergence.

Our analysis implies that for decreasing TBM and CM the trend growth estimates are the largest in absolute terms in low income and lower middle-income countries. Whereas in TBM one sees a speeding up of declining mortality rate for non high-income countries, just the opposite effect is valid for CM, where the speed of decreasing CM is speeding up only in high-income countries. There is more evidence of $\sigma$-convergence in TBM than in CM in 1970–2012. However, one can argue that average TBM and CM decline for all country groupings in years 1970–2005, but in the period after 2005 they increase up to 2012 especially for cancer in non high–income countries. There is more evidence of $\sigma$-convergence in TBM than in CM in 1970–2012. However, one can argue that average TBM and CM decline for all country groupings in years 1970–2005, but in the period after 2005 they increase up to 2012 especially for cancer in non high–income countries. In different income groups, one sees clear convergence with unconditional Barro-regressions for both TBM and CM covering the period 1970–2012. For conditional $\beta$-convergence, when HCT and socio-economic variables are added into models, low income countries have the fastest convergence. However, when analyzing the period 1995–2012, TBM in low-income countries still shows the fastest $\beta$-convergence, but with cancer rates the convergence disappears with HCT and socio-economic variables.

The results indicate that the cancer mortality rates are very country specific and when conditioning the rates in different income groups the convergence is not always present anymore. Thus, the global (conditional) convergence still seems to be missing for cancer mortality rates albeit the fact that the average rates are declining almost till the end of the sample period. Contrary to CM results, the TBM results show that
a catch-up is taking place in poorer countries through the diffusion of HCT from the richer nations. One can argue that CM is not responding as well as TBM to diffusion of HCT. The fact that cancer treatments are more expensive than TB treatments mean that it can take still some time for old and new cancer treatments to diffuse into poorer countries.

3.4 PAPER 4


Today, the health of most people in the world depends on their resources and ability to use and adopt health knowledge and HCT. Incomes marginally have still a large positive effect on health at least in the poorest countries. Good health can be considered as a form of human capital that has a beneficial effect on productivity, has the capacity to generate higher earnings and more consumption including for health care, and gives us longer life cycles. However, the income effects on health and health effects on income are not equally distributed. Wealthier and healthier people can provide higher investments in health capital leading to higher incomes and better health.

In response to this income-health endogeneity, the paper proposes a simultaneous three equation model between levels of GDPc, HS and HEc for the data set of 194 countries in years 1990–2014. In this framework INEQ is one of driving factors of income level and growth, and HS. However, without paying attention to a country’s starting level of GDPc, impacts of INEQ on GDPc and HS are not properly analyzed. To obtain a compact approach on net health-income effects, the analysis is cast in the framework of Kuznets’ hypothesis, maintaining a positive inequality relationship with income for poor countries contrary to the rich ones. We test for this low-income high-inequality trap and argue that it can be escaped by raising the HE/GDP ratio with cost-effective HCT. Our argument is that the poorest countries can do this even when the Kuznets’ hypothesis is present. This needs large improvements in their HS and inequalities to create a strong push effect in the growth direction. The following three equation model captures the inter-linked health and income effects that are conditioned with prevailing INEQ in the economy.

\[
\ln\text{GDPc}_i = \alpha_0 + \alpha_1 \ln\text{HS}_i + \alpha_2 \text{INEQ}_i + \alpha_3 \text{INEQ}^2_i + \alpha_4 \ln\text{POP}_i + \alpha_5 \ln\text{EDUC}_i + \alpha_6 \ln\text{TECH}_i + \epsilon_{1i},
\]

\[
\ln\text{HS}_i = \beta_0 + \beta_1 \ln\text{GDPc}_i + \beta_2 \text{INEQ}_i + \beta_3 \tilde{X}_{1i} + \epsilon_{2i},
\]

\[
\ln\text{HEC}_i = c_0 + c_1 \ln\text{GDPc}_i + c_2 \ln\text{HS}_i + c_3 \tilde{X}_{2i} + \epsilon_{3i}.
\]

The first equation is a typical empirical GDPc level equation based on the production function, augmented with the economy’s HS and a 2nd order polynomial in INEQ to
measure the non-linear GDPc effects. Population and educational variables measure the effects of labor and human capital inputs on income. Variable TECH stands for the capital input measured as the nation’s technological status improves. The second equation gives the determination of nation’s HS related to GDPc level, INEQ, and other health improving variables X. Finally, HEc is determined by GDPc level, HS and some HE related variables X. A similar system is also specified for GDPc growth.

To test Kuznets’ non-linear GDPc–INEQ hypothesis we divide our data (194 countries) in three income groups in years 1995–2014: (i) S1: countries where average GDPc level is less than 1 000 US$, (ii) S2: group of countries with GDPc level between 1 000–10 000 US$, and (iii) S3: group of countries with GDPc level above 10 000 US$.

In general terms, we argue that the income level (and growth) of economy is determined simultaneously with its HS and HE levels. This means that in the models at least variables lnGDPc, ΔlnGDPc, lnHS and lnHEc are endogenously determined. The structure of the model is identifiable and needs IV estimation approach. This is conducted with the following approach. First, all our modeling variables are measured with the mean values of country specific values for the period 1995–2014. This makes the measurements independent of cyclical effects. Subsequently we can focus on average long run results. Second, we use means of variable measurements from the period 1990–1994 as instrument variables in model estimation. Thus, our instrumentation leads to predetermination of variable values in relation to model estimation periods. Variables which are dated at the beginning of the sample period 1990–1994 minimize the problem of endogeneity. Note that if the variables are highly persistent, endogeneity may still persist. More precisely, we argue (i) that the variable set for period 1990–1994 does not correlate with the model errors, and (ii) that our additional instruments do not correlate either with the model errors or with the left-hand side endogenous variables. These conditions create the need for finding proper instruments that are not ill-conditioned either by weak instruments or by endogeneity. Additional instruments consist of variable values of alcohol consumption per capita, percentage of population using improved drinking water source, geographic area of country in km², and the means of GDPc values of countries that have the same level of development as country i. Data sources are mainly World Development Indicators (World Bank 2016), Global Health Observatory Data (WHO 2016), UNO Data (UN 2017), and UNICEF statistics (2016). Conducted IV tests do not reject our modelling approach.

Estimation results (SURE, GMM-2SLS) indicate that Kuznets’ hypothesis is still valid: the positive inequality relationship with income level is still relevant for poor countries with GDPc level less than 1 000 US$. The modern version of hypothesis maintaining that INEQ has negative effect on the growth of GDPc is not rejected for all country groups with different GDPc levels. Our hypothesis of positive simultaneous health-income relationship is not rejected, e.g. HS measured with IM has a negative effect on the level GDPc and increasing GDPc lowers IM. In the poor economies the net effect between GDPc and IM in lnGDPc and lnHS equations is large. A 10 % decrease in IM means 13.2 % increase in GDPc level, that feeds back (ceteris paribus) to a lower IM with an estimate value of 2.34 %. However, the INEQ effects found in poor economies on GDPc and IM make this less evident as IM, contrary to LE effects (see Paper 1), is not directly affected by INEQ measured with GINI coefficient. Thus, the problem of IM in poor economies is not so much caused directly by the INEQ but by the low level of GDPc that is however rising with INEQ. In all country income groups, the summed elasticity of lnGDPc and lnDOC on total lnHEc is larger than 1.
From the economic growth policy perspective, obtained results mean that the policy combining exogenously determined fight against high IM and INEQ refers to higher GDPc level in long run. In the poorest countries Kuznets' hypothesis and low-income-high-inequality trap may still be present. These can be avoided by breaking the negative relationship between INEQ and HS augmented with large investments in health with higher HE/GDPc and HS/HE-ratios.
4 CONCLUSIONS

The dissertation has shown how income, health and inequality are intricately interrelated to each other. The target of the thesis is to analyze the similarities and dissimilarities that exist in the income-health relationship in the presence of income inequality when comparing the developed and the developing countries. We observe that only in recent times economists have realized that health is an important part of human capital formation. Hence investments in health have become almost synonymous with investments in human capital at least in the poor countries. In accordance with this modern trend, economists have also started rethinking in details the relationship between income, health, and inequality on macroeconomic level with microeconomic foundations. With this background, the dissertation provides some new results on this research agenda.

The dissertation contributes to existing literature in many ways. Paper 1, proposes a novel bias correcting method to analyze the income effects on average health while not being affected by data aggregation problems. Paper 2 applies some new and alternative instrumental variable methods to dynamic panel data models with effects of public and private health expenditures on health status in countries belonging to different income categories. In paper 3, trend growth empirics and convergence metrics – usually used in macroeconomics – are applied to health economics data to test the impact of global diffusion of health care technologies for chosen health status variables like mortality rates for certain diseases. Such applications are rarely found in health economics literature. Finally, paper 4 combines the modern rendition of Kuznets’ hypothesis (1955, 1966) with the research agenda by Deaton (2003, 2006, 2007, 2013) and health economics approach by Grossman (1972). A novel approach is proposed to test for the Kuznets’ hypothesis with global data. There are hardly any results in the literature where Kuznets’ hypothesis is used to analyze simultaneous effects between income inequality, health expenditures, health status and income.

In details, the first paper stresses the importance of individual data in deriving results for health as a function of income and its distribution. However individual data is sparse for the most vulnerable developing countries and we can’t ignore the aggregate income effects on health. Since the part of the inequality effect on average health with macro data is built on the aggregation of individual concave income functions, the entailed aggregation problem is solved with a method that is based on the first order Taylor approximation of the log of average incomes. Two bias correcting model alternatives are proposed while preserving the individual level interpretation of estimated income effects on average health. The results show that bias correcting models give quite different results of $lnGDPc$ effects on life expectancy across the sample countries in the 41 years under consideration. In the sample period, the not-corrected model gives too large income effect estimates compared to the biased corrected ones. However, the income inequality effects estimated with GINI coefficients are not affected by the model alternatives. In order to give more transparent income and inequality effects on life expectancy distribution across the sample countries, the bias correcting model is estimated with the quantile regression approach.

The bias corrected estimated coefficients for $lnGDPc$ are significant all throughout the four decades in both OLS and median LAD regressions. However, the coefficient values on $lnGDPc$ decrease over the years for all countries. The results with quantile
regression other than median for four different decennials in the sample show that
the poorest countries' income gradient is still much higher than for the rich nations.
For the \textit{GINI} coefficients with 148 countries, one sees a similar diminishing inequality
effect. Richer countries are comparatively less affected by changes in \textit{GINI} than the
poorer ones. These results are not new to the literature, but the contribution of the
paper is the estimation solution that it not sensitive to aggregation. The proposed
bias-correcting method is novel, and it is also applicable in other fields.

The second paper introduces some less used methods to overcome econometrical
problems of dynamic panel data model estimation. The short panel bias of \textit{FE/RE}
estimator and the efficiency problems of \textit{IV} approach for the first difference model
are well-known. In response to this we use estimation methods that are planned to be
robust enough against near unit-root case and are allowed to avoid strict exogeneity
assumptions. To analyse effectively with these methods the public and private health
expenditure effects on life expectancy (\textit{LE}) and infant mortality (\textit{IM}), the \textit{K}-means
cluster method is used to identify clusters of 195 countries with average country
specific growth rates of \textit{LE} and \textit{IM} in sample period 1995–2014. We divide the
countries into two groups based on their average level of \textit{GNIc} during the sample
period. Generally, different estimation results suggest that if public expenditures
increase 10 \%, it increases \textit{LE} by 0.36 \% and reduces \textit{IM} by 2.31 \%. Similarly, for
private expenditures we find 0.23 \% and -0.83 \% mean elasticities respectively. These
quite novel estimates imply that globally public health expenditures are more effective
to improve health status than private expenditures. Although the new estimators
provide some new valuable information on expenditure effects on \textit{LE} and \textit{IM} on the
global scale, they do not show promised robustness. Albeit the existing problems with
methods we can argue that correctly targeted public health investments and their
marginal increase can matter much in low income countries.

From the health policy perspective these results are interesting. The policy option
is the shift of resources from private to public sector. However, in practice, this is
difficult, as it can harm the sovereign consumers, who now will have to pay more
taxes and face increased regulations. Less rich countries’ policy option for increased
public health investments needs to be backed with equity arguments since private
care is expensive. An alternative option is to subsidize the production of health care
services, so that the poorest can afford them also. The present analysis in this regard
posit future new research that can enable us to get a detailed picture of different health
contributions of private and public health expenditures across income and wealth
distributions between and within countries.

To see the interactions between health status and health care technology, which
forms a big chunk of health expenditures, and how such expenditures improve
economic growth and well-being, the global time evolution of two extensively
prevalent global diseases are analyzed in the third paper of the thesis. The results
imply that for decreasing \textit{TB} and cancer rates the trend growth estimates are the
largest in absolute terms in case of low income and lower middle-income countries.
Whereas in \textit{TB} one sees accelerating effect on the declining rate of mortality for non
high-income countries, just the opposite effect is valid for cancer rates. Overall, there
is more evidence of \textit{σ convergence} in case of \textit{TB} than in case of cancer rates in years
1970/1995–2012. However, one can argue that average \textit{TB} and cancer mortality rates
are going down for all country groupings in 1970/1995–2005 periods, but thereafter
till 2012 this declining process slows especially for cancer in non high-income nations.
For \textit{absolute β convergence}, for both cancer and \textit{TB}, one sees clear convergence between
the years 1970/1995–2012 for different country income groups. With conditional \( \beta \) convergence for low-income countries, when health care technology and socioeconomic variables are added to test models, the mortality rates have the fastest convergence respectively.

Although these interesting and novel results indicate that cancer mortality rates are very country specific, we conclude that cancer mortality rates do not respond as well as TB to the diffusion of health technology. The fact that the prevalence, diagnosis and treatment of TB has been present for a while, but the diagnosis and treatment of cancer effectively are comparatively a newer phenomenon, explains the obtained results. In addition, as almost all R&D on health technology happens in large scale only in the richer countries, we need here international agreements and actions to get effective cancer treatments exported to poorer countries.

As income-health relationship lie in the core of health economic analysis, both at micro and macro levels, the simultaneous relationships between health status, health expenditures, and GDPc level (and growth) in presence of income inequality must be addressed. The final paper of the thesis proposes a novel system model approach on the sample data set of 194 countries in the years 1990–2014. Cross-section country mean-values of relevant variables in the period 1995–2014 are used as the estimation sample. The cross-section data is divided into three country groups based on the mean GDPc level values for the sample period. This is done to test for the Kuznets’ hypothesis maintaining a positive relationship between GDPc level and income inequality in poor countries contrary to the rich ones. The simultaneous three equation models for GDPc, IM and HE are estimated with system IV methods.

The estimation results indicate that the simultaneous effects between IM and GDPc level are found in all country groups sustaining decrease in mortality rates with an increase in GDPc level. There is positive feedback effect from declining mortality rates to GDPc. Estimation results do not reject the Kuznets’ hypothesis and we find a positive GDPc level and GINI coefficient relationship in poor countries. Additional results on GDPc growth identify negative GINI effects for all country income groups.

Results imply that in the poorest countries, in the framework of the Kuznets’ hypothesis, low-income and high-inequality trap can be eliminated with larger health expenditures shares in GDP along with usages of cost-effective health care technology. However, breaking the negative relationship between INEQ and HS in poor economics forms the most effective policy option to the health promotion policies. These conclusions are still valid for many poor and low-income countries. However, our results are based on rigorous econometric system modeling, estimation, and testing that is almost missing in the relevant literature.

On the general level the thesis confirms that income-health relationship is still alive and well on the global scale. Some positive progress has taken place in recent decennials in incomes and health, despite the presence of harmful inequalities. The dissertation shows that by reducing incomes and health inequalities, the poor economies can step in to the rapid progressing health-income nexus, wherein all the fruits of advances of present day high level health care technology can be utilized.
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ARTICLES

ARTICLE I

ARTICLE II

ARTICLE III

ARTICLE IV
ARTICLE I

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ABSTRACT

A large amount of data consisting of 148 countries for the years 1970 to 2010 is analysed in the context of the health–income relationship. The literature suggests that the biased income–health effect obtained with macro data can be a result of the aggregation of individual concave income functions on average health. This aggregation problem is analysed in detail, and a bias-correcting method is proposed to overcome it. The results with new model alternatives show that they correct the income effects on average health in the right direction, that is, they produce smaller parameter estimates than biased models. Augmenting the results with the quantile regression approach, which is sensitive to health differences between countries, indicates that the poorest countries’ income gradient is still much larger than that of rich countries. However, the median life expectancy effect of the log of GDP per capita across the countries decreased during the sample decennials. The results for income inequality measured with the Gini coefficient indicate that the effects of inequality on health are still significant in the poorest countries but non-significant among rich countries after the year 2000. We argue that the proposed bias-correcting method retains the interest in macro health modelling and offers new model alternatives in other contexts.

1. Introduction

Aggregation from microunits to macroentities is a lasting problem in economics. The literature reports many different forms of biases that macro or aggregative relationships will generate when compared with the underlying micro-level presentations. Typically these aggregation problems are case dependent, and in some cases conditions can be derived to preserve the structural micro model at the macro level (see e.g. Denton and Mountain, 2011; Monteforte, 2007; Pesaran, 2003). However, for the vast majority of cases, the conditions are very demanding or unknown, and quite often even a simple micro relation estimated with macro data has different parameter values from the micro data results. This is especially true for non-linear micro relations estimated with aggregate data, such as mean values. The major reason for these problems is that micro-model estimations with mean values are used instead of aggregating non-linear microrelationships.

For example, if the micro theory states that relationship $y = a + \beta \ln x$ is valid and we have data from two micro units $\{(y_1, x_1), (y_2, x_2), \ldots, (y_n, x_n)\}$, then $\bar{y} = a + \bar{\beta} \ln \bar{x}$ is not the same as $\bar{y} = a + \beta (\ln \bar{x} - \ln \bar{x})$, and the OLS estimate from $\bar{y} = a + \beta \ln \bar{x}$ for $\beta$ is biased.

Interestingly, the log function with health status as a function of income is found to be highly important in the health economics literature. Besides the involved aggregation problem when estimating the function with aggregative data, it surprisingly produces a spurious negative correlation between health status and spread of income distribution. Thus, when we regress different countries’ index of health status on their GDP per capita level, for example, we obtain the result that larger income dispersion decreases health status. Evidently, this result is relevant to any other field in economics that uses a similar type of concave functions. Especially, any empirical research that connects aggregate measurements non-linearly based on micro-level arguments should be beware of the distributional effects that non-linearity exposes.

Partly in response to this aggregation problem in health–income relationship analysis, a vast literature exists that is shared by public health researchers, sociologists, and economists on health, incomes, and income inequality (Cutler et al., 2006; Deaton, 2006, 2003, 2002a, 2001; Judge et al., 1998; Kawachi and Kennedy, 1999; Leigh et al., 2009; Lynch et al., 2004; Marmot, 2002; Mullahy et al., 2004; Subramanian and Kawachi, 2004; Wagstaff and van Doorslaer, 2000). The main outcome of this perhaps disparate literature is that the income level matters for health, especially for poor people, and the
focus of research must be on the individual-data level. New and more sophisticated approaches are still used to analyse the connections between health, incomes, and inequalities with the addition of different health outcomes and many covariates and auxiliary variables (e.g. Kim et al., 2008; Mackenbach, 2012; Zheng, 2012).

We do not dwell on this new literature. Instead, we try to understand what the earlier literature offers on the methodological level and to provide some pathways to follow, especially when still working with aggregate data (Section 2). In particular, we show that the aggregation bias in earlier aggregate models can be reduced when attention is paid to the evident errors-in-variables problem in these models. Section 3 proceeds with the data presentation and three model alternatives to test the average health status dependency on the log of GDP per capita and the GINI index across nearly 150 countries in the years 1970 to 2010. The main estimation results are given in Section 4. We show that there exists — contrary to biased results — a U-shaped evolution between the life expectancy and the log of GDP per capita during the years 1970–2010 obtained with our new bias-correcting methods. In addition the proposed method provides results that are comparable with micro-level results. The conclusions are presented in Section 5.

2. Aggregation bias of the InGDPc variable in the health–income model

2.1. Health status as a function of income

Much of the so-called income gradient literature on health has focused on the individual-level relationship between income (\(Y\)) and health (\(H\)), which has a concave form:

\[
HS = f(Y) \text{ with } f' > 0 \text{ and } f'' < 0.
\]

(1)

The functional form in (1) is the absolute income hypothesis (AIH), which implies that

a) The health and the (absolute) level of income are positively correlated, and

b) The positive slope of the relationship decreases with income.

The AIH implies that the proportional relationship between health and good health is the same at all income levels, which infers that the absolute increase in health for each dollar of income is much larger at the bottom of the income distribution than at the top (Deaton, 2002a, p. 4). The logarithmic function is a concave function, and it has been shown to have some good statistical properties in this context (Jones and Wildman, 2008). This means that the regression model augmented with other health-affecting covariates and controls \(X_i\), like

\[
HS = \alpha + \beta \ln Y_i + \delta X_i + u_i
\]

(2)

is a promising starting point for health–income gradient analysis.

The problem here is that the AIH is not a robust hypothesis at the aggregate level. This is because the aggregation is distribution free only when the relationship between health and income is linear. Thus, regressing the average health on the average income non-linearly and on income distribution measures produces results that are artificial, since all the higher moments – not only mean income – exist for any non-linear function of a (random) variable.

2.2. Aggregate approach

Consequently, individual-level model results do not necessarily hold for aggregate data-level models, which are widely used, for example, in the social policy literature. Now the regression models appear as follows:

\[
HS = \alpha + \beta \ln Y_i + \delta X_i + u_i
\]

(3)

where \(k\) can refer to different regions, income classes, or social groups.

Similar AIH distributional effects are now valid as above if we refer to the average income differences between the \(k\) units in the sample. However, this agenda is not fully warranted, since the model in Eq. (3) is still prone to the aggregation problem.

If we take the starting point that health is an individual-level phenomenon, we should derive Eq. (3) in an appropriate way, starting from the individual level; that is, Eq. (3) is not aggregated correctly if Eq. (2) is the true model for individual behaviour in unit \(k\). This can be shown in the following way. Assume that for all individuals in \(k\), the following income gradient model is valid (excluding the additional terms in Eq. (3) for simplicity):

\[
H_{Si} = \alpha + \beta \ln Y_i + \epsilon_i
\]

(4)

The k-level aggregate equation is obtained by adding this equation over \(n_k\) income receivers in aggregation unit \(k\):

\[
H_S = \frac{1}{n_k} \sum_{i=1}^{n_k} H_{Si} = \alpha + \beta \left( \frac{1}{n_k} \sum_{i=1}^{n_k} \ln Y_i \right) + \epsilon_i
\]

(5)

This aggregate does not include any biases, since the aggregation is made over the individual-level income gradient functions; that is, \(\sum_{i=1}^{n_k} f(Y_i) = f(\sum_{i=1}^{n_k} Y_i)\). The aggregation in Eq. (5) provides the mean of log incomes, or the log of geometric mean income, \(\overline{Y} = \exp(\sum_{i=1}^{n_k} \ln Y_i)\). Thus, the correct form of Eq. 3 under the assumption that model parameters \(\alpha\) and \(\beta\) are equal across \(k\) different units is

\[
H_S = \alpha + \beta \ln \overline{Y} + \epsilon_i
\]

(6)

where \(\overline{Y} = \frac{1}{n_k} \sum_{i=1}^{n_k} Y_i\). Consequently, using the wrong presentation for individual behaviour at the aggregate level in Eq. (3) biases the income gradient estimates.

The genesis of the aggregation problem in Eq. (3) can be seen when we take the second-order Taylor approximation around the mean income for the concave income gradient function (Eq. (1)):

\[
H_S = f(\overline{Y}) + f'(\overline{Y})(Y - \overline{Y}) + \frac{1}{2} f''(\overline{Y})(Y - \overline{Y})^2 + K_c
\]

(7)

Thus, the sample estimate for the expected health level – the sample mean \(\overline{HS}\) – is related negatively to income dispersion because of \(f''(\overline{Y}) < 0\). Therefore, the larger is the spread of income in the economy, the lower is the average health level for the given mean income.

This transfer or “concavity-induced income inequality” effect (Deaton and Muellbauer, 1980; Stoker, 1983) is also called a statistical artefact of aggregation (Gravelle, 1998). The finding that there is a negative partial correlation between the average health and a measure of spread of income may therefore not be evidence that individual health is adversely affected by income inequality. It is simply the result of the curvilinear relationship between income and health level operating at the individual level (Gravelle et al., 2002, p. 579; Lynch et al., 2004, p. 61). Note that the result in Eq. (8) is still more complicated when we observe that income distributions are typically not symmetric but skewed; that is, \(E(\overline{Y} - \text{TFA}\{\overline{Y}\}) \neq 0\). To add a positive comment about the aggregate model, Eq. (3) entails that it reflects in an average sense the relationship between health and incomes at the aggregate level, but its individual-level implications are biased and even spurious.

The transfer result in Eq. (8) is most alarming for the income inequality hypothesis (III). It states that

a) Individual health is affected by income inequality, and
b) Greater income inequality produces worse health among the population.

Part a is the outcome of our individualistic approach to health, and part b asserts that the greater income inequality has an effect on the health status distribution; that is, the mean health level.

We see clearly that the AII and III are closely related to each other at the aggregate level as long as health is a concave function of income at the individual level; consequently, the second moment of income (dispersion) reduces the expected health. The curvilinear relation between income and health at the individual level is a sufficient condition to produce health differences between populations with the same average income but different distributions of income (Deaton, 2002b).

To overcome the problem of aggregation, we must turn to the individual-data model, which also entails some variables like the GINI coefficient to measure the income inequality effects on health. However, when individual data on health and incomes are not available, we need an aggregate model alternative that has a consistent aggregation solution; that is, it preserves the micro-economic presentation. In the following we propose a method that has some merits when we are bound to aggregate data and provides results that are not sensitive to aggregation biases.

2.3. Correcting for aggregation bias

The evident non-linear aggregation problem over individuals (Stoker, 1993) can be avoided by using \( \ln E(X) \) as a mean income variable. Thus, Eq. (6) preserves the individual-level interpretation. However, we seldom know the geometric mean of incomes \( \Gamma = \exp(\sum_i \ln Y_i) \). To calculate it, we need individual-level observations, and if we have them we should conduct individual-level regressions, not problematic aggregate-level regressions. Thus, the problem is basically that we are provided with only aggregate-level information, like \( H_N \) and \( \Sigma \), and are not able to obtain the correct aggregation form for the income gradient hypothesis. To overcome this problem, the following approximation method can be used. Jensen’s inequality implies for concave functions that

\[
\sum_{i=1}^{N} f(X_i) = f\left(\sum_{i=1}^{N} X_i - \theta\right) \tag{9}
\]

Applying this idea to the logarithm function in the context of the aggregate mean income gives

\[
\frac{1}{N} \sum_{i=1}^{N} \ln(Y_i) = \ln(\Gamma - \theta) \Rightarrow \theta = \Gamma - \exp\left(\frac{1}{N} \sum_{i=1}^{N} \ln Y_i\right) \tag{10}
\]

As we do not know the mean of log incomes – the log geometric mean is \( \ln \Sigma = \frac{1}{N} \sum_{i=1}^{N} \ln Y_i \) – and cannot solve for \( \theta \), we use the following practical second-best solution:  

The second-order Taylor approximation around \( \Gamma \) for \( \ln(\Gamma - \theta) \) has the following form:

\[
\ln(\Gamma - \theta) \approx \ln \Gamma - \theta / \Gamma \tag{11}
\]

For large values of \( \Gamma \), we can omit the second-order term. This gives the result

\[
\ln(\Gamma - \theta) \approx \ln \Gamma - \theta / \Gamma \tag{12}
\]

Applying the first-order result to our basic region- or group-level health model like Eq. (6) gives

\[
H_N = \alpha + \beta \left(\ln \Sigma - \frac{\theta_i}{\Gamma}\right) + \mu_i = \alpha + \beta \ln \Sigma - \beta \theta_i / \Gamma + \mu_i \tag{13}
\]

The model presents two options. First, if we make a heroic parametric assumption that \( \theta_i = \theta \) for all income units \( k \), we have

\[
H_N = \alpha + \beta \ln \Sigma - \gamma \theta / \Gamma + \mu_i \tag{14}
\]

This means that the parameter \( \theta \) (if needed) is identifiable from the GLS estimate for \( \gamma \).

Next we propose also a two-stage estimation procedure to estimate \( \beta \) consistently by using approximation result \( \ln(\Gamma - \theta) \approx \ln \Gamma - \theta / \Gamma \). Now we first regress \( \ln \Sigma \) on \( \theta_i / \Gamma \), and use estimate \( \hat{\theta}_i \) to correct for aggregation bias, i.e. we calculate in \( \Gamma = \ln \Sigma - \hat{\theta}_i / \Gamma \) and estimate model

\[
H_N = \alpha + \beta \ln \Sigma + \mu_i \tag{15}
\]

This procedure corresponds to the errors-in-variable problem (see Appendix A1) that is an alternative formulation of aggregation bias found in model \( H_N = \alpha + \beta \ln \Sigma + \mu_j \).

Finally, if we take the random-coefficient model (RCM) approach, which assumes \( \theta_i = \theta + \epsilon_i \), where for example \( \epsilon_i \sim N(0, \sigma_i^2) \), the model has the form

\[
H_N = \alpha + \beta \ln \Sigma - \chi_i - \frac{1}{\Gamma} + \mu_i \tag{16}
\]

The classical RCM approach needs a panel data setting, but in the modern RCM context (mixed or hierarchical models), we can use also cross-sectional data, where \( \chi_i \) is the classifying parameter for different regions or income groups.

The last question is whether we should also include in models 14–16 the GINI coefficient to measure income inequality and test for the income inequality hypothesis (IIH). Adding GINI coefficients to models can be defended with the arguments that GINI is based on person-level information and models 14 and 15 are approximations of a model that preserves the individual-level approach; that is, the spurious “con- vency-induced income inequality” effect is less evident in these aggregation bias-corrected models.

3. Data and test models

3.1. Data

Annual data from 148 countries (both developed and developing) covering 41 years (1970–2010) were collected from different sources. Data for life expectancy at birth as a measurement of the average population health were collected from the World Development Indicators (World Bank, 2014a).

Gross domestic product per capita data were PPP converted into GDP per capita (Laspesyes) at 2005 constant prices. They were collected from the Penn World Table 7.1 (Heston et al., 2012). The World Bank IBRD-IDA (2014b) database and the UN database (2014) were consulted for Georgia, Qatar and Latvia. Gapminder.org (2014) was accessed for data on Armenia, Azerbaijan, Belarus, Bosnia Herzegovina, Estonia, Lithuania, Kazakhstan, Slovenia, Turkmenistan and Ukraine.

Income inequality data were obtained from SWIID Version 4.0 from September 2013 (Bolt, 2009). The “gini market” data were taken, which were the estimate of the Gini index of inequality in equivalent (square root scale) household market (pre-tax, pre-transfer) income. Here the Luxembourg Income Study data were used as the standard. UNU WIDER (2014) data were also collected with reference to the unit of analysis. The data were more often weighted with household than personal weight. In connection to the area covered, data that covered both urban and rural areas were obtained as available in the database.
When considering the quality rating, only high- (i.e. 1) and average-quality (i.e. 2) rated observations were considered. Household income data mean cross income. With reference to revision, the newest observations were considered as far as possible. Data from the Inequality Project hosted by the University of Texas (University of Texas Inequality Project, 2014) complemented the data already mentioned.

3.2. Models and estimation strategy

The following 3 models with 148 countries in the years 1970–2010 (k=1,…,148, t=1,…,41, 6068 observations) were estimated:

Model A: \( L_{kt}^ε = \beta_0 + \beta_1 \ln G_{pk}^* + \gamma_1 G_{pk}^* + \gamma_2 \ln G_{lk}^* + \varepsilon_{kt}, \)

Model B: \( L_{kt}^ε = \beta_0 + \beta_1 \ln G_{pk}^* + \gamma_1 G_{pk}^* + \gamma_2 \ln G_{lk}^* + \varepsilon_{kt,2}, \)

Model C: \( L_{kt}^ε = \beta_0 + \beta_1 \ln G_{pk}^* + \gamma_1 G_{pk}^* + \gamma_2 \ln G_{lk}^* + \varepsilon_{kt,3}. \)

\( LE \) is the life expectancy at birth in years. \( GDPc \) is the GDP per capita at 2005 constant prices. These values were then multiplied by 0.01 and the natural log was taken (\( \ln GDPc^* \)). Income inequality was measured by \( GINI \) coefficients. The \( \varepsilon \)s are the error terms of the models.

The first equation (model A) is based on micro(economic) foundations within a person’s incomes and prevailing income distribution in his/her social environment have effects on his/her health. The variable \( 1/GDP_{lk}^* \) is the correction term stemming from the inadequate aggregation of the log of income effect on health at the micro level. Model B is the reference model that includes the aggregation bias, and the coefficient \( \beta_1 \) is expected to be biased upward. The model lacks the (micro)economic interpretation. Finally, model C corrects for the aggregation bias via the estimation of the errors-in-variables bias (for more details, see Appendix A; Stock and Watson, 2011, pp. 361–364).

\( \ln GDPc_k^* \) in model C is equal to

\[
\ln GDPc_k^* = \ln GDPc_k + \hat{\delta}_k \]

with \( \hat{\delta}_k \) coming from OLS regressions in \( GDPc_k \) on \( 1/GDP_{lk}^* \) for each of the sample countries separately with time series observations. Note that in this two-step estimation strategy for model C we need to correct the standard errors of coefficients in the second step estimation.

We consider first the following panel data models in models A–C.

Group mean model: \( \gamma_1 = \beta_0 + \beta_1 + \gamma_2 + \varepsilon_1, \) \( k = 1, \ldots, N \)

Fixed-effects model: \( \gamma_{kt} = \alpha_i + \beta_1 x_{kt} + \varepsilon_{kt}, \) \( k = 1, \ldots, N, \quad t = 1, \ldots, T \)

Random-coefficient model (RMC): \( \gamma_{kt} = \alpha_i + \beta_1 x_{kt} + \gamma_{kt}, \) \( k = 1, \ldots, N, \quad t = 1, \ldots, T \)

The group mean model is used here as it averages out the random measurement errors and collects observed and latent group heterogeneity in the group means (see Greene (2013), Section 11.3.4). The fixed-effects model is an obvious choice in this context. We estimate the model in the robust form; that is, augmented with White and Newey–West standard error corrections for group heterogeneity and autocorrelation in models A and B. For model C we use the clustering approach. Standard errors are calculated with clustering the errors at the country level. The fixed-effects model with AR(1) errors allows explicitly for error autocorrelation to be present in the model. Finally, the random-coefficient model (RMC) is used only for model A, in which we specify the aggregation correction to take place at the group level, meaning that each country has a specific correction parameter \( \gamma_k = \beta_k \varepsilon_k \), where \( \beta_k = \hat{\theta}_k + \hat{\varepsilon}_k \) (see above, Eq. (16)).

To have as many results as possible for the suggested aggregation correction method, we also estimate models A–C for 41 yearly cross-sections. This also gives a more detailed and transparent picture of how the life expectancy–income relationship evolved during the sample years. However, the cross-sectional OLS estimation gives only the average effect results for each year. We also need some distribution effects, as the countries in the sample show large discrepancies in life expectancy. We could estimate models for example on a yearly basis for different income level or health status country groups with separate OLS regressions. However, this would give a vast amount of inefficient OLS regression results. We can obtain better results more efficiently with the quantile regression approach, which has been quite popular in recent years (see e.g. Huang et al., 2007). It estimates the conditional quantile functions instead of the mean conditional function as OLS regression does.

By focusing only on the conditional mean function \( F(x) \), the OLS regression gives an incomplete summary of \( \{y_i, x_i\}_i \). When the sample size is small, the OLS model errors are heteroskedastic and non-normal-like in our yearly samples. Median and quantile regression methods have advantages beyond this, providing a richer characterization of the data. Median regression is more robust to outliers than OLS regression. Moreover, quantile estimators can be consistent under a weaker stochastic assumption than possible OLS estimation (Cameron and Trivedi, 2005, p. 85).

The linear quantile regression model can be defined as

\[
Q(x;q) = x \beta_q \quad \text{such that} \quad \text{Prob}[y \leq x \beta_q] = q, \quad 0 < q < 1.
\]

\( q \) refers to different quantiles, like \( q = 0.1, 0.2, \ldots, 0.5, 0.6, \ldots, 0.9 \), where \( q=0.5 \) gives the median regression (i.e. MAD or LAD estimator). When \( x \) is normally distributed, \( MEDIAN(x) = x \beta_q \) (Greene, 2013, p. 243; for more details, see Koenker (2005), Koenker and Hallock (2001)).

4. Results

4.1. Panel model results

Before presenting the estimation results, we show in general terms that the log of \( GDPc \) is a suitable transformation in this context. Fig. 1 shows on the left side the cross-plot between life expectancy (LE) and \( GDPc \) and then on the right side that between LE and \( \ln GDPc^* \) for all the data points in the sample (6068 observations). We add to the figures the marginal distributions, 95% correlation Cll, and non-parametric curve fitting results. The left graph shows a clearly concave relationship between life expectancy and \( GDPc \).

After taking the ln – transformation of \( GDPc \), the relationship turns linear. Some data points, at an especially low life expectancy, do not fit into this setting, but their role is non-significant. The left graph is very important per se as it shows how unequal the yearly \( GDPc \) distributions are. The same is true for life expectancy. Note also the flat part of the curve above 30,000 US dollars per year.

The panel model estimation results are collected in Table 1. The estimated coefficient values for the variables \( \ln GDPc^* \) and \( \ln GDPc^* \) support our results concerning the aggregation bias in model B. For all the panel data model estimations, the coefficients for \( \ln GDPc^* \) and \( \ln GDPc^* \) are smaller in models A and C than in model B. In the group mean and FE-AR(1) models, the coefficient differences are quite large, indicating that the results in model B are clearly biased. Note that the coefficient values are much smaller in the FE-AR(1) model estimations than in the other models, because they are based on quasi-differenced variable values depending on the size of the estimated AR(1) process. The estimated values for the AR(1) coefficients were in the range of 0.90 to 0.95. The model alternative with country-specific error autocorrelations gave similar results without altering the reported values in Table 1. Homogeneity of the country-specific fixed effects (a specific fixed effect \( a_k = \text{fixed} \) was rejected for all the FE models. Similarly, the \( x^2 \) test rejected the non-randomness of the coefficients in the RC model.
With respect to the GINI variable, the results in Table 1 are poor. Only in the group means models is the coefficient value for GINI significant in statistical terms with a non-positive sign implied by the income inequality hypothesis (IIH).

### 4.2. Cross-sectional results

The insignificant income inequality results with the FE and RCM panel models indicate that our panel model approach has been too general by masking the country heterogeneity and time developments in the life expectancy levels and their relations to income inequality. Note that the health sectors in both developing and developed countries experienced vast changes during the sample period of 1970–2010. We argue that yearly cross-sectional estimations track these time-dependent changes in the model parameter estimates better than the FE or RCM estimations do. Thus, to gain a more transparent picture of aggregation bias with respect to model B, next we estimate the models for each sample year separately. This will produce 41 different coefficient estimates for each year in the sample, proving how lnGDPc and GINI affected LE during the sample period of 1970–2010.

Thus, for models A–C, the total number of observations in each case is 148. Standard errors of the coefficient estimates were estimated with White’s diagonal HCSE corrections.

The following graphs (Fig. 2, below) show the yearly based cross-sectional regression coefficients from the estimated models (more detailed estimation results can be provided upon request). The most interesting result is the U-shaped evolution between the life expectancy and the log of GDP per capita during the years 1970–2010 obtained...
with our new bias-correcting methods. Model B, the biased reference approach, does not show this type of behaviour. The result indicates that model B is misspecified and harmed by the errors-in-variables problem. The relationship between the log of GDP per capita and the average health status, like life expectancy, is more complex between countries than model B implies. The extent to which the results are only an outcome of our bias-correction procedures or something else is an open question. However, the evident large negative correlation between the variables lnGDPc and 1/GDPc may induce some multi-collinearity into the analysis. The correlations are in the range of −0.818 to −0.886. These are not the levels causing refutation of the results with model A. Note that model C is not sensitive to multi-collinearity and it produces results that are closer to model A than model B. The estimated values for coefficients with lnGDPc and lnGDPc* decreased from the beginning of the sample period until the mid-1990s. They have the minimum values in the years 1994–1996, and after this period they start to rise again. This cannot be due to any business cycle-dependent phenomenon, since the observed U-shape is too smooth in shorter time periods. Thus, we argue that, across the sample of 148 countries, the average income effects on average health almost halved in the years 1970–1995 but afterwards started to increase again. The effects of income inequality on life expectancy are less conflicting between the models (see Fig. 3, below). The negative inequality effect on health became less prominent during the sample years. Irrespective of the model, in 1970 it was −0.4 and at the end of the sample period, 2010, it was −0.1 or 0 (with model C), meaning that the III effect measured with the GINI coefficient has lost its negative impact on life expectancy. This robust result is highly interesting, since it is similar to individual-data findings in developed countries. Income inequality, despite increasing globally after 1990, lost its impact on health after 2000. However, note that this is a mean regression result across the sample countries. It does not necessarily apply to all countries at different income levels. This means that we have to look at countries with different average health levels.

4.3. Quantile regression results

In general quantile regression methods provide a convenient approach to show how a regression model gives different response results of regressors when we concentrate on different parts of the model error term distribution. We estimated models A–C with quantile methods for data values of samples from different decades (i.e. for the 1970s, 1980s, 1990s, and 2000s). We report only the values for model A for the variables lnGDPc and GINI. Appendix A2 gives details of the decennial OLS and quantile regressions for model A. Note that the OLS residuals from the models are non-normal, in some cases having large negative tails (see Appendix A2). The results for models B and C are comparable and can be provided upon request (Fig. 4).

We observe that, for the countries with the lowest level of life expectancy (the poorest countries), specifically quantiles less than 0.3, the income gradient is still much higher than that for the countries with high life expectancy (quantiles more than 0.7), independently of the period used. However, the “income effect quantile curve” has shifted downwards, especially for the middle quantiles 0.3–0.7, from the level of the 1970s. We take this as evidence of

i) A decreasing median effect of the log of average income across the countries on life expectancy during the years 1970–2010, and
ii) In the poorest countries, the income health effects are still vastly more prominent than those in rich countries.

Fig. 5 presents the results for the income inequality variable with GINI coefficients. The inequality effects are still much larger in the poor countries than in the rich countries. The “inequality effect quantile curve” has shifted upwards, especially for the non-poor countries, and the upper 95% CI obtains the 0-line in the 2000s for the rich countries. Thus, we argue that

i) Income inequality has a smaller median effect on life expectancy across the countries in present times than earlier, and
ii) In the poorest countries, the income inequality health effects are still significant both in statistical and in health terms.

4.4. Related results in literature

The obtained results raise an interesting question: How they relate to the results found in literature? Before any comparison can be made we have to exclude all model specification that do not use the correct absolute income hypothesis (AIH) specification, i.e. health is a concave
Fig. 4. Quantile regression results with 95% CIs for the variable lnGDPc (model A) in the sample decennials.

Fig. 5. Quantile regression results with 95% CIs for the variable GINI (model A) in the sample decennials.
function of income. In their literature review Wagstaff and von Doerlaert (2000) notice that quite few studies - especially in public health literature - use the correct functional form (see also Wildman et al. (2003), Deaton (2003)). Note that our results being aggregation consistent, i.e. maintaining the micro relation, we could compare our results both to individual and aggregation level results. However individual level studies focus typically on observed dichotomous health level variable (i.e. 0=death/alive or sick/well) or on ordinal subjective health valuations, and the models are estimated with discrete choice methods. The coefficient values on log of income in these studies are not fully comparable with community or country based OLS models where continuous health measures like average life expectancy are regressed on log of mean income.

The study by Babones (2008) comes closest to our approach. He uses similar model and his data is from years 1970 and 1995 with 134 countries. He reports standardized coefficient estimates for log of GDP per capita on life expectancy at birth to be between 0.580 and 0.827 for both years and for different sample configurations. He uses crude aggregation bias correction method based on the lognormal distribution. He concludes that “…the ecological correlation between income and health is an overestimate of the individual correlation between income and health.” (p. 1622). The estimated income inequality effects on life expectancy measured with GINI-coefficient are between −0.412 and −0.163. Note Ellison (2002) gives similar results for 120 countries in year 1991 after experimenting with different income levels and functional forms. More recently Biggs et al. (2010) report the log of GDP per capita effects on life expectancy at birth to be 0.64. When they control for decreasing inequality and poverty the estimates are larger. They use fixed effects panel model for 22 Latin American countries in years 1960–2007.

Although the micro-based methods and results are not directly comparable with macro outcomes some results with valid correspondence can be found. Mackenbach et al. (2003) study with the LOESS-function the shape of the relationship between household equivalent income and self-assessed health in seven European countries during 1990s. They report that the relationship is generally curvilinear and characterized by less improvement in self-assessed health per unit of rising income. This result can be observed in all sample countries. A $10,000 additional household equivalent income is associated with an increase of 0.09–0.29 points of self-assessed health. Olsen and Dahl (2007) uses European Social Survey (ESS) data from year 2005 with 21 countries to model continuously survey health responses (self-assessed health with five categories) on many individual socio-economic variables and some macro variables. They use hierarchical linear model and report that log of GDP per capita is the indicator that is most strongly associated with better health after controlling for individual-level characteristics. The income effects for women and men were 0.618 and 0.478 in full sample and for labour force they were 0.067 and 0.493. They conclude that results are in line with previous findings (Castilla, 2004; Beckfield, 2004; Fritzell and Lundberg, 2005). Note that income inequality health effects – if present in these studies – were either non-significant or very small.

5. Discussion and conclusions

A large amount of data consisting of 148 countries in the years 1970–2010 was analysed in the context of the health–income relationship. The current literature emphasizes individual data, deriving results for health and incomes. However, the aggregate approach is still active, because country-level data on GDP per capita, income inequality, and average health status are still widening and gaining a longer time span. Both at the individual and at the aggregative data level, some results indicate that the absolute income effect (AIIH) on health is still strong but the inequality effect (IIH) is disappearing from developed countries.

The literature also suggests that part of the inequality effect obtained with macro data is a result of a concave mean income function on average health. We showed that this estimation strategy is seriously biased because of incorrect aggregation, which we analysed in detail. A method based on the first-order Taylor approximation is suggested to overcome this aggregation-induced errors-in-variables bias. Two bias-correcting model alternatives are provided that correct for aggregation bias and still preserve the individual-level interpretation of estimated income effects on average health.

The results show that bias-correcting models produce quite different results for the log GDP per capita effects on life expectancy across the sample countries in the years 1970–2010 from the biased non-corrected reference model. Especially in the period from 1985 to 2005, the biased model with yearly cross-sections gives income effect estimates that are too bias-corrected ones. However, the income inequality effects estimated with the GINI coefficients are not affected by the model alternatives. Across the models the inequality effects on life expectancy are still negative and are not significant in statistical terms when examining the income inequality effects estimated with the quantile regression models. To achieve more transparent income and inequality effects on life expectancy distribution across the sample countries, the bias-correcting model was also estimated with the quantile regression approach, which is sensitive to the life expectancy data distribution. It produces income regression effects at different quantiles of the life expectancy model error distribution.

The coefficients for the log of GDP per capita were significant throughout the four decades in both OLS and quantile regressions. In the decade of 1970–1979, when lnGDPc increased by 1%, life expectancy increased on average by 0.061 years in the OLS regressions and 0.051 years in the median LAD regressions. We know that the income gradient is higher for poorer countries than for richer ones. However, the coefficient values of lnGDPc fall over the years for all the countries. In the last decade (2000–2010), the respective lnGDPc values were 0.047 and 0.044 in OLS and median LAD, respectively. The results with quantile regression other than the median for four different decennials in the sample show that the poorest countries’ income gradient is still much higher than that of the rich countries.

When comparing the OLS and quantile regression results for the period 1970–1979 with the bias-correcting model, the significant coefficient estimates for the GINI variable are very similar (~0.351 for OLS and ~0.312 for median LAD). In the 1980s the effects of income inequality on life expectancy decreased marginally in absolute values. All the coefficients were, however, significant for both the regression types. Between 1990 and 1999 the income inequality effects on life expectancy were ~0.154 with OLS and ~0.062 with median LAD. In the 1990s the GINI coefficient with median LAD was less than that with OLS. One reason for this was that the changes in income inequality amongst the poorest countries still affected life expectancy more than the same changes did in the richer countries. In the last decade (2000–2010), when examining the GINI for the 148 countries, one can identify a similar diminishing inequality effect, and the richer countries were comparatively less affected by the changes in the GINI than the poorer ones. Note that the full-sample panel data model estimates for income distribution effects were insignificant.

In general terms we argue that our bias-correcting approach to the health–income relationship with panel FE and RCM, cross-section OLS, and quantile regressions is a promising modelling alternative that has shown its merits in this context. It corrects the (absolute) income effects in the right direction, and the results for income inequality do not conflict with the results found in the current literature based on micro data. The method retains the interest in macro data modelling and offers new model alternatives in other contexts. Future work with the method will show its full potential.
Acknowledgements

We gratefully acknowledge helpful comments from the editor and two anonymous referees. Errors remain ours.

Appendix A1. Errors in the variable problem in model $HS_{\beta} = \alpha + \beta \ln T + \epsilon$

The basic theory of measurement error and errors-in-variables bias in the OLS model has the following structure. The correct model with variable $x$ measured without errors is $y = \alpha + \beta x + \epsilon$.

However, if the true $x$ is unknown and we observe a poorly measured signal of it, say $x^*$, we have $x^* = x + \mu$, where $\mu$ is the random measurement error. It can be shown that the bias in the OLS estimation for $\beta$ is (see Stock and Watson (2011), pp. 361–363)

$$\beta_{\text{OLS}} - \beta = -\frac{\text{COV}(x + \mu, \mu)}{\text{VAR}(x^*)}$$

where $\beta$ is the true value. Now we apply this approach to the generic model $HS_{\beta} = \alpha + \beta ln Y + \gamma \ln G + \delta GINI + \epsilon$.

Note that, as $\mu = \frac{\gamma \ln G}{\theta}$ is not independent of $\ln Y$, the bias is

$$\hat{\beta}_{\text{OLS}} - \beta = -\frac{\text{COV}(\ln Y, \frac{\gamma \ln G}{\theta})}{\text{VAR}(\ln Y)}$$

Because of the result

$$\text{COV}(\ln Y, \frac{\gamma \ln G}{\theta}) = \theta \text{COV}(\ln Y, \frac{1}{\theta})$$

and since we know that $\text{COV}(\ln Y, \frac{1}{\theta}) < 0$, the bias now takes the form

$$\hat{\beta}_{\text{OLS}} - \beta = -\frac{\theta \text{ COV}(\ln Y, \frac{1}{\theta})}{\text{VAR}(\ln Y)} > 0.$$  

(A3)

The results state that the OLS estimate for $\beta_{\text{OLS}}$ in the model $HS_{\beta} = \alpha + \beta_{\text{OLS}} \ln T + \epsilon$ is biased upwards and the size of the bias is $\theta \text{ COV}(\ln Y, \frac{1}{\theta}) / \text{VAR}(\ln Y)$.

Now, if we use the augmented regression model A,

$$HS_{\beta} = \alpha + \beta_{\text{OLS}} \ln Y + \gamma \ln G + \delta GINI + \epsilon,$$

then the OLS estimate for $\beta_{\text{OLS}}$ in this model is smaller than that in the measurement error model; that is, $\beta_{\text{OLS}} < \beta_{\text{OLS}}$ because in the model the term

$$\text{COV}(\ln Y, \frac{1}{\theta})$$

corrects for the measurement error because $\ln G$ and $\frac{1}{\theta}$ are correlated.

An alternative approach to correct for bias is to use directly the approximation

$$\frac{1}{\theta} \sum_{k=1}^{N} \ln Y_k \approx \ln T - \frac{\hat{\theta}}{\theta}$$

by regressing $\ln Y$ on $\frac{1}{\theta}$. Thus, we regress on the time series observation of each country $k$

$$\ln GDP_{k,t} = \hat{\delta}_k + \frac{1}{\hat{GDP}_{k,t}} + \hat{q}_{kt}$$

(A4)

to obtain country-specific OLS estimates $\hat{\delta}_k$ and use these to obtain the transformed values

$$\ln GDP_{k,t}^* = \ln GDP_{k,t} - \frac{\hat{\delta}_k}{\hat{GDP}_{k,t}}.$$  

(A5)

In practice all this means that we should estimate equations

$$HS_{\beta} = \alpha + \beta_{\text{OLS}} \ln GDP + \frac{\hat{\gamma}}{\hat{GDP}} + \hat{\delta}_k GINI + \epsilon,$$

$$HS_{\beta} = \alpha + \beta_{\text{OLS}} \ln GDP + \hat{\delta}_k GINI + \epsilon,$$

$$HS_{\beta} = \alpha + \beta_{\text{OLS}} \ln GDP + \hat{\delta}_k GINI + \epsilon$$

(A)

(B)

(C)

and compare OLS estimates $\hat{\beta}_{\text{OLS}}$ with $\hat{\beta}_{\text{OLS}}$ to evaluate the estimate bias in model B.
Appendix A2. OLS and quantile (median) regression results for model A in the four sample decennials

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<td>Coefficient (p-value)</td>
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<td>54.922</td>
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<td>2.144</td>
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<tr>
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<td>0.734</td>
<td>0.764</td>
<td>0.715</td>
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<td>145.56*</td>
<td>925.12*</td>
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1) Bera–Jarque test for residual normality; χ²(2) test with the 5% critical value of 5.91.

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References

Mullaly, J., Roberts, R., Wible, B. 2004. Health, income and inequality: review and
ARTICLE II
Health expenditure, longevity, and child mortality:
Dynamic panel data approach with global data

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Abstract

Effects of public and private health expenditures on life expectancy at birth and infant mortality are analysed on a global scale with 195 countries in the years 1995 to 2014. The global data set is divided into country categories according to growth in life expectancy, decrease in infant mortality rate, and level of gross national income per capita. Some new dynamic panel model estimators, argued to be more efficient with high persistence series and predetermination compared to popular but complex GMM estimators, show that public health expenditures are generally more health-promoting than private expenditures. However, the health effects are not as great as primary education effects. Although the new estimators provide some new and valuable information on health expenditure effects on life expectancy and infant mortality on a global scale, they do not show desired robustness.

Keywords: health expenditures, low and high incomes, life expectancy, dynamic panel methods
JEL codes: I15, I18, H51, C30

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1. Introduction

Globally, health spending is highly unequal. It is even more unequally distributed than the national income of countries (Deaton 2013). Countries that spend little on health also have poorer health conditions. Although OECD countries have less than 20% of the world’s population, they accounted for over 80% of world spending on health at the turn of the new century, whereas the poorest three quarters of the world’s population accounted for only 7% of the world’s health expenditures (HE). Looking across regions, Africa contains about 10% of the world's population, yet it accounted for 3% of the world's health spending. Asia and the Pacific (including China), with almost 30% of the world's population, accounted for only 4% of the world’s health spending (OECD 2014; Poullier et al. 2002; WHO 2015).

Although health spending should affect health conditions, it is important to note that the efficiency of transforming spending into better health outcomes varies significantly. Even at low levels of HE per capita spending, some countries achieve better health than others. This means that there may be an opportunity for public policy to also make a difference. On the contrary, among high-spending countries, sometimes additional spending bears little relationship to improvements in life expectancy. This is one reason behind the concern in wealthier countries for cost containment.

During most of the second half and especially the last decades of the 20th century, public HE grew at a faster rate than the national income in developed countries. Empirical studies show that demographic factors, such as population ageing, have a positive effect on public HE growth, but rather of a second order, when compared with other drivers, such as income level, technology, relative prices, and institutional settings (Medeiros and Schwierz 2013; Oliviera Martins and de la Maisonneuve 2006).

The focus of this paper is on the following questions: The first one is that can public and private HE explain health status variables like life expectancy at birth and infant mortality across different countries? This question is answered on a global scale with 195 countries for the years 1995 to 2014. Our analysis concerns the varying health impacts of public and private HE. We expect them to have different effects, as their provision and demand are not distributed uniformly within the population in each country. In addition, the nature and the quality of public health services differ significantly from those of private ones, especially in the poorest countries. Thus, the second question of interest is as follows: Do increased public and private expenditures in not
high-income countries bring more improvements in health status compared to high-income countries? This is the well-known income gradient hypothesis. We apply some novel dynamic panel data methods (Keane and Runkle 1992, Hahn et al., 2007) to answer the above two questions. Combining these with $K$-means cluster analysis we obtain some new results with health expenditures effect on health status on global level.

This paper is divided into five sections. In the following section, we provide a short review of material relevant to our questions and of some seminal papers. The third section elucidates the data used and describes the models applied and the methods involved. The fourth section presents the results. Subsequently, the paper ends with section 5, which includes discussions and conclusions on the results.

2. Health expenditures and health status

2.1. Background

A major share of $HE$ is publicly financed (i.e. through taxes or compulsory social insurance contributions), at least in OECD countries. While some believe that this and general income level may raise public $HE$ because of additional demand resulting from a decrease in the net price of care, others suggest that the public financing of $HE$ serves as a restraining factor. Research has shown that income increases are not the only primary drivers of the increase in the health share of the $GDP$ (Gerdtham and Johnsson 2000). An interesting possibility is that institutional factors such as the spread of insurance coverage have not only directly encouraged spending but also have induced the adoption and diffusion of new medical technologies (Acemoglu and Finkelstein 2008). However, in high-income countries, macro-economic pressures on public budgets spill over to health budgets.

One approach to reducing the fraction of public financing is to increase out-of-pocket payments or private insurance. There are, however, major problems with this substitution. First, there is a limit as to how much out-of-pocket payments can be increased if the goal of equity is concerned. Second, private insurance as a means of financing poses a problem, because those with the highest potential expenditure also quite often have the lowest incomes. The public fraction of $HE$ is highest in rich countries, which also often have the highest total expenditure. Private financing dominates in low-income countries, where direct out-of-pocket payments are more important than private insurance (Gerdtham and Johnsson 2000).
Focusing on the three major sources of HE — tax-financed spending, social security spending, and private spending — one sees clear differences in the structures of health care systems. South Asia is the region with the largest private sector share and virtually no reliance on social security systems. Africa and the Middle East rely heavily on private financing, but appear to have larger public tax-based (or externally supported) sources. In East Asia and the Pacific region, private spending is also high, but the public share has a significant portion in social security, driven almost exclusively by China (Hu and Ljungwall 2013; OECD 2014; Poullier et al. 2002; WHO 2015). The Americas also rely heavily on private financing, but somewhat less than other regions. It is only in Europe and partly in the OECD that health systems depend less on private financing and rely instead on significant shares of both social security and tax-based funding.

The estimates of public expenditure on health (HEPUB) range from as low as 7% to almost 100% of all health spending. The wealthier and healthier countries tend to rely more heavily on public sources of funds as a share of total spending. Generally, HEPUB as a share of total health spending is poorly correlated with per capita GDP, even if the correlation is statistically different from zero (Ke et al. 2011; Poullier et al. 2002). However, once again, countries and regions vary significantly along this dimension. One way of looking at this wide range of public commitment to HE is to compare countries in groups with similar per capita income levels (GDPc) or health outcomes.

Although private health spending (HEPRIV) is overwhelmingly paid out-of-pocket, the share of private health insurance in total HE is insignificant in most countries. Prepaid private insurance accounts for more than 5% of HEPRIV in only about one-third of the world's countries. In those countries where private insurance has some significance, this averages only 26% of private spending, while private spending as a whole accounts only for an average of 10% of all health spending (Liang and Mirelman 2014; Poullier et al. 2002; WHO 2015). The bulk of private spending is paid out-of-pocket at the time of service. This out-of-pocket spending accounts for a much greater share of HE in relatively poor countries than in richer ones. The high level of out-of-pocket spending or very low public spending in some low- and middle-income countries stands out as one of the most troubling areas for public health policy.
There is a growing call for bilateral and multilateral agencies to increase their financial support to the health systems in low-income and high-disease-burden countries (e.g. Ebola epidemic in Western Africa in 2014–2015). There is also a growing concern among countries that provide grants and loans that their funds are targeted effectively to the populations with most need. However, most external funding goes to countries with large populations. In other cases, countries seem to be picked out for special assistance because they are recovering from war or dealing with severe hunger. Political attachments between particular countries also play a role in this context, but general, non-targeted aid seems to be unsuccessful (Deaton 2013).

Expenditure on health naturally depends on the number of people in need of health care. This is determined by factors such as population size and age composition. Expenditure is perceived to increase considerably at older ages, as older people often require costly medical treatment due to multi-morbidities and chronic illnesses. Improvements in life expectancy may therefore lead to increases in HE if not accompanied by improvements in general health status. The relation between life expectancy and HE is complicated by the fact that it is also influenced by the proximity to death, at least in the high-income countries.

2.2. Econometric studies with HE effects on health outcomes

Improvements in human and health capital are critical catalysts to economic growth and development in the macroeconomic literature (Lopez-Casasnoves et al. 2005). Good health improves not only individuals’ consumption and production in the short run, but also returns from investments in productive activities and education in the long run. At the macro level, investment in the health workforce and infrastructure is expected to improve health conditions and hence the health capital of the population. However, in many developing regions where resources are relatively scarce, HE has received less attention in government budgets. At the African regional level, Anyanwu and Erhijakpor (2007) found with a panel data fixed effect model that total HE was a significant contributor to health outcomes, with a 10% increase in total health care expenditure per capita resulting in a 21% and 22% decrease in under-five and infant mortality rates, respectively. Akinkugbe and Afeikhena (2006) also provided evidence that the effect of health care expenditure as a ratio of GDP on life expectancy, under-five mortality, and infant mortality was positive and significant in developing regions.
Filmer and Pritchett (1997) provided evidence that while health care spending impacted on child mortality, it was not the dominant driver of this health outcome. Factors such as education, technological change, and social capabilities have been identified by some researchers as major drivers of health outcomes rather than health care spending (Caldwell 1986; Easterly and Levine 1996; Lleras-Muney and Sherry 2008; Musgrove 1996). To understand why public spending on health has failed to have a strong effect on reducing mortality, Filmer and Pritchett (1999) stressed in their study that they were not suggesting that medical services are not (potentially) effective. They were not arguing that penicillin, immunizations, or oral rehydration therapy are ineffective as health interventions. But the impact of \textit{HEPUB} on health is much more complicated than the effectiveness of particular services purchased. For public spending to improve health cheaply (i.e. if money mattered), three things need to happen. Firstly, public spending is required to create effective health services. Secondly, the existence of those new public services is expected to change the total amount of effective health services consumed by the population. Thirdly, the additional services consumed must be cost-effective in improving health. If any one of these conditions is not met, the actual cost of services becomes high (Deaton 2013, chapter 9; Filmer and Pritchett 1999).

Partly contrary to these results, some studies claim to have found a clear significant impact of \textit{HEPUB} on health (Anand and Ravallion 1993; Baldacci 2002; Bidani and Ravallion 1997; Jamison et al. 1996). For example, Bidani and Ravallion (1997) used a particular functional form to separate out the impacts of various variables on the poor and the non-poor. They found health status effects of \textit{HEPUB} spending for the poor but not for the non-poor. Their findings highlighted the importance of considering the incidence of the health benefits (i.e. some benefits helped best only the poor, so cuts without reallocation also fall on the poor). Note that Filmer and Pritchett (1999) also found that the impact on the poor versus the non-poor depended on the composition and efficacy of public spending.

One of the purposes of the study by Novignon et al. (2012) was to investigate the impact of total health care spending on various health outcomes after controlling for country-specific demographic structures and economic conditions. A differential analysis of public and private health care spending was also performed. The hypothesis was that there was no significant relationship between health spending and health outcomes in Sub-Saharan Africa. A further hypothesis was that there was no significant difference in the effects of public (\textit{HEPUB}) and
Results from the fixed and random effects models showed that one percentage point increase in total HE (as % of GDP) was more likely to increase life expectancy at birth by approximately 0.6–0.7 years. Disintegrating the effect of total HE showed that a 1% increase in both HE\textsubscript{pub} and HE\textsubscript{priv} significantly increased life expectancy at birth by about one and 0.4–0.5 years, respectively. Similar results were obtained for death rate (per 1,000 people) and infant mortality rate (per 1,000 live births).

For OECD countries, the HE effects on health are not directly comparable to the above low-income country results (see e.g. Barthold et al. 2014; Heijink et al. 2013; Jaba et al. 2014; Nixon and Ulman 2006; van Baal et al. 2013). Note that private vs. public expenditure effects are analysed in very few papers. Only the papers by Cremieux et al. (2005), Lichtenberg (2000), and Or (2000) provide some information on this question. Cremieux et al. (2005) use data on Canadian provinces over the period 1975–1998. They focus on public and private spending on drugs with many additional variables with panel fixed effects regression methods. Results show that for life expectancy at birth, increases in drug and private spending, effects are somewhat larger than public expenditure effect. Lichtenberg (2000) provides time series evidence from the US over the period 1960–1997 for life expectancy at birth in dynamic models where public and private expenditures predict life expectancy together with GDP and new molecular drug approvals. Public expenditure’s short- and long-term effects are statistically significant, but private effects are not precise, especially when lagged GDP is added in the model. Or (2000) uses similar methods as Cremieux et al. (2005) but explains premature death in 21 OECD countries in 1970–1992 with total HE and with share of public HE of total HE augmented with public health, environmental factors, and GDP. Note that Heijink et al. (2013) control for vast number of variables and time trends in their analysis, but HE remains a significant determinant of avoidable mortality.

3. Models, data, and methods

3.1. Setup

The argument that income level — either personal or GDP per capita level — determines the health conditions of individuals and the population is profound in the health economics literature. However, the heterogeneity in health status between individuals or nations, even at the same income levels, requires a more detailed relationship between health conditions and specific
expenditures targeted to promote health and care provision. The distinction between public and private expenditures here is important, since the former is likely a policy variable determined by the level of GDPc and the political agenda by the state and local public authorities, and the latter reflects the individual-level resources devoted to health care. Thus, both are endogenous variables in the long run. However, past findings suggest that the exogenous direct and delayed effects of \( HEPUB \) on life expectancy and on infant mortality are positive and significant. Taking \( HEPRIV \) as an exogenous variable is less warranted, as it is a form of derived demand (i.e. sickness and illness force people with short-run income constraints to put their money on \( HE \)).

Next, we propose dynamic panel data models to determine the levels of life expectancy at birth \((LE)\) and infant mortality rate \((IM)\), depending on \( HEPUB \) and \( HEPUB \). We prefer the logarithmic forms of the variables in the following dynamic panel fixed effects \((FE)\) model.

\[
\begin{align*}
\ln{LE}_i &= \alpha_0 + \alpha_1 \ln{LE}_{i-1} + \alpha_2 \ln{HEPUB}_i + \alpha_3 \ln{PCR}_i + \alpha_4 \ln{RDE}_i + \epsilon_{i,t} \\
\ln{IM}_i &= \beta_0 + \beta_1 \ln{IM}_{i-1} + \beta_2 \ln{HEPUB}_i + \beta_3 \ln{PCR}_i + \beta_4 \ln{FS}_i + \epsilon_{i,t}
\end{align*}
\]

In the first model, life expectancy is determined by private and public expenditures, and by exogenous variables of primary education rate \((PCR)\) and level of R&D expenditures per capita \((RDE)\). In the model for infant mortality, we replace \( RDE \) for food supply \((FS)\). We stress that \( HE \) are direct means and resources to achieve good health and care among the population in the country, not the income level of the country as such. Thus, in the above models, the level of education, level of technology, and \( FS \) per capita refer generally (among many other similar variables) to the country’s development level that sustains life expectancy and lowers infant mortality.

One-period lagged health variables \( LE_{i,t} \) and \( IM_{i,t} \), in models reflect the dynamics of health status (i.e. past health status affects the current one). Note, however, that both equations can be recursively solved for current and past values of other variables in the models and for the starting values of life expectancy and infant mortality (i.e. \( LE_{i,0} \) and \( IM_{i,0} \)). These and other variables’ effects on current values of \( LE_{i,t} \) and \( IM_{i,t} \) are determined by the sizes of adjustment parameters \((\alpha_i\) and \(\beta_i\)). If they are close to but below one, the past variable values can still have large effects on current-level health status (see Eq. 3 below).
In general terms, the model captures more directly the income–driven health part of the bi-directional income–health relationship (Weil 2009, chapter 6). Income per capita, and other indicators of the living standard determine the health status of a country’s population. For example, if primary schooling is missing and the FS per capita is low, the income level of the country is typically low and the average health status is also low. Evidently, the so-called growth process has not started or it has halted because of missing factors that are important to sustain income generation. Although the needs for health care and medication are most urgent, the resources for them are sparse, even missing, or used elsewhere.

Our main argument is that, at least for poor countries, the resources devoted to public health provision – the \( HE_{pub} \) – are more important for the population’s health status than the private expenditures. The reason for this stems from the large (income) inequalities prevailing in most poor countries supporting high incomes and \( HE_{priv} \) only for a small fraction of the population. The large population share of the poor can only get health benefits of public health care that is not exclusive.

3.2. Data

Annual data from 195 countries in the years 1995 – 2014 were collected from different sources. The life expectancy variable \( LE \) (life expectancy at birth in total years) was collected from world development indicators (World Bank 2015). Additional data for life expectancy for some countries were taken from Gapminder (2015). The infant mortality rate \( IM \) (number of deaths of less than one-year-old infants/1,000 live births) was provided by the World Bank and WHO. Total, public, and private \( HE \) as % shares of \( GDP \) (i.e. \( HE^s, HE^s_{pub}, \) and \( HE^s_{priv} \)) were taken from the Global Health Expenditure Database of the WHO (2015). Further health spending data for OECD countries were taken from OECD (2015) and derived for non-OECD countries with data on \( GNI \) per capita (formerly \( GNP \) per capita). \( GNIc \) is the gross national income, converted to U.S. dollars using the World Bank Atlas method, divided by the mid-year population (World Bank 2016). \( HE^s_{pub} \) and \( HE^s_{priv} \) were derived as fractions of \( GNIc \) with \( HE^s_{pub} \) and \( HE^s_{priv} \). The variable \( PRC \) (total of primary education completion rate as a % of the relevant age group) is the % of students completing the last year of primary school (World Bank...
R&D expenditures \((RDE)\) were derived from R&D expenditure as a \(\%\) of \(GDPc\) (World Bank 2015). Finally, we obtained the variable \(FS\) (kilocalories per person per day) from the World Bank (2016).

3.3. Country groups

In order to analyse effectively the public and private expenditure effects on life expectancy and infant mortality, we used the following country grouping strategy. We need different country clusters and groups to identify how public and private expenditure determines life expectancy and infant mortality that are quite heterogeneous across the sample countries and sample years. The \(K\)-means cluster method identified two clusters of countries with average country-specific growth rates of life expectancy in the sample period 1995–2014 (i.e. \(\frac{1}{T-1} \sum_{t=2}^{T} \Delta \ln LE_{t}\)). Table 1 reports the cluster mean values and number of cluster countries. We observe that in cluster 1 the mean growth rate of life expectancy was 3.5 times larger than in cluster 2. Typically, cluster 1 includes some of the poorest countries that have experienced significant health benefits from their care systems started in recent years.

Table 1. Clusters in average growth rate of life expectancy

<table>
<thead>
<tr>
<th>Cluster</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cluster mean</td>
<td>0.00872</td>
<td>0.00257</td>
</tr>
<tr>
<td>Number of countries</td>
<td>42</td>
<td>153</td>
</tr>
</tbody>
</table>

Next, the \(K\)-means method was also applied to growth rates of infant mortality rates. Due to the heterogeneous growth rates of infant mortality, the method proposed three clusters for average growth rates of infant mortality rates \(\left(\frac{1}{T} \sum_{t=2}^{T} \Delta \ln IM_{t}\right)\). Here cluster 3 comprises countries that belong to both \(LE\) growth clusters, that is, countries whose development process started before the sample period and their rapid progress in health status can also be seen in fast-declining infant mortality rates (e.g. China, Turkey, Brasil). Cluster 1 contains some of the poorest countries but also some developed countries that have already obtained a low level of infant mortality that is not declining anymore. Thus, most cluster countries here belong to life expectancy growth cluster 2. In infant mortality cluster 2, a typical country is a rich country (i.e. European country) with relatively low growth in life expectancy, but also some non-rich countries with a rapidly rising life expectancy (e.g. India).
Table 2. Clusters in average growth rate of infant mortality

<table>
<thead>
<tr>
<th>Cluster</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cluster mean</td>
<td>-0.0125</td>
<td>-0.0349</td>
<td>-0.0615</td>
</tr>
<tr>
<td>Number of countries</td>
<td>56</td>
<td>100</td>
<td>39</td>
</tr>
</tbody>
</table>

Finally, we divided the countries into two groups based on their average level of GNIc during the sample period. If a country’s average GNIc level was below 2,440 US$ during the sample years, it belonged to group 1 (77 countries, 39.5% of countries); countries with a level higher than 2,440 US$ formed group 2 (118 countries, 60.5%). Note that in the sample, the mean income is 1,0085 US$ and the median is 3,298 US$. Thus, 2,440 US$ is close to 75% of the global median income in the years 1995–2014. This means that group 1 countries are globally the poorest countries.

3.4. Summary statistics

Tables 3–5 provide detailed summary statistics in different clusters and income groups. In Table 3, clusters based on average life expectancy growth across the sample countries show that between low- and high-growth countries the difference between life expectancy is 11 years. Thus, during the sample period (1995–2014) the high level of life expectancy means less growth in life expectancy than at a lower level of life expectancy. The level of private and public HE per capita is 7 to 10 times larger in cluster 2 than in cluster 1.

Table 3. Summary statistics for life expectancy growth clusters

<table>
<thead>
<tr>
<th>CLUSTER 1</th>
<th>ΔlnLE</th>
<th>LE</th>
<th>HE_prv</th>
<th>HE_pub</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.0081</td>
<td>60.638</td>
<td>39.718</td>
<td>60.906</td>
</tr>
<tr>
<td>SE of mean</td>
<td>0.0012</td>
<td>0.286</td>
<td>2.174</td>
<td>5.976</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.035</td>
<td>8.287</td>
<td>63.016</td>
<td>147.119</td>
</tr>
<tr>
<td>CV</td>
<td>4.402</td>
<td>0.137</td>
<td>1.586</td>
<td>2.415</td>
</tr>
<tr>
<td>Median</td>
<td>0.0076</td>
<td>60.101</td>
<td>17.275</td>
<td>12.387</td>
</tr>
<tr>
<td>Sample size</td>
<td>798</td>
<td>840</td>
<td>840</td>
<td>840</td>
</tr>
<tr>
<td>Cluster 2</td>
<td>ΔlnLE</td>
<td>LE</td>
<td>HE_prv</td>
<td>HE_pub</td>
</tr>
<tr>
<td>Mean</td>
<td>0.0027</td>
<td>71.523</td>
<td>271.344</td>
<td>636.829</td>
</tr>
<tr>
<td>SE of mean</td>
<td>0.0002</td>
<td>0.141</td>
<td>8.285</td>
<td>0.236</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.0145</td>
<td>7.815</td>
<td>458.309</td>
<td>1119.407</td>
</tr>
<tr>
<td>CV</td>
<td>5.297</td>
<td>0.109</td>
<td>1.889</td>
<td>1.757</td>
</tr>
<tr>
<td>Median</td>
<td>0.0027</td>
<td>73.651</td>
<td>98.058</td>
<td>173.977</td>
</tr>
<tr>
<td>Sample size</td>
<td>2507</td>
<td>3060</td>
<td>3060</td>
<td>3060</td>
</tr>
</tbody>
</table>
Table 4. Summary statistics for infant mortality clusters

<table>
<thead>
<tr>
<th>CLUSTER 1</th>
<th>ΔlnIM</th>
<th>IM</th>
<th>HE_priv</th>
<th>HE_pub</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>-0.0146</td>
<td>40.583</td>
<td>234.764</td>
<td>420.675</td>
</tr>
<tr>
<td>SE of mean</td>
<td>0.0006</td>
<td>0.954</td>
<td>18.083</td>
<td>26.263</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.0024</td>
<td>37.924</td>
<td>603.199</td>
<td>882.296</td>
</tr>
<tr>
<td>CV</td>
<td>-1.482</td>
<td>9.756</td>
<td>2.577</td>
<td>2.048</td>
</tr>
<tr>
<td>Median</td>
<td>-0.0161</td>
<td>31.551</td>
<td>47.969</td>
<td>118.399</td>
</tr>
<tr>
<td>Sample size</td>
<td>1064</td>
<td>1120</td>
<td>1120</td>
<td>1120</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CLUSTER 2</th>
<th>ΔlnIM</th>
<th>IM</th>
<th>HE_priv</th>
<th>HE_pub</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>-0.0341</td>
<td>33.754</td>
<td>219.487</td>
<td>592.067</td>
</tr>
<tr>
<td>SE of mean</td>
<td>0.0006</td>
<td>0.618</td>
<td>7.410</td>
<td>25.985</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.0024</td>
<td>39.491</td>
<td>531.352</td>
<td>1162.122</td>
</tr>
<tr>
<td>CV</td>
<td>-0.721</td>
<td>9.903</td>
<td>0.786</td>
<td>2.577</td>
</tr>
<tr>
<td>Median</td>
<td>-0.0331</td>
<td>22.610</td>
<td>122.637</td>
<td>83.022</td>
</tr>
<tr>
<td>Sample size</td>
<td>1900</td>
<td>2000</td>
<td>2000</td>
<td>2000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CLUSTER 3</th>
<th>ΔlnIM</th>
<th>IM</th>
<th>HE_priv</th>
<th>HE_pub</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>-0.0592</td>
<td>26.900</td>
<td>207.348</td>
<td>427.396</td>
</tr>
<tr>
<td>SE of mean</td>
<td>0.0011</td>
<td>1.108</td>
<td>9.205</td>
<td>22.888</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.0028</td>
<td>28.449</td>
<td>257.096</td>
<td>799.483</td>
</tr>
<tr>
<td>CV</td>
<td>-0.855</td>
<td>1.057</td>
<td>1.239</td>
<td>1.823</td>
</tr>
<tr>
<td>Median</td>
<td>-0.0579</td>
<td>15.301</td>
<td>116.807</td>
<td>160.625</td>
</tr>
<tr>
<td>Sample size</td>
<td>741</td>
<td>780</td>
<td>780</td>
<td>780</td>
</tr>
</tbody>
</table>

The distributions of expenditures are skewed towards low values corresponding to GNIc distributions among the global countries. Note also the large standard deviation (and CV) values showing large heterogeneity, especially in ΔlnLE and expenditure observations.

Similar remarks are valid for the infant mortality growth clusters, but now we also observe that the levels of HE across the clusters are not as large as in the above life expectancy clusters. Particularly, the level of HE_{PRIV} does not vary across the ΔlnIM clusters and levels of IM. Thus, infant mortality seems to be independent of HE_{PRIV}. However, the level of IM clearly determines the speed of its decline (i.e. the lower the level of infant mortality rate is, the larger the decrease is). Note also that IM distributions are skewed towards low values.

Table 5 provides summary statistics for GNI per capita level groups. The most interesting result is that the rate of decrease of infant mortality (ΔlnIM) is almost the same in both GNIc groups, albeit there is a huge difference between the levels of infant mortality (IM). The difference between life expectancy (LE) is 12 years, but in poor countries the growth rate of life expectancy is almost two times greater than in non-poor countries. However, a huge difference remains between the country groups in levels of HE.
Generally, these findings with respect to our dynamic panel data models mean that we do not expect much success in infant mortality modelling, as the variable seems to be insensitive to the level of private expenditure. However, the large within heterogeneity in clusters and income groups masked by the above location statistics need a country-level fixed effect (FE) modelling approach that can provide some valuable results across the cluster countries.

3.5. Dynamic panel data models

Consider the following dynamic fixed (FE) or random (RE) effect model (for more details, see Pesaran 2015, chapters 26–27):

$$y_t = \alpha_i + \lambda y_{i,t-1} + \beta' x_t + \mu_{it}, \quad i = 1, 2, \ldots, N \quad \text{and} \quad t = 1, 2, \ldots, T.$$ 

Typically, regressors, $x_t$, are assumed to be strictly exogenous (i.e. $E[\mu_{it} | x_t] = 0$) for all $i$ and $t$. However, the assumption of strict exogeneity is not valid by construction for lagged dependent variable $y_{i,t-1}$, since even if we assume that $E[\mu_{it} \alpha_i] = E[\mu_{it} y_{i,t}] = 0$, the FE/RE demeaning term $E[\mu_{it} y_{i,t-1}] \neq 0$ will not vanish for short panels. In the process without regressors $x_t$, this will cause bias for the FE or RE estimators of $\lambda$ with its size depending on the true value of $|\lambda| < 1$ and the length of panels (Nickell 1981; Pesaran 2015, p. 679)

$$\lim_{N \to \infty} (\hat{\lambda}_{FE/RE} - \lambda) = -\frac{(1 + \lambda)}{T} + O(T^{-1}).$$
The bias is order of $1/T$ and vanishes when $T \to \infty$. For example, when $\lambda$ is close to 1 (the non-stationary case) and $T = 20$, the bias is close to $-0.1$. Note that if regressors $x_{it}$ are included in the model, the size of bias for $\lambda$ and $\beta$ depends on the correlation between $y_{it-1}$ and $x_{it}$. If regressors $x_{it}$ are only weakly exogenous (i.e. allowing for feedbacks from $\mu_{it-1}$) or if they are endogenous variables, the $FE/RE$ bias for $\beta$ is still present, even if no lagged dependent variable is found in the model.

The generic problems of the above dynamic panel model can be seen when we solve for $y_{it}$ recursively from the initial values of $y_{i0}$

$$y_{it} = \lambda^t y_{i0} + \sum_{j=0}^{t-1} \lambda^j \beta' x_{it-j} + \frac{1-\lambda^t}{1-\lambda} \alpha_i + \sum_{j=0}^{t-1} \lambda^j \mu_{it-j}, \quad t = 1, 2, ..., T.$$ 

When $\lambda$ is close to one, initial values $y_{i0}$ and unit-specific effects $\alpha_i$ have large and permanent effects on the $y_{it}$ observations determining the properties of dynamic panel data model estimators. As the process for $y_{it-1}$ has a similar presentation, we obtain, abstracting from terms for regressors and errors:

$$y_{it} = y_{it-1} + (\lambda^t - \lambda^{t-1}) y_{i0} + \lambda^{t-1} \alpha_i.$$ 

This shows that initial effects, but not necessarily the unit-specific $\alpha_i$ effects, have a small role in determining the one-period differenced values of $y_{it}$. Subsequently, the following difference model has also been popular to eliminate the unit-specific effects on $\lambda$ and $\beta$ estimates:

$$\Delta y_{it} = \lambda \Delta y_{it-1} + \beta' \Delta x_{it} + \Delta \mu_{it}.$$ 

However, this will not solve the (OLS) estimation problems for the model parameters, since

$$E[\Delta y_{it-1} \Delta \mu_s] = E[\lambda \Delta \mu_{it-1} \Delta \mu_s] \neq 0.$$ 

Because of $E[\Delta \mu_{it-1} \Delta \mu_s] = \begin{cases} 2\sigma^2, & \text{for } s = 0 \\ -\sigma^2, & \text{for } s = 1 \\ 0, & \text{for } s > 1 \end{cases}$
we need at least two-period lagged values of $y_{t-j}$ and $\Delta y_{t-j} (j \geq 2)$ that do not correlate with $\Delta \mu_t$ (but correlate with $\Delta y_{t-j}$). We can use them as instruments for $\Delta y_{t-1}$ as long as $\lambda < 1$, but as $\lambda \to 1$, we face the weak instrument problem for $y_{t-2}$ because $E[y_{t-2}, \Delta y_{t-1}]$ depends on the size of $\lambda$ (for more details, see Pesaran 2015, p. 682).

The short panel bias of FE/RE and the efficiency problem of the IV approach for the first difference model started the search for IV/GMM-type estimators, leading to consistent and more efficient estimators like GMM estimators by Arellano and Bond (1991), Ahn and Schmidt (1995), and Blundell and Bond (1998). These surprisingly popular methods are extremely complex estimators, which are unbiased and efficient only when no residual serial correlation is found, the dynamic lag order of the model is correctly specified, we have strictly exogenous regressors, no correlation is found between explanatory variables and unit-specific effects $\epsilon_t$, errors are homoscedastic, the sample length is small (i.e. $T/n \to 0$ convergence), low autocorrelation is present in endogenous series, and the problem of weak or too many instruments is not present (see e.g. Dang et al. 2015; Gouriéroux et al. 2010; Hahn et al. 2007; Kiviet et al. 2017).

Empirical drawbacks of the IV/GMM agenda have led to a large group of alternative estimators that have tried in several different ways to correct for $1/T$ time series bias. Chudik and Pesaran (2015) divide this literature into the following broad categories: (i) analytical corrections based on an asymptotic bias formula (Bruno 2005; Bun 2003; Bun and Carree 2005, 2006; Bun and Kiviet 2003; Hahn and Kuersteiner 2002; Hahn and Moon 2006; Kiviet 1995, 1999), (ii) bootstrap and simulation-based bias corrections (Everaert and Ponzi 2007; Phillips and Sul 2003, 2007), and (iii) other methods, including jackknife bias corrections (Dhaene and Jochmans 2012) and the recursive mean adjustment correction procedures (So and Shin 1999). In addition, some methods on long differences have been proposed (Hahn et al. 2007; Han and Phillips 2013; Han et al. 2014) on forward filtering (Keane and Runkle 1992; Keane and Neal 2016; Pesaran 2015, chapter 27.2) and the transformed likelihood method (Hayakawa and Pesaran 2015; Pesaran 2015, chapter 27.6).

In the following, we take methods that are planned to be robust enough against near unit-root case and avoid strict exogeneity assumption. This means that we use a long difference IV
method, LDIV, proposed by Hahn et al. (2007) as well as the Keane–Runkle estimator (1992), which allows for predetermined variables as instruments.

The LDIV technique uses long differencing, i.e. \( \Delta_k y_i = y_i - y_{i-k} \) with \( k = 2, 3, 4, \ldots \), instead of first differencing and iterated two-stage least square (2SLS) in estimating persistent dynamic models with a short time dimension. The LDIV estimator uses lagged levels of the regressors (including \( y_{i,t-4} \)) and the residuals as instruments. The setup for the model is (Hahn et al. 2007, pp. 586–587; Huang and Ritter 2009, p. 269):

\[
5) \quad \Delta_k y_i = \lambda \Delta_{k-1} y_i + \dot{\beta}' x_i + \Delta_k \mu_i
\]

where we can use \( y_{i,t-4}, x_{i,t-4} \) (if strictly exogenous or predetermined) as instrument variables. After obtaining 2SLS estimates for model 5) we calculate the residuals

\[
y_{i,t-1} - \hat{\lambda} y_{i,t-2} - \hat{\beta}' x_{i,t-1}, \ldots, \quad y_{i,t-4} - \hat{\lambda} y_{i,t-5} - \hat{\beta}' x_{i,t-4}.
\]

Next we use these as additional instrumental variables with \( y_{i,t-4}, x_{i,t-4} \) to estimate 5) once again. This is the first iteration. Next new 2SLS estimations are then further iterated via the new results. Typically, less than five iterations are sufficient for convergence.

The Keane–Runkle (1992) estimator (KRPRE) uses the idea of forward filtering or decomposition from the time-series literature to improve the efficiency of the estimates when the error contains some form of serial correlation. Under Cholesky transformation, the orthogonality conditions implied by predetermination are maintained (Keane and Neal 2016). In practice, a key feature of the approach is to use only one or two lags of the predetermined variables as instruments rather than all available lags back to the first period like in complex GMM estimation. Keane and Runkle assume that \( x_i \) are predetermined, in the sense that \( E[x_i, \mu_i] = 0 \), for \( t \geq s \). This is a natural approach in this context where public and private HE in current and previous periods drive life expectancy but not necessarily vice versa. However, life expectancy targets or its unobserved determinants in coming periods \( t + i (i = 1, 2, \ldots) \) will affect public and private HE in the future (i.e. \( E[x_i, \mu_i] \neq 0 \), for \( r > t \)). Note that in the first difference model \( x_i \)
is correlated with $\mu_{t-1}$ because $x_n$ is predetermined but not strictly exogenous. However, $y_{i,t-2}$ and $x_{i,t-1}$ are now valid instruments.

In the Keane–Runkle method, the model 2) has a general covariance specification for $v_i = \alpha + \mu_i$. That is, $E[v'v] = I_N \otimes \Sigma$, where $v$ is a stacked $NT \times 1$ vector of $v_i = (v_{i1}, v_{i2}, \ldots, v_{iT})'$ and $\Sigma = E[v_i v_i']$. To implement the $KPR$ estimator, we need an estimate for $\Sigma$. It is obtained from consistent preliminary $2SLS/IV$ estimation of model 2) using the instruments $Z$ to obtain the $2SLS/IV$ residuals $\hat{v}_i$ and $\hat{\Sigma} = \frac{1}{N} \sum_{i=1}^{N} \hat{v}_i \hat{v}_i'$. Note that a similar two-step procedure can be applied also for difference model 4).

4. Estimation results

4.1. Life expectancy model

Tables 6 and 7 provide the dynamic panel data model estimation results for $\ln LE$ based on standard $FE$, weighted $FEW/TR$ with trend, $LDIV$, and $KPR$ estimation methods. These methods have validity in this context, since in preliminary data analysis the panel data non-stationarity tests (not reported) supported the stationarity alternative. The $FE$ methods assume that all explanatory variables are strictly exogenous, but in $LDIV$ and $KPR$ this erroneous assumption is noticed (i.e. lagged $\ln LE$ is not exogenous), and we need $IV$ methods to overcome this problem. In addition, in $KPR$ we use a less restricted approach on variables $\ln LE-1$, $\ln HEPRIV$, and $\ln HEPUB$ when we assume that they are predetermined with periods $t \geq s$. Thus, the $FE$ approach is expected to be biased in short panels with $1/T$ rate. However, using a sample length of $T = 19$, its role is less important than the evident sample heterogeneity and trending behavior of life expectancy panels. To obtain greater robustness in estimation, we use trend variable and cross-section weights in $FEW/TR$ estimation (i.e. we estimate the model with a feasible GLS specification, assuming the presence of cross-section heteroscedasticity). This has a large effect on the point estimates of model parameters, but their qualitative effects remain the same as in standard $FE$ estimation. In the high life expectancy growth rate cluster (cluster 1), $HEPRIV$ and $HEPUB$ have imprecise, statistically non-significant effects on life expectancy, but $HEPUB$ and primary education rate ($\ln PCR$) predict it with correct signs at 10% significance level. In the low life expectancy growth rate cluster (cluster 2), private expenditures also have some significance along with R&D expenditures. Note that education effects on life expectancy are generally larger
than HE or R&D effects. Note that DW values refer to a Durbin-Watson type test statistics calculated on the stacked set of pooled model residuals. Thus the reported DW values are indicative, i.e. when the values are close to 2 we take it as a sign of appropriate model lag choice.

Table 6. InLE models with ΔlnLE clusters (p-values in parentheses)

<table>
<thead>
<tr>
<th>Cluster 1</th>
<th>N = 42, T = 19</th>
<th>FE1</th>
<th>FE1W/TR</th>
<th>LD1-3</th>
<th>KRPRE4</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>1.499 (0.053)</td>
<td>0.539 (0.001)</td>
<td>-</td>
<td>2.300 (0.000)</td>
<td></td>
</tr>
<tr>
<td>InLE</td>
<td>0.597 (0.004)</td>
<td>0.862 (0.000)</td>
<td>0.597 (0.029)</td>
<td>0.194 (0.001)</td>
<td></td>
</tr>
<tr>
<td>lnHE_priv</td>
<td>0.086 (0.283)</td>
<td>-0.021 (0.248)</td>
<td>0.053 (0.477)</td>
<td>-0.049 (0.012)</td>
<td></td>
</tr>
<tr>
<td>lnHE_pub</td>
<td>0.005 (0.030)</td>
<td>0.001 (0.073)</td>
<td>0.005 (0.260)</td>
<td>0.061 (0.000)</td>
<td></td>
</tr>
<tr>
<td>lnPCR</td>
<td>0.028 (0.094)</td>
<td>0.005 (0.001)</td>
<td>0.021 (0.205)</td>
<td>0.062 (0.001)</td>
<td></td>
</tr>
<tr>
<td>lnRDE</td>
<td>0.009 (0.077)</td>
<td>0.0005 (0.194)</td>
<td>0.011 (0.147)</td>
<td>-0.002 (0.867)</td>
<td></td>
</tr>
<tr>
<td>DW value</td>
<td>2.44</td>
<td>2.36</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cluster 2</th>
<th>N = 153, T = 19</th>
<th>FE1</th>
<th>FE1W/TR</th>
<th>LD1-3</th>
<th>KRPRE4</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>1.089 (0.024)</td>
<td>0.512 (0.000)</td>
<td>-</td>
<td>3.381 (0.000)</td>
<td></td>
</tr>
<tr>
<td>lnLE</td>
<td>0.727 (0.000)</td>
<td>0.975 (0.000)</td>
<td>0.709 (0.001)</td>
<td>0.122 (0.036)</td>
<td></td>
</tr>
<tr>
<td>lnHE_priv</td>
<td>0.002 (0.111)</td>
<td>0.0007 (0.103)</td>
<td>0.0024 (0.002)</td>
<td>0.012 (0.000)</td>
<td></td>
</tr>
<tr>
<td>lnHE_pub</td>
<td>0.0004 (0.005)</td>
<td>0.0004 (0.002)</td>
<td>0.0025 (0.002)</td>
<td>0.009 (0.001)</td>
<td></td>
</tr>
<tr>
<td>lnPCR</td>
<td>0.008 (0.002)</td>
<td>0.003 (0.158)</td>
<td>0.005 (0.000)</td>
<td>0.052 (0.000)</td>
<td></td>
</tr>
<tr>
<td>lnRDE</td>
<td>0.002 (0.007)</td>
<td>0.0004 (0.132)</td>
<td>0.018 (0.029)</td>
<td>0.004 (0.029)</td>
<td></td>
</tr>
<tr>
<td>DW value</td>
<td>1.93</td>
<td>1.73</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

1) SEs calculated with White’s cross-section method
2) SEs adjusted for cross-section clusters
3) Instruments: lnLE(-6), lnHE_priv(-5), lnHE_pub(-5), lnPCR(-5), lnRDE(-5), res(-1 to -4)
4) Instruments: ΔlnLE(-1), ΔlnHE_priv(-1 to -2), ΔlnHE_pub(-1 to -2), lnPCR, lnRDE, constant
5) Instruments: ΔlnLE(-1), lnHE_priv(-1 to -2), lnHE_pub(-1 to -2), lnPCR, lnRDE, constant

Results with the LDIV method are not promising. A search over a suitable difference length provided a five-period difference approach. Models with a trend variable were also considered, but they provided non-significant and partly wrongly signed estimates. Results with the KRPRE method were quite different from the above-mentioned ones. This method seems to scale down the adjustment coefficient but provides larger point estimates for other model parameters. Note that lnHEPRIV gets a negative coefficient estimate for cluster 1. SEs of KRPRE estimates are not corrected for possible heteroscedasticity. Also, an instrument validity test is needed for LDIV and KRPRE estimations before their full validity can be evaluated. We do not report any model diagnostic values like $R^2$ or F-test and residual diagnostics except DW values.
for FE models, as the different model estimators are based on different methods and provide statistics that are not comparable.

Table 7 provides the lnLE model estimates in GNIPc-level groups. Results with FE methods are comparable with the results above. HE_PUB effects are positive and significant on life expectancy, but the size of the effects is less than for primary education. Surprisingly, the education effects are non-significant for non-poor countries. Results with the LD IV method are close to standard FE results, but non-significant HE effects are obtained. KR PRE estimation results are once again in their own category in income level group 1. This depends partly on the demeaning of variables that provided the best results. All but adjustment coefficients are significant, and the rest are comparable with FE and LD coefficients when these are solved for long-run presentation. However, for income group 2, although estimated with demeaned data, results are quite different. In general, these results — and many others not represented here — show that the KR PRE method has some stability and robustness problems.

Table 7. lnLE models with GNIPc groups (p-values in parentheses)

<table>
<thead>
<tr>
<th>Group 1</th>
<th>N = 77, T = 19</th>
<th>FE1</th>
<th>FE1WTR</th>
<th>LDIV2,3</th>
<th>KRPRE5</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td></td>
<td>1.309 (0.022)</td>
<td>0.503 (0.000)</td>
<td>-</td>
<td>0.013 (0.122)</td>
</tr>
<tr>
<td>lnLE</td>
<td></td>
<td>0.855 (0.000)</td>
<td>0.872 (0.000)</td>
<td>0.617 (0.001)</td>
<td>0.025 (0.532)</td>
</tr>
<tr>
<td>lnHE_priv</td>
<td></td>
<td>0.004 (0.029)</td>
<td>-0.0001 (0.177)</td>
<td>0.003 (0.212)</td>
<td>0.043 (0.000)</td>
</tr>
<tr>
<td>lnHE_pub</td>
<td></td>
<td>0.004 (0.014)</td>
<td>0.001 (0.009)</td>
<td>0.004 (0.200)</td>
<td>0.014 (0.065)</td>
</tr>
<tr>
<td>lnHE</td>
<td></td>
<td>0.023 (0.019)</td>
<td>0.006 (0.000)</td>
<td>0.021 (0.060)</td>
<td>0.104 (0.001)</td>
</tr>
<tr>
<td>lnRDE</td>
<td></td>
<td>0.006 (0.028)</td>
<td>0.001 (0.000)</td>
<td>0.006 (0.068)</td>
<td>0.012 (0.044)</td>
</tr>
<tr>
<td>DW value</td>
<td></td>
<td>2.42</td>
<td>1.84</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 2</th>
<th>N = 118, T = 19</th>
<th>FE1</th>
<th>FE1WTR</th>
<th>LDIV2,4</th>
<th>KRPRE6</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td></td>
<td>0.796 (0.000)</td>
<td>0.426 (0.000)</td>
<td>-</td>
<td>-0.021 (0.322)</td>
</tr>
<tr>
<td>lnLE</td>
<td></td>
<td>0.806 (0.000)</td>
<td>0.899 (0.000)</td>
<td>0.761 (0.000)</td>
<td>0.746 (0.000)</td>
</tr>
<tr>
<td>lnHE_priv</td>
<td></td>
<td>0.001 (0.152)</td>
<td>0.0005 (0.004)</td>
<td>0.021 (0.351)</td>
<td>0.012 (0.033)</td>
</tr>
<tr>
<td>lnHE_pub</td>
<td></td>
<td>0.005 (0.01)</td>
<td>0.0006 (0.003)</td>
<td>0.005 (0.026)</td>
<td>0.063 (0.001)</td>
</tr>
<tr>
<td>lnHE</td>
<td></td>
<td>0.000 (0.970)</td>
<td>0.000 (0.671)</td>
<td>0.002 (0.897)</td>
<td>0.026 (0.152)</td>
</tr>
<tr>
<td>lnRDE</td>
<td></td>
<td>0.002 (0.000)</td>
<td>0.0003 (0.001)</td>
<td>0.0014 (0.001)</td>
<td>-0.024 (0.069)</td>
</tr>
<tr>
<td>DW value</td>
<td></td>
<td>1.84</td>
<td>1.69</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

1) SEs calculated with White’s cross-section method
2) SEs adjusted for cross-section clusters
3) Instruments: lnLE(-6), lnHE_priv(-5), lnHE_pub(-5), lnPCR(-5), lnRDE(-5), res(-1 to -4)
4) Instruments: lnLE(-16), lnHE_priv(-15), lnHE_pub(-15), lnPCR(-15), lnRDE(-15), res(-1 to -14)
5) Instruments: ΔlnLE(-1), ΔlnHE_priv(-1 to -2), ΔlnHE_pub(-1 to -2), lnPCR, lnRDE, constant
6) Instruments: ΔlnLE(-1), ΔlnHE_priv(-1), ΔlnHE_pub(-1), lnPCR, lnRDE, constant
4.2. Infant mortality model

These problems are clearly evident for infant mortality model estimation, as the country-level time series have $AR(2)$ presentation more likely than $AR(1)$ models. Although the $KR_{PRE}$ method needs consistent $IV$ estimation in the first stage, which runs easily into problems when the sum of the adjustment coefficient is close to one, the second-stage estimation will not correct these but also runs into problems. For these reasons, we did not provide results on $lnIM$ models with $ΔlnIM$ clusters and take a critical stance on $KR_{PRE}$ estimation in $ΔlnIM$ clusters.

Table 8 provides $FE$ and $LDIV$ results for $lnIM$ models with $ΔlnIM$ clusters. We observe that $LDIV$ based on a long difference works with $AR(1)$ presentation. Estimation results show that increases in $HE_{PUB}$ reduce infant mortality, but the $FS$ has an infant-reducing effect only in cluster 3, where the decline in infant mortality is largest. Surprisingly $HE_{PRIV}$ has an increasing infant mortality effect in cluster 3. In other clusters, the effects are imprecise, except in cluster 1 where the negative effects are statistically significant.

Table 8. $lnIM$ models with $ΔlnIM$ clusters ($p$-values in parentheses)

<table>
<thead>
<tr>
<th>Cluster 1</th>
<th>FE$^1$</th>
<th>FE$^*_{WTR}$</th>
<th>LDIV$^2,3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 56, T = 18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$C$</td>
<td>-0.106 (0.475)</td>
<td>0.060 (0.047)</td>
<td>0.977 (0.001)</td>
</tr>
<tr>
<td>$lnIM_{-1}$</td>
<td>1.501 (0.000)</td>
<td>1.598 (0.000)</td>
<td>0.977 (0.001)</td>
</tr>
<tr>
<td>$lnIM_{-2}$</td>
<td>-0.542 (0.000)</td>
<td>-0.739 (0.000)</td>
<td>-</td>
</tr>
<tr>
<td>$lnHE_{PRIV}$</td>
<td>-0.003 (0.002)</td>
<td>-0.0005 (0.254)</td>
<td>0.002 (0.301)</td>
</tr>
<tr>
<td>$lnHE_{PUB}$</td>
<td>-0.005 (0.000)</td>
<td>-0.0007 (0.017)</td>
<td>0.004 (0.018)</td>
</tr>
<tr>
<td>$lnPCR$</td>
<td>-0.006 (0.094)</td>
<td>-0.0007 (0.336)</td>
<td>0.004 (0.567)</td>
</tr>
<tr>
<td>$lnFS$</td>
<td>0.036 (0.032)</td>
<td>0.011 (0.003)</td>
<td>0.046 (0.026)</td>
</tr>
<tr>
<td>DW value</td>
<td>2.35</td>
<td>2.44</td>
<td>-</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cluster 2</th>
<th>FE$^1$</th>
<th>FE$^*_{WTR}$</th>
<th>LDIV$^2,4$</th>
</tr>
</thead>
<tbody>
<tr>
<td>N = 100, T = 18</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$C$</td>
<td>0.210 (0.021)</td>
<td>0.126 (0.000)</td>
<td>-</td>
</tr>
<tr>
<td>$lnIM_{-1}$</td>
<td>1.079 (0.000)</td>
<td>1.491 (0.000)</td>
<td>0.975 (0.001)</td>
</tr>
<tr>
<td>$lnIM_{-2}$</td>
<td>-0.119 (0.492)</td>
<td>-0.532 (0.000)</td>
<td>-</td>
</tr>
<tr>
<td>$lnHE_{PRIV}$</td>
<td>-0.003 (0.292)</td>
<td>-0.0008 (0.101)</td>
<td>-0.004 (0.345)</td>
</tr>
<tr>
<td>$lnHE_{PUB}$</td>
<td>-0.006 (0.001)</td>
<td>-0.0007 (0.046)</td>
<td>-0.004 (0.291)</td>
</tr>
<tr>
<td>$lnPCR$</td>
<td>-0.020 (0.005)</td>
<td>-0.004 (0.00)</td>
<td>-0.018 (0.031)</td>
</tr>
<tr>
<td>$lnFS$</td>
<td>0.0009 (0.941)</td>
<td>0.002 (0.313)</td>
<td>0.003 (0.108)</td>
</tr>
<tr>
<td>DW value</td>
<td>2.20</td>
<td>2.47</td>
<td>-</td>
</tr>
</tbody>
</table>
Table 9 provides results on lnIM models with GNIc groups and also includes some KRPRE estimation results. Generally, results in Table 9 are not favourable for HE variables. Public expenditures reduce infant mortality in non-poor countries, but similar effects are not found for private expenditures in either income group. Education effects reducing infant mortality are clearly present in Table 9 compared to Table 8. FS effects are mortality-reducing in poor countries.

Generally, we observe that different methods provide quite varying results on point estimates, but clear qualitative accordance is found in the signs of coefficient estimates across the estimation methods. The LDIV method provides results that are comparable to FE results, but KRPRE produced results that are less clear. Nevertheless, we calculate long-run estimates or elasticities for HE variables from estimation results at 10% or below the significance level in the Table 9. lnIM models with GNIc groups (p-values in parentheses)
above tables. Note that we did not calculate $SE$s of the long-run estimates. That can be done with a delta method. Table 10 provides the sum-up. It shows that across the different data configurations and model estimations, 22 $HE_{PUB}$ coefficient estimates from 36 possible ones were significant and their mean of long-run elasticities is 0.0364 for life expectancy and -0.223 for infant mortality. Thus, a 10% increase in public expenditures will increase life expectancy by 0.36% and reduce infant mortality by 2.31%. For private expenditures, we find 0.23% and -0.83% mean elasticities for a much smaller number of 10%-level significant estimates.

Table 10. Long-run elasticities

<table>
<thead>
<tr>
<th>Variables</th>
<th>Number of significant estimates</th>
<th>LR-elasticity estimate range</th>
<th>Mean LR-elasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>$lnHE_{priv} \rightarrow lnLE$</td>
<td>7</td>
<td>[-0.0607, 0.0560]</td>
<td>0.0229</td>
</tr>
<tr>
<td>$lnHE_{pub} \rightarrow lnLE$</td>
<td>11</td>
<td>[0.0074, 0.248]</td>
<td>0.0364</td>
</tr>
<tr>
<td>$lnHE_{priv} \rightarrow lnIM$</td>
<td>5</td>
<td>[-1.333, 0.50]</td>
<td>-0.0833</td>
</tr>
<tr>
<td>$lnHE_{pub} \rightarrow lnIM$</td>
<td>11</td>
<td>[-0.6435, 0.0756]</td>
<td>-0.2231</td>
</tr>
</tbody>
</table>

These estimates and related ones in the literature imply that $HE_{PUB}$ are more effective to improve health status than private expenditures for a large number of countries. However, we cannot say that this is also true for the poorest countries in our sample (see Table 6: cluster 1, and Tables 7 and 9: $GNI_c$ group 1), since we have too few estimates to prove this. Some results with a larger data set (34 OECD countries in the years 1970–2012) and different methods have shown that public expenditure effects in non-poor countries dominate the private ones (see e.g. Linden...
and Ray 2017). At the moment, partly because first-class data on life expectancy and infant mortality from the poorest countries are not available, the question with poor countries is not fully answerable.

5. Discussion and conclusions

The HE effects on life expectancy at birth and infant mortality were analysed with dynamic panel data models for 195 countries in the years 1995–2014. The target questions of ‘Can public and private HE explain the health status variables like life expectancy at birth and infant mortality across different countries?’ and ‘Do increased public and private expenditures in non-high-income countries bring more improvements in health status compared to high-income countries?’ were analyzed with country clusters sensitive to life expectancy growth rates and rates of decrease in infant mortality, and with low and high GNI per capita levels.

The first question gets an affirmative answer, and obtained results show that HEPUB are generally more health-promoting than private expenditures. However, either of the HE effects is not as large as primary education effects. We were not able to give a positive answer to the second question. In country clusters and groups identifying poor countries, positive public expenditure effects are found but private expenditure effects are either non-significant or of comparable size to public effects. This outcome is partly sensitive to the estimation methods used. The new dynamic panel model estimators introduced in this context, LDIV and KRPRE estimators, are not sufficiently robust to provide an answer to the second question. We can observe like Kiviet et al. (2017, pp. 46-48) on GMM estimators that ‘However, not too much is known yet about the actual accuracy in practical situations on the abundance of different not always asymptotically equivalent implementations of estimators and test procedures’, and “Our results demonstrate that, especially under particular unfavorable settings, there is great urge for developing more refined inference procedures for structural dynamic panel data models’ to also be valid on LDIV and KRPRE estimators used here. For the LDIV, we need some guidance on how to determine the long difference length. KRPRE needs some more robust developments under error heterogeneity and longer AR settings than AR(1).

The assumptions of strictly exogenous variables and predetermination made in the analysis are not harmless. Both the LDIV and KRPRE methods allow for settings where HE are endogenous (i.e. life expectancy and infant mortality determine the levels of private and public expenditures).
This is not only the starting value or initial-level problem, but reasonable arguments can support
the idea that the current levels of health status also determine $HE$. Typically, these arguments
rest on the population’s age structure and on the ‘healthier are wealthier’ type of argument where
a specific distinction is not made between private and public expenditures. On the contrary, our
approach stressed the fact the $HE_{PUB}$ is a policy-driven variable in the short run (i.e. some sort of
exogeneity is a natural starting point in analysis). Some model estimations were conducted with
endogenous $HE$, but they did not provide any results beyond what we have presented already.
However, this important question must be addressed in detail in future research.

From a health policy perspective, the obtained results are interesting. In many country types
analysed here there is still room for health improvements with larger $HE$. Globally, private
expenditures seem less health-productive than $HE_{PUB}$. Thus, the policy option would be a shift of
resources from the private to the public sector. However, in practice this would be difficult by
harming the consumer’s sovereignty with taxes and regulation. Also, first we must know in detail
to what extent private and public health care services are complements or substitutes. Note here
that all three components of care — price, volume, and quality — have their impact on both the
care provisions and their utilization. Typically, at least in non-developed countries, the price and
quality components dominate in the private sector, as much of care provision here is based on
imported medical skills and goods. As in many developed countries, care differentiation refers to
the sector financing but not to the provision of health care, and the distinction between the two is
not evident. In our data, high-income countries’ private and public expenditures correlate
positively with each other much more strongly than in low-income countries (0.707 and 0.277,
respectively). As the expenditure leakage is smaller in poor countries, correctly targeted $HE_{PUB}$
and their marginal increases matter greatly in low-income countries. Thus, a general policy
option is to subsidise the use of health care services so that the poorest can have access to and
resources for them.

**Acknowledgements**

We thank two anonymous referees for helpful comments.
Bibliography


ARTICLE III
Errata

Page 85, paragraph 3: “HCT” and not “HTC”.

Pages 92-93, equations (1) to (4): lower case “t” and not upper case “T”:

\[ y_{i,t} = \exp(a_{i,0} + b_1 t + b_2 t^2), \text{ with } t = 1, 2, \ldots, T \]  
(1)

\[ \ln y_{i,t} = a_{i,0} + b_1 t + b_2 t^2 \]  
(2)

\[ \ln y_{i,t} = a_{i,0} + b_1 t + b_2 t^2 + \varepsilon_{i,t} \]  
(3)

\[ \ln y_{i,t} = a_{i,0} + b_1 t + \varepsilon_{i,t} \text{, and} \]  
\[ \Delta \ln y_{i,t} = b_{11} + b_{12} t + \varepsilon_{i,t}^* \]  
(4)

Page 94: no “ln” in left side denominator of equation (6):

\[ \ln(y_{i,t}/y_{i,t-1}) = a + \beta \ln y_{i,t-1} + \mu_{i,t} \]  
(6)

Page 95:

\[ \sigma_i^2 \approx (1 + \beta)^2 \sigma_{t-1}^2 + \sigma_u^2 \]

Page 96: lower case “t” and not upper case “T”:

\[ A_1: \quad \ln y_{i,t} = a_{i,0} + b_1 t + \varepsilon_{i,t} \]

\[ A_2: \quad \Delta \ln y_{i,t} = b_{11} + b_{12} t + \varepsilon_{i,t}^* \]

Page 97, last paragraph: “income economies” and not “in economies”.

Page 102, last paragraph: “Tables 2 and 3” instead of “Tables 3 and 4”; “models A, B, C and D” and not “models B, C, and D”; “heroscedasticity consistent White standard errors” instead of “White-diagonal variance”.

Page 104, 2nd last line: “method of estimating the conditional mean response of a variable in the sample” instead of “method of regression mean in the sample”.

Page 107: lower case “t” and not upper case “T”:

\[ A_1: \quad \ln y_{i,t} = a_{i,0} + b_1 t + \varepsilon_{i,t} \]

\[ A_2: \quad \Delta \ln y_{i,t} = b_{11} + b_{12} t + \varepsilon_{i,t}^* \]
HEALTH CARE TECHNOLOGIES AND GLOBAL CONVERGENCE OF TB AND CANCER MORTALITY RATES

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University of Eastern Finland

ABSTRACT
Analysis of cancer and TB mortality rates with 144 and 196 countries respectively for 1970 – 2012 is done. To get more reliable picture how these rates are affected by health care technologies, health care resources and relevant socio-economic variables are added to the analysis. Methods of trend growth and convergence analysis, found in economic growth empirics, are used to analyze the effects of global catch-up of health care technologies through diffusion between more and less advanced countries. The results show that there is evidence of larger declining trend growth process in low income countries for both illnesses when compared to higher income countries. However, the speed of declining mortality rate processes has been slowing in high income countries in recent decades. Both σ- and β-convergence is found to be present for TB. Conditional β-convergence in TB is larger when HCT and socio-economic factors are added to the test models. For cancer mortality, no clear evidence of σ-convergence is found. However, when technologies and socio-economic factors are added to the β-convergence model, the convergence rates are the largest in lower income countries for both illness. Contrary to this, in 1995 – 2012, β-convergence of cancer with technologies and socio-economic variables disappear.

Keywords: sigma convergence, beta convergence, trend growth, health care technology diffusion
Cancers figure among the leading causes of morbidity and mortality worldwide, with approximately 14 million new cases and 8.2 million cancer related deaths in 2012 (WHO, 2015l). The number of new cases is expected to rise by about 70% over the next 2 decades. Around one third of cancer deaths are due to high body mass index, low fruit and vegetable intake, lack of physical activity, tobacco and alcohol use. Tobacco use is the most important risk factor for cancer causing around 20% of global cancer deaths and around 70% of global lung cancer deaths (De Martel et al., 2012). Infection with HIV substantially increases the risk of cancer such as cervical cancer. Cancer can be reduced and controlled by implementing evidence based strategies for cancer prevention, early detection of cancer and management of patients with cancer (WHO, 2015l). At every stage, e.g., early diagnosis, screening, treatment and palliative care health care technologies (HCT) are used.

Tuberculosis is caused by the bacteria Mycobacterium tuberculosis and is second only to HIV/AIDS as the greatest killer worldwide due to a single infectious agent. In 2013, 9 million people fell ill with TB and 1.5 million died from the disease (WHO, 2015a). Globally in 2013, an estimate of 480,000 people developed multidrug resistant TB (MDR-TB) (WHO, 2015a). BCG vaccine continues to be inadequate but certainly a big step towards prevention of the illness (Zwerling et al., 2011). Between 2000 and 2013, an estimated 37 million lives were saved through TB diagnosis and treatment with the help of HCTs (WHO, 2015a). Radio therapy equipments, MRI and CT scanners all help in diagnosis, treatment and cure as in the case of cancer.

The development and possible convergence of rate of bad health outcomes (like cancer and tuberculosis mortality) across different countries over time seems to be neglected topic in health economics. If the spread of
mortality rates across the countries decreases in time, then some convergence happens between the countries. Likewise, if the absolute growth rates of declining mortality rates are larger in poor countries compared to rich ones, then the levels of mortality rates in poor countries will eventually reach the levels of rich countries. We argue that convergence is most likely an indication of global diffusion and efficient use of HCTs between the countries. In this sense different metrics of convergence are important in showing the long run trends in disease mortality rates between the countries.

This paper gives implicit results on HS convergence as affected by usages of HCT globally and attempts to validate the global catch up hypothesis caused by HCT diffusion. The target is test for convergence and to show impacts of HCT on cancer and TB mortality in different GNI per capita level countries. We defend the idea that the poorest countries can quite quickly adopt improvements in their HS if they can afford and get access to technological advances found in rich countries. The social inequalities and the low level of health expenditures in non-rich countries however slower this important and urgently needed catch-up. To get more reliable picture how cancer and TB mortality rates are affected by HCT, other health care resources and relevant socio-economic variables like total health expenditure per capita, GNI per capita, proportion of population using improved sanitation facilities-total, adult alcohol consumption, and share of regular daily smokers in the population are added to in to analysis.

The paper is organized as follows. The next section elucidates on existing research. The section thereafter gives the data, models, and the methods used, followed by the section that reports the results. The last section concludes the paper with a discussion.
EARLIER LITERATURE

Technology And Health

Technology is a crucial ingredient of health care. BHJ defines technology as any intervention which influences health and society (Berger, 1999). In response two trends are observed during the latter half of the twentieth century. First, there is a rising trend in health expenditure. Second, patients survive globally major illnesses like cancer and TB to live long. The combination of technological improvements in medical treatment and rising incomes is the driving force behind these two trends.

Technological innovation has yielded remarkable advances in health care. Breakthroughs in a variety of areas have improved health care delivery and patient outcomes. However, the proliferation of HCT and its expanding uses have contributed to increasing health care costs and the former has often been cited as a culprit for the latter. However, this relationship is variable, complex and evolving (Newhouse, 1992). Creation and development of new technologies are driven by both demand and supply side forces.

There are many benefits of HCT innovations. The most important is the value of better health, i.e. longer life as well as improved quality of life after early and/or proper diagnosis of illnesses like TB and cancer. A second benefit of HCT innovation is its effect on income formation. One part of this benefit is an increase in production that results from technology allowing people to work and earn more. Offsetting this productivity benefit are the medical and nonmedical costs of additional years of life. The net value of medical technology change is the difference between the benefits and costs (Cutler & McClellan, 2001). A positive net value implies that the technological change is worth it in total.
A variety of factors may influence health over time, of which medical technology is only one. It is widely accepted that technological change has accounted for the bulk of medical care cost increases over time (Cutler & McClellan, 2001). Technological change is bad only if the cost increases are greater than the benefits that technological change brings about. Cutler et al. (2001) reported on a series of studies that examined the costs and benefits of medical technology changes. These studies showed that medical spending as a whole was worth the increased cost of care. HCT change affects treatments in two ways, namely, treatment substitution and treatment expansion. HCT could be expensive, but it still could be worth paying for, if it extended the length or quality of life or otherwise resulted in positive social returns. So, understanding cost effectiveness is more important than understanding costs alone.

The paper by Sorenson, Drummond and Khan (2013) critically appraises this conjecture the existing literature with the aim of offering a more detailed analysis of the relationship between HCT diffusion and health expenditure. Selected 86 articles are reviewed and relevant information is extracted into a standardized template and analyzed for key cross-cutting themes, e.g., impact of technology on costs, factors influencing this relationship and methodological challenges in measuring such linkages etc. Based on their analysis, they argue that attention also needs to be focused on exploring whether investments in medical technology result in better value as measured by therapeutic benefits, cost effectiveness, and other important health outcomes and under which conditions technologies allow for the most effective and efficient use of available health care resources.

If the new HCT supplements the existing instrumentation and its purpose is to expand the treatment into the conditions that have not been treated previously
due to scientific (as the methods of treatment were unknown) or economic (as the methods of treatment were known, but enormous costs made it unfeasible on a larger scale) reasons, one can say that it could have a cost increasing effect (expansion effect). On the other hand, extra savings may be expected if a decrease in the relative price of a given type of treatment (due to e.g. the introduction of a new technology) reduces the use of other, more expensive substitute types of care (substitution effect).

Some technologies may improve the efficiency of care delivery by reducing procedure time, length of stay or number of hospitalizations, thereby increasing the capacity of the hospital to treat additional patients. Overall costs may rise as a result, but such outlays will likely result in improved health outcomes for a greater number of patients. Technological advancements may generate consumer demand for care (and, perhaps more intense, costly services, even if not cost-effective), and demand for insurance (Sorenson et al. 2013).

Past literature reviews imply that HCT diffusion is an important contributor to improved health status as measured by life expectancy and mortality rates. Preston (1996) and Soares (2005) argued that increases in life expectancy have occurred independently of increases in per capita income. While not denying the importance of other factors, Soares (2005) placed emphasis on changes in mortality determined from technological innovations in medical and biological sciences. Both authors give evidence that the positive cross-sectional relationship between life expectancy and per capita income had shifted upward steadily over time. Kremer (2002) emphasized the importance of modern medical technologies in allowing tremendous improvements in health even at low income levels. Jamison, Sandbu and Wang (2001) documented the importance of different rates of technological progress
across countries for the declining cross-country variation in infant mortality rates. Becker, Philipson and Soares (2005) argued that in the last 50 years, countries starting with modest longevity levels experienced life expectancy gains significantly larger than countries starting with high longevity thresholds. They attributed the convergence in life expectancy in large part to the diffusion of existing knowledge that had helped reduce mortality from major diseases. Fogel (1994) referred to a potential explanation for the acceleration of life expectancy improvements in the huge social investments made in biomedical research in most developed countries, whose payoffs were not counted in some of them as part of national income in the past, even though they produced a large stream of benefits during in the past decades. Preston (1996) suggested also that mortality changes in developing countries came about through the provision of public programs and the dissemination of knowledge.

Baltagi, Moscone and Tosetti (2011) modeled differences across OECD countries in health productivity as a function of traditional factor inputs, life styles conditions and technological progress. The authors first explored available data on medical technology to explain health productivity in the OECD countries. Baltagi et al. (2011) assumed that technology was unobserved and hence used proxy for it by means of a spatial process. Baltagi et al. (2011) like Ertur and Koch (2007) allowed technological progress in a country to be related to the technology adopted by neighboring countries. That technology could show a geographical pattern had earlier studied by Keller (2004). In the medical literature, a consolidated body of research supported long-ago the important role of interpersonal communication and social networks in the diffusion of medical technologies (Coleman, Katz & Menzel, 1966). Again, Baltagi et al. (2011) like Birke (2009) chose a survey on the role of
social networks in explaining individual choices in a large variety of economic, social and health behavior. Papageorgiou, Savvides and Zachariadis (2007) studied the impact of a set of measures of international medical technology diffusion on health status and concluded that technology diffusion is an important determinant of improved health status and mortality rates. Their data was on 63 countries over the period 1961 to 1995.

**Technology Diffusion And Health Convergence**

Literature shows that medical innovation diffuses steadily across the world contributing to significant improvements in life expectancy. International medical diffusion occurs through two distinct channels. Firstly, imports of medical goods, such as drugs, vaccines and medical equipment. Papageorgiou et al. (2007), Caselli and Wilson (2004), and Eaton and Kortum (2001) consider that production of goods embodying medical technology is concentrated in a small number of R&D intensive countries while the rest of the world typically imports these goods. Secondly, through the direct flow of medical knowledge from a few frontier countries to the rest of the world, a flow that is facilitated by information networks created by medical students from non-frontier countries who study in frontier ones (Papageorgiou et al., 2007).

Above remarks mean that HTC is a major factor in explaining the differences in countries HS and in possible HS convergence between them. The theoretical basis of economic convergence is derived from the neoclassical growth model which gives the result that in the long run all countries move towards a common steady state level of income per capita because of decreasing marginal product of capital, i.e. the steady state is obtained under certain conditions. Thus, convergence is closely connected to the long term (economic) growth. In case of long run mortality rates, one would expect convergence of the HSs due to the
existence of upper bounds of many health indicators as well as due to diminishing returns of inputs, e.g. health expenditures, efforts in education, economic development (Gächter & Theurl, 2011). A number of methods are proposed to study the question of convergence.

In $\sigma$ – convergence cross sectional standard deviation or coefficient of variation (or some other measure of variability) of a variable across a group of homogenous countries decreases over time. As standard deviation is a measure of the spread of data and is defined as a numerical measure of the average variability of around the mean. If its value diminishes with successive measures over time, this will support the hypothesis of convergence (Nixon, 2000). In order to statistically test for $\sigma$ - convergence it is necessary e.g. to analyze the trend of standard deviation values in the data over successive points of time.

The disadvantage of $\sigma$ - convergence is that it may be disproportionately influenced by discontinuities, outliers and short run shocks. Additionally, the existence of a diminution of standard deviation or coefficient of variation does not confirm that a country approaching the sample mean will remain there. The second approach limits these shortcomings, positing that convergence exists if a poor economy tends to grow at a faster (but diminishing) rate than a rich one. Now the poor country tends to catch up the rich country. This property corresponds to the concept known as $\beta$ - convergence or regression to the mean (Barro and Sala-i-Martin, 1992a; Sala-i-Martin, 1996 & Boyle & McCarthy, 1997). Within this method of analysis two forms of convergence are distinguished, namely, unconditional or absolute and conditional rates of growth.

By convergence literature, economists typically refer to the large literature, typified by the seminal papers by Baumol (1986), Barro and Sala-i-Martin (1992b) and Mankiw, Romer and Weil (1992), exploring $\beta$ - convergence. Sala-i-Martin (1996) surveying this literature,
concluded that the estimated speeds of $\beta$-convergence are so surprisingly similar across cross-sectional data sets, that one can say that economies close the gap between present levels of income and balanced growth levels by, on average, 2% annually. Past panel data studies find even higher rates of $\beta$-convergence (Evans, 1997), as do the county-level U.S. studies of Higgins, Levy and Young (2006) and Young, Higgins and Levy (2013). Despite the literature’s stress on $\beta$ - convergence, economists have acknowledged that it is not a sufficient condition for $\sigma$-convergence (Barro & Sala-i-Martin, 1992b). Quah (1993) and Friedman (1992) both suggested that $\sigma$-convergence should be of greater interest because it speaks directly as to whether the distribution of income across economies was becoming more equitable.

The underlying principles of the neoclassical growth model have applicability to the study of convergence in health care expenditure as there is a strong correlation between the latter and GDP income (Nixon, 2000). His findings provide statistically significant evidence for $\beta$-convergence in GDP and this is a driver of the results of health expenditure convergence. The results of Nixon (2000) show that statistically significant $\sigma$-convergence in health care expenditure outcomes occurred in the present countries of the EU over the period 1960-95 and in 1980-95 for $\beta$ - convergence. The analyses reveal a common trend in that Southern European OECD countries which have generally exhibited convergence towards the mean in health expenditure and convergence towards the EU mean in for health outcomes.

Previous studies before Clark (2011) generally concluded that a nation’s level of economic development and /or its growth rate were positively associated with improvements in human welfare, particularly among less developed countries. However, other works suggested that economic growth might not have played a large role in
mortality improvements during the twentieth century. Research showed that economic growth produced only modest welfare benefits in the developing world. Indeed, even when economic development was found to significantly improve a country’s infant survival rate, child survival rate or life expectancy among developing countries, it often produced effects that were smaller than other predictors, such as education or gender-related measures.

In his study, Clark offered new evidence regarding (a) inequality trends in life expectancy and infant mortality, as well as (b) the role that economic development may have played in producing these trends (Clark, 2011). He first examined whether life expectancy averages and infant mortality rates converged across 195 countries during the 1955 - 2005 period. Consistent with prior work, he found that cross-national inequality in life expectancy declined during the 1955 - 2005 period, but that this convergence stalled during the post- 1990 era. Moreover, and contrary to previous work, he found that infant mortality rates diverged continuously across the sample period. He then developed a narrative to explain these contrasting trends, suggesting that cross-national health outcomes followed a welfare Kuznets curve. He also estimated the differential impact of economic development on life expectancy and infant mortality. According to this model, economic development improved life expectancy more than it reduced infant mortality among poor countries, whereas the situation was reversed among wealthier nations. In sum, Clark (2011) argued that economic development had contributed to both convergence in life expectancy and divergence in infant mortality.

Moser, Shkolnikov and Leon (2005) investigated to what extent worldwide improvements in mortality over the past 50 years have been accompanied by convergence in the mortality experience of the world's population. The
global mortality distribution at a point in time was quantified using a dispersion measure of mortality (DMM). Trends in the DMM indicated global mortality convergence and divergence. Their analysis used United Nations data for 1950-2000 for all 152 countries with populations of at least 1 million in 2000 (99.7% of the world's population in 2000). DMM for life expectancy at birth declined until the late 1980s but increased since then, showing a shift from global convergence to divergence. In contrast, the DMM for infant mortality indicates continued to converge since 1950. They concluded that the shift from global convergence to divergence was being driven by reversals in adult mortality. With respect to the former Soviet Union, including the Russian Federation, there was strong evidence that the reversals in life expectancy at birth were almost exclusively due to increases in adult mortality. So, based on the research by Moser et al. (2005) one could say that although in one sense the world became a better place as mortality declined, in another way it became worse as the distribution of life expectancy at birth worldwide started to diverge.

Health is an important dimension of welfare comparisons across individuals, regions and states. Particularly from a long-term perspective, within country convergence of the health status has not been that often investigated. Gächter and Theurl (2011) in their research studied the relation between initial levels of the health status and its improvement at the local community level in Austria in the time period 1969-2004. Method wise they used age standardized mortality rates from 2381 Austrian communities as an indicator for the health status and analyzed the convergence/divergence of overall mortality for (i) the whole population, (ii) females, (iii) males and (iv) the gender mortality gap. Convergence and divergence was studied by applying different concepts of cross regional inequality, namely, weighted standard deviation, coefficient of variation and Theil coefficient of inequality.
The researchers used weighted OLS, quantile regression and Kendall’s rank concordance to test for absolute and conditional \( \beta \) - convergence in mortality. They found mixed results with reference to \( \sigma \) - convergence, while the weighted standard deviation indicated an increase in equality for all four variables, the picture appeared less clear when correcting for the decreasing mean in the distribution.

However, they found highly significant coefficients for absolute and conditional \( \beta \) -convergence between the periods. While these results were confirmed by several robustness tests, they also found evidence for the existence of convergence clubs. In order to test for differences in the \( \beta \) - coefficients within the distribution the authors also ran quantile regressions for the lower and upper quartile of the distribution. Once again, the impression of divergences in the \( \beta \) - coefficients in different parts of the distribution was confirmed, albeit the conclusion of \( \beta \) - convergence across communities is unaffected by this result.

Goli and Arokiasamy (2014) did testing of the convergence hypothesis for trends in maternal and child mortality indicators during 1990 to 2008 by using three different types of convergence metrics. They found discrepancies in the progress achieved in terms of Millennium Development Goals (MDG) namely, for MDG 4 (reduce child mortality) and MDG 5 (improve maternal health). Graphical assessment indicated clear evidence of catching-up process for all the maternal and child mortality indicators, but the \( \beta \) - convergence model estimates showed lack of convergence. The results of the absolute \( \beta \) - convergence estimates suggested a divergence in the progress of the Maternal Mortality Ratio (MMR) across the countries for the entire period and convergence for the recent period. The progress in all child mortality indicators was \( \beta \) - divergent. Such divergence increased subsequently.
The past and current research on mortality and birth rates, and on life expectancy have shown that convergence is a vital and important concept in analysis of population health across the countries in log run (Gächter et al., 2011 for additional references). The concepts of $\sigma$ - and $\beta$ - convergence play here an important role and they give the unifying standard also for the global mortality rate analysis.

**DATA, MODELS AND METHODS**

*Data*

Panel data for countries are obtained for 43 years (1970-2012). The first study variable is TB mortality/100,000 persons including HIV positive cases ($TBM_{kt}$) for 196 countries. The second is cancer mortality/100000 persons ($CM_{kt}$) and includes summation of deaths caused by 24 types of cancer in 144 different countries. Health technology variables (HCT’s) include radio-therapy equipment, mammography machines, magnetic resonance imaging (MRI) units, computed tomography (CT) scanners, all in terms of per 1000,000 inhabitants, BCG vaccines in terms of % of live births who received it, doctors working in any medical field (including oncologists) per 1000 people, and hospital beds/1000 patients besides health expenditure variables for OECD and other countries in per capita and in % of GNI per capita terms. The socioeconomic variables included in study are kilocalories per person in-take per day, alcohol consumption in liters of pure alcohol per person per year, % of regular daily smokers in the population, % of population using improved drinking water source, and proportion of population using improved sanitation facilities.

The differentiation of the countries into 4 groups was done by their income levels using the World Bank’s Atlas method (using current US $). Group 1 (low income
economies) consist of countries which have a GNI per capita of $1045 or less. 30 countries fall into this category. Group 2 (lower middle-income economies, 49 countries) refers to GNI per capita of $1046 - $4125. The third category or group 3 (upper middle-income economies) has 55 countries with a GNI per capita of $4126 - $12735. Group 4 (high income economies, 62 countries) has a GNI per capita in the range of $12736. The data sources are given in details in Appendix A). Note that we analyze periods 1970 – 2012 and 1995 – 2012 separately because in years 1970 -1995 the mortality rates in many countries were not measured with high precision.

Models and Methods

Trend growth models. Elementary growth analysis is built on trend model presentation like
\[ y_i = a \exp(b_1 T + b_2 T^2) \]
where \( a \) refers to the starting value of process and coefficients \( b_1 \) and \( b_2 \) measure the growth rate and the acceleration of growth process. When analyzing the time evolution of TB and cancer mortality rates we expect growth rates to be negative with enforcing acceleration, i.e. \( b_1 < 0 \) and \( b_2 < 0 \), if mortality rates are decreasing effectively. Taking logarithms of 1) gives a linear model
\[ \ln y_i = a_0 + b_1 T + b_2 T^2 \]
(2)
that is easily estimated with OLS-methods. However, when the model is cast in the panel data framework like cross-section fixed effect (FE) model
\[ \ln y_{i,t} = a_{i,0} + b_1 T + b_2 T^2 + \epsilon_{i,t} \]
(3)
some estimation and inference challenges have to be solved. First, mortality rates are expected to be smooth and slowly evolving series making the error term in model 3 to
be highly auto correlated. Second, mortality rates are very country specific (i.e. clustered) even within same income level groups. Finally, the trend variables $T$ and $T^2$ are highly collinear. All these symptoms make the statistical inference to be non-standard. We do not try to handle the first problem because we need to use quite complicated bootstrap methods to derive sample valid standard errors (Vogelsang, 1998 & Linden, 2002). Note, however, that FE-OLS coefficients are still consistent and unbiased. The second problem can be corrected with weighed estimation by using cross-section weights. Finally, the last problem can be solved by estimating following two models:

$$\ln y_{it} = a_{i0} + b_1 T + \varepsilon_{it}$$

and

$$\Delta \ln y_{it} = b_{i1} + b_2 T + \varepsilon^*_{it}$$

(4)

where $b^*_2 = 2 \times b_2$ and $\varepsilon^*_{it} = \Delta \varepsilon_{it}$. Note that we allow in the second equation for country specific accelerations to be present in model. This model has standard properties as $\varepsilon^*_{it}$ are expected to be not auto correlated. However, both heteroskedastic and clustered errors are still present. Hence the model is estimated with country specific weights and with White’s HSCE –corrections.

The outlined model alternatives help us to analyze if the growth process between the different income level country groups are different. For example, if estimates for $b_1$ and $b_2$ are larger in absolute terms for low-income countries than for high-income countries we have evidence of global catch-up of mortality rates.

**σ – and β convergence.** Following Sala-i-Martin’s (1996) example, Young, Higgins and Levy (2008) assume that β-convergence holds for a group of homogeneous economies. Thus, the path natural log-income of the $i$th economy can be approximated by
\( lny_{it} = a + (1 + \beta)lny_{it-1} + u_{it} \)  
(5)
where \(-1 < \beta < 0\). The error term \(u_{it}\) is independent over \(t\) and \(i\), and has mean zero and finite variance \(\sigma_u^2\). Because \(a\) is assumed to be constant across economies, steady state growth rates are identical, but economies can have different growth paths, depending on their initial states, to it. This is the case of unconditional or absolute \(\beta\)-convergence, i.e. average growth rates of poor economies are unambiguously greater than those of rich economies because of higher marginal product of capital or because of global catch-up effects. Allowing for heterogeneity across the economies \((a_i \neq a_j)\), \(-1 < \beta < 0\) would imply the case of conditional or relative \(\beta\)-convergence. The average growth rate of an economy is an increasing function of its distance from its own steady state growth level of income. This is a weaker case of \(\beta\)-convergence and increases the set of possible scenarios where it does not imply \(\sigma\)-convergence.

To analyze the relation between \(\sigma\)- and \(\beta\)-convergences, by arranging the above equation (5) one gets

\[
l n(\frac{y_{it}}{lny_{it-1}}) = a + \beta lny_{it-1} + u_{it}
\]
(6)

Thus, \(\beta < 0\) implies a negative correlation between income growth and initial log income. The sample variance of log income in \(t\) is given by

\[
\sigma_t^2 = \frac{1}{N} \sum_{i=1}^{N} (lny_{it} - \mu_t)^2
\]
where $\mu_t$ is the sample mean of (log) income at time $t$. The sample variance is close to the population variance when $N$ is large, and equation (5) can be used to derive the evolution of $\sigma_t^2$:

$$\sigma_t^2 = (1 + \beta)^2 \sigma_{t-1}^2 + \sigma_u^2$$

Only if $-1 < \beta < 0$ is this difference equation stable, so $\beta$-convergence is necessary for $\sigma$-convergence. If $\beta \geq 0$, the variance increases over time. If $\beta = 0$, the variance is constant (full convergence), and if $\beta < -1$, log of income would oscillate potentially from positive to negative values and back (making little economic sense).

Moreover, as $(1 + \beta) < 1$, the approach to stable $\sigma^2$ value is monotonic. Economies can be $\beta$-converging toward one another while, at the same time, random shocks are pushing them apart. Despite $\beta$-convergence, if the initial dispersion of income levels is, by chance, small relative to the variance of random shocks then the dispersion of incomes will converge toward its steady-state value from below. Note in equation (6) above parameter $\beta$ governs the speed at which the variance approaches its steady-state value because, according to the equation (5), it governs how long the effect of shocks persist.

Conditional $\beta$-convergence does not imply $\sigma$-convergence arise. In empirical applications the country specific factors ($a_i$'s) are often modeled as linear functions of various economic and/or socio-demographic variables. Intuitively, consider two economies, A and B, where both economies begin at the same level of income. However, assume that B begins on its balanced growth path while A begins far below its balanced growth path, and assume that $\beta$-convergence holds. The initial variance $\sigma_0^2$ will be zero, but $\sigma_t^2$ will grow over time as A grows faster than B and approaches a higher balanced growth path. Indeed, $\beta$-convergence is the reason for the increasing variance. In
real economies, $\sigma$-convergence would also depend on whether or not disturbances are correlated and have constant variances across time and economies.

Next, we first analyze convergence with trend models and then with $\sigma$- and unconditional $\beta$-convergence models for our sample countries for TB and cancer mortality in year 1970 - 2012. We calculate estimates for different income group countries (i.e. the convergence clubs). To get a detailed picture of global differences in TB and cancer mortality we use also the distribution sensitive estimation approach, i.e. the quantile regression. Different quantile process coefficients related to the levels of cancer and TB mortality will be analyzed. After this we turn more specifically on conditional $\beta$-convergence allowing initial levels of variables of health technology (HTC) and socio-economic variables to affect the convergence.

RESULTS

TB mortality

**Trend estimates.** Table 1 depicts the results of models

\begin{align*}
A_1 : & \quad \ln y_{i,t} = a_{i,0} + b_T + \varepsilon_{i,t} \\
A_2 : & \quad \Delta \ln y_{i,t} = b_{1,1} + b_T + \varepsilon_{i,t}^*
\end{align*}

We observe (see Table 1) that for low and lower middle-income countries the trend growth estimates are largest (in absolute terms). We report results with countries on periods 1970 – 2012 and 1995 – 2012 separately because in years 1970 – 1995 the TB mortality rates in many countries were not measured with high precision. The estimates are 1.5-2.0 times larger than for high-income countries. For TB in Table 1 the acceleration estimates
(\(b_i\)'s) are negative for low, lower middle and upper middle-income countries showing that speed of decreasing TB-mortality rates is increasing. Contrary to this in high income countries we find halting-up. Note that \(t\)-values for level models are only indicative.

### Table 1.

<table>
<thead>
<tr>
<th>MODEL</th>
<th>Low income (N = 90)</th>
<th>Lower middle income (N = 49)</th>
<th>Upper middle income (N = 55)</th>
<th>High income (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(A_{1,1970-2012}: b_1)</td>
<td>(-0.030) ((-47.87))</td>
<td>(-0.026) ((-66.27))</td>
<td>(-0.019) ((-66.72))</td>
<td>(-0.020) ((-93.83))</td>
</tr>
<tr>
<td>(A_{2,1970-2012}: b_2)</td>
<td>(-0.00065) ((-5.39))</td>
<td>(-0.00041) ((-5.67))</td>
<td>(-0.00009) ((-1.21))</td>
<td>(0.0001) ((1.42))</td>
</tr>
<tr>
<td>(A_{1,1995-2012}: b_1)</td>
<td>(-0.042) ((-35.70))</td>
<td>(-0.033) ((-53.55))</td>
<td>(-0.027) ((-42.19))</td>
<td>(-0.022) ((-41.28))</td>
</tr>
<tr>
<td>(A_{2,1995-2012}: b_2)</td>
<td>(-0.0042) ((-15.30))</td>
<td>(-0.0029) ((-12.93))</td>
<td>(-0.00007) ((-0.45))</td>
<td>(0.00056) ((2.49))</td>
</tr>
</tbody>
</table>

\(\sigma\) –convergence. In the framework of \(\sigma\) – convergence the Figure 1 below shows a very clear convergence from 1970’s to early 1990’s in the upper and lower middle in economies for TB mortality. Till about the 1990s all the four country groups have a downward slope in yearly cross-section SD’s showing a \(\sigma\) – converging trend. Thereafter they diverged but new convergence paths were found next at the end of century. Subsequently, especially the low and the high-income countries had a downward slope in \(\sigma\) – convergence trend.
The upper middle-income countries register higher SD values than the other groupings (Figure 2). These are a disparate group including former Warsaw Pact countries. Since becoming independent many of these countries have undergone various economic transformation and subsidies in the health sector have been eliminated or reduced. Further HCT is not always readily available as in high income countries due to lower total health expenditure per capita and institutional bottlenecks (Rechel et al., 2014). R&D expenditure is also less than in high income countries. The outcome of all these are revealed in high incidence of TB mortality. This group also has other countries following the Soviet style economic policies. Since the breakdown of former Soviet Union many have been embroiled in problems in the health sector as one witnessed in former Warsaw pact countries.

The average of TB mortality is seen to be going down for all income group over the years (Figure 3) suggesting the number of cases per year is going down too in all the 196 countries (Appendix B gives the growth rates between periods 2012 and 1970 and 2012 and 1995 for different income groups). The best scenario is seen in the high-income countries and comparatively the worst one is observed in the low-income economies. There is a tendency for intersection for the low income and the lower middle economies in the years after 2012 suggesting possible convergence if the low-income countries continue their catching up with lower middle-income countries with regards to TB mortality convergence (see Figures 1 and 2).
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β – convergence: cross section approach. We derive the unconditional β - convergence results with cross section by estimating the following model with OLS- and Quantile estimation methods

$$ln\left(\frac{y_{i,t}}{y_{i,t_0}}\right) = \alpha + \beta ln y_{i,t_0} + u_i.$$  
(A)

Now β < 0 implies a negative correlation between log of mortality growth and initial log of mortality rate. It should be noted that this so-called Barro regression is a difference equation that converges to stable solution when - 1 < β < 0 and the convergence is faster when β is closer to -1.

Next, we specify conditional β – convergence with technology variables, i.e. the initial level t₀ (year 1970 or 1995) HTC variables that may have their own impact on the change rate on TB and cancer mortality rates. Thus, we
allow HTC –variables condition the initial level convergence, i.e. the speed of convergence measured with $\beta$ parameter

$$\ln\left(\frac{y_{i,t}}{y_{i,t_0}}\right) = \alpha + \beta \ln y_{i,t_0} + \nu' X_{i,t_0}^{TECH} + u_i.$$  

(B)

For period $t_0$ the technological variables $X_{i,t_0}^{TECH}$ include:

- $HCTC1_{kt}$: radiotherapy equipment per 1000,000 inhabitants
- $HCTC2_{kt}$: total mammography machines - total in hospitals and in ambulatory care providers per 1000,000 inhabitants
- $HCTC3_{kt}$: magnetic resonance imaging (MRI) units per 1000,000 inhabitants and includes MRI units in hospitals and ambulatory care providers,
- $HCTC4_{kt}$: computed tomography (CT) scanners, total per 1000,000 inhabitants (includes hospitals and ambulatory care providers),
- $I_{kt}$: BCG vaccine in terms of % of live births,
- $DOC_{kt}$: doctors working in any medical field/000 people, and
- $HOSB_{kt}$: hospital beds/1000 patients,

Contrary to this model we assume next that the socio-economic factors condition the convergence,

$$\ln\left(\frac{y_{i,t}}{y_{i,t_0}}\right) = \alpha + \beta \ln y_{i,t_0} + \delta' X_{i,t_0}^{SOC} + u_i.$$  

(C)

For period $t_0$ the technological variables $X_{i,t_0}^{SOC}$ include:

- $THEXPC_{kt}$: total health expenditure per capita (PPP, constant 2011 international $)$,
$GNIPC_{ki}$: GNI per capita calculated using the World Bank Atlas method (current US $),
$FS_{ki}$: food supply: kilocalories per person per day,
$AW_{ki}$: % of population using improved drinking water source,
$ISA_{ki}$: proportion of population using improved sanitation facilities-total,
$AC_{ki}$: alcohol consumption/adult (15+) in liters of pure alcohol per person per year and,
$TP_{ki}$: % of regular daily smokers in the population 15 + years.

Finally, we combine both the technical and the socioeconomic indicators in one model:

$$ln\left(\frac{y_{i,t}}{y_{i,t_0}}\right) = \alpha + \beta ln y_{i,t_0} + \psi' X_{i,t_0}^{TECH} + \delta' X_{i,t_0}^{SOC} + u_t$$

(D) All the cross-section OLS regressions $t$-values were estimated with White–diagonal variance corrections because residual heteroscedasticity and non-normality were found in the residuals diagnostics. The final estimation equations contained only variables having 5% level significant coefficient values and variables causing multicollinearity were excluded. We don’t report the estimated values of $\psi$ and $\delta$ because our focus is on the convergence parameter $\beta$ and how it is affected by the presence of control variables. The detailed estimation results are provided by request from the authors. Thus Tables 3 and 4 (below) reports the estimation results only on the speed of convergence (the size of $\beta$) from models B, C, and D. Interestingly, $\beta$–convergence increases as we add new variables in to model. This is true to all country groups except for the low and the upper middle-income countries in years 1970 – 2012 and for lower middle-income countries in years 1995 – 2012.
We argue that this “speeding up” phenomena is a result starting values of variables included in $X_{t,0}^{TECH}$ and $X_{t,0}^{SOC}$. More favorable is the initial level of these variable faster is the convergence. We notice that the socioeconomic variables (model C) make a somewhat greater effect to the convergence compared to the technology variables (model B). However, the combined model D) provides the largest speed-ups especially in years 1995-2012 for some country groups. However, these results are less warranted when observe that the quantile estimation does not fully support the OLS –estimation results above. The absolute size of $\beta$ increases also in median estimation (in Tables 3 and 4) with variable addition, but not so much as in OLS estimation.

Table 2.
Cross section estimates of convergence parameter $\beta$ with models’ A - D: 1970 – 2012

<table>
<thead>
<tr>
<th>MODEL</th>
<th>Low income N = 90</th>
<th>Lower middle income N = 49</th>
<th>Upper middle income N = 55</th>
<th>High income N = 62</th>
<th>Total (median regression) N = 196</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>-0.612 (-5.56)</td>
<td>-0.360 (-3.97)</td>
<td>-0.346 (-4.59)</td>
<td>-0.406 (-3.64)</td>
<td>-0.281 (-8.06)</td>
</tr>
<tr>
<td>B</td>
<td>-0.616 (-4.99)</td>
<td>-0.381 (-3.25)</td>
<td>-0.348 (-4.26)</td>
<td>-0.443 (-3.27)</td>
<td>-0.353 (-7.25)</td>
</tr>
<tr>
<td>C</td>
<td>-0.676 (-5.04)</td>
<td>-0.425 (-4.85)</td>
<td>-0.328 (-4.19)</td>
<td>-0.489 (-3.19)</td>
<td>-0.384 (-7.80)</td>
</tr>
<tr>
<td>D</td>
<td>-0.606 (-4.75)</td>
<td>-0.442 (-3.91)</td>
<td>-0.336 (-3.89)</td>
<td>-0.494 (-2.98)</td>
<td>-0.415 (-7.73)</td>
</tr>
</tbody>
</table>
Table 3.
**Cross section estimates of convergence parameter $\beta$ with models: A – D: 1995 – 2012**

<table>
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<th>MODEL</th>
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<tr>
<td>A</td>
<td>-0.263 (-10.54)</td>
<td>-0.154 (-8.83)</td>
<td>-0.057 (-2.73)</td>
<td>-0.147 (-7.73)</td>
<td>-0.069 (-10.92)</td>
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<tr>
<td>B</td>
<td>-0.355 (13.11)</td>
<td>-0.134 (-5.39)</td>
<td>-0.045 (-2.13)</td>
<td>-0.152 (-8.35)</td>
<td>-0.102 (-10.18)</td>
</tr>
<tr>
<td>C</td>
<td>-0.368 (-8.05)</td>
<td>-0.242 (-1.61)</td>
<td>-0.109 (-2.57)</td>
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<tr>
<td>D</td>
<td>-0.658 (-7.17)</td>
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<td>-0.179 (-2.17)</td>
<td>-0.214 (-2.44)</td>
<td>-0.298 (-4.57)</td>
</tr>
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</table>

Figure 4 depicts the quantile process $\beta$-coefficients (i.e. model estimation results at different error quantiles) of model A. The results indicate that it is the countries with largest absolute tuberculosis mortality growth rates (the lowest quantiles - typically the low-income countries, see Table 2: row A, Figure 3 and Appendix B) that have the largest $\beta$-convergence and countries with smallest absolute mortality growth rates have the smallest $\beta$-convergence. The observed OLS model residual heteroskedasticity and non-normality make the quantile estimation results to vary in different error quantiles (Davino, Furno & Vistocco, 2014). As OLS estimation is a method of regression mean in the sample it masks the different distribution features of data.
Table 3. Cross section estimates of convergence parameter $\beta$ with models: A – D: 1995 – 2012

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Figure 4. Unconditional quantile process coefficients ($q = 0.1, 0.2, \ldots, 0.9$) in 1970 – 2012 and 1995 – 2012 respectively with model A)
Figure 5
Unconditional quantile process coefficients \((q = 0.1, 0.2, \ldots, 0.9)\) in 1970–2012 and 1995–2012 respectively with model D

Figure 5. reports the quantile estimates with both control variable sets \(00,,\) and \(TECH\ SOC\) \(XX\). There are not big differences between the quantile process estimates between models A and D. Note that in growth empirics literature the constant term depicts the steady state growth rate. The quantile process estimates of constant term are mostly negative in model A) indicating that the negative growth rates in TB mortality are typical for countries with low level of mortality. For model D the constant term estimates are too imprecise to support this result.

Cancer mortality Trend estimates. Tables 4 depicts the results of models:

\[ \text{ln} y = a + b T + \varepsilon \]

We report results with 144 countries on periods 1970–2012 and 1995–2012 separately because in years 1970–1995 the cancer mortality rates in many countries were not measured with high precision. We observe that for low and lower middle-income countries the trend growth estimates are larger (in absolute terms) than for upper middle income and high-income countries in period 1970–2012. This is not found in period 1995–2012. However, the acceleration estimates \((b)\) are negative only for high income countries only in the period 1970–2012 showing that cancer mortality is decreasing with increasing speed in these countries. Contrary to this one finds non-speeding-up in the other three groupings. A similar scenario is seen in the period 1995–2012.
Figure 5. reports the quantile estimates with both control variable sets \( X_{i,t0}^{TECH} \) and \( X_{i,t0}^{SOC} \). There are not big differences between the quantile process estimates between models A and D. Note that in growth empirics literature the constant term depicts the steady state growth rate. The quantile process estimates of constant term are mostly negative in model A) indicating that the negative growth rates in TB mortality are typical for countries with low level of mortality. For model D the constant term estimates are too imprecise to support this result.

**Cancer mortality**

**Trend estimates.** Tables 4 depicts the results of models:

\[
A_1: \quad \ln y_{i,t} = a_{i,0} + b_1 T + \epsilon_{i,t}
\]

\[
A_2: \quad \Delta \ln y_{i,t} = b_{1,1} + b_2^* T + \epsilon_{i,t}^*
\]

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Table 4

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<th>High income</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A_{1,1970-2012} )</td>
<td>-0.013</td>
<td>-0.011</td>
<td>-0.008</td>
<td>-0.005</td>
</tr>
<tr>
<td></td>
<td>(63.99)</td>
<td>(-78.72)</td>
<td>(-48.71)</td>
<td>(-37.75)</td>
</tr>
<tr>
<td>( b_1 )</td>
<td>0.0003</td>
<td>0.0003</td>
<td>0.0003</td>
<td>-0.0004</td>
</tr>
<tr>
<td></td>
<td>(7.68)</td>
<td>(7.31)</td>
<td>(6.25)</td>
<td>(-12.24)</td>
</tr>
<tr>
<td>( A_{1,1995-2012} )</td>
<td>-0.007</td>
<td>-0.005</td>
<td>-0.003</td>
<td>-0.009</td>
</tr>
<tr>
<td></td>
<td>(-18.39)</td>
<td>(-11.84)</td>
<td>(-8.93)</td>
<td>(-66.39)</td>
</tr>
<tr>
<td>( b_1 )</td>
<td>0.00016</td>
<td>0.00025</td>
<td>0.0002</td>
<td>-0.00006</td>
</tr>
<tr>
<td></td>
<td>(1.17)</td>
<td>(2.13)</td>
<td>(1.48)</td>
<td>(1.89)</td>
</tr>
</tbody>
</table>

\( \sigma \)-convergence. Dispersion of cancer mortality rates for the high income countries are higher than the other income groups since 1970 (Figure 6). This is partly a result of higher average mortality rates in these countries (Figure 8). However when taking the low income countries as the starting point, one sees that there has been \( \sigma \)-convergence only in certain periods. Generally one does not see evidence of general or trending \( \sigma \)-convergence for non-high-income countries. Between 2000-2012 there is a weak \( \sigma \)-convergence in lower income and low income countries (Figure 7). However for the high income countries \( \sigma \)-convergence is evident only after year 1995 albeit the average cancer rates (Figure 8) are also declining for this income group in whole sample (Appendix B for group specific growth rates). The middle income and the lower income categories followed the same trajectory as the lower income group had. We stress the result that contrary to weak \( \sigma \) convergence the average cancer mortality rates declined during all the sample years. The high-income countries have still the higher-than-the-others average
Table 4


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<tbody>
<tr>
<td></td>
<td>N = 28</td>
<td>N = 27</td>
<td>N = 35</td>
<td>N = 54</td>
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</tbody>
</table>

A1, 1970-2012:

| b   | (-63.99) | (-78.72) | (-48.71) | (-37.75) |

A2, 1970-2012:

| b   | (7.68)    | (7.31)    | (6.25)    | (-12.24)  |

A1, 1995-2012:

| b   | (-18.39)  | (-11.84)  | (-8.93)   | (-66.39)  |

A2, 1995-2012:

| b   | (1.17)    | (2.13)    | (1.48)    | (1.89)    |

σ−convergence. Dispersion of cancer mortality rates for the high income countries are higher than the other income groups since 1970 (Figure 6). This is partly a result of higher average mortality rates in these countries (Figure 8). However when taking the low income countries as the starting point, one sees that there has been σ−convergence only in certain periods. Generally one does not see evidence of general or trending σ−convergence for non-high-income countries. Between 2000-2012 there is a weak σ−convergence in lower income and low income countries (Figure 7). However for the high income countries σ−convergence is evident only after year 1995 albeit the average cancer rates (Figure 8) are also declining for this income group in whole sample (Appendix B for group specific growth rates). The middle income and the lower income categories followed the same trajectory as the lower income group had. We stress the result that contrary to weak σ−convergence the average cancer mortality rates declined during all the sample years. The high-income countries have still the higher-than-the-others average cancer mortality rates. After 1995 the high-income level has started to decline faster and in other income groups average rates have haltered.
Figure 8.
*Average Cancer mortality 1970 – 2012*

**Average Cancer mortality / 100,000 for 1970-2012 in 144 countries differentiated by income levels**

---

**β – convergence: cross section approach.** In the cross-section approach, we take the same A – D models and use OLS and quantile methods to derive convergence estimates. In Table 5 the $t$-values show that almost all $\beta$ coefficients are significant at 10% level. Both in case of median regression and high-income category for model A the estimate for $\beta$ is non-significant. For upper lower middle-income countries adding socioeconomic variables have been an obstacle in the speeding up process of convergence. Low income countries have the best convergence situation in model D but some $\beta$ estimates are unstable.
In the cross-section approach, we take the same A–D models and use OLS and quantile methods to derive convergence estimates. In Table 5, the \( t \)-values show that almost all \( \beta \) coefficients are significant at 10% level. Both in case of median regression and high-income category for model A the estimate for \( \beta \) is non-significant. For upper lower middle-income countries adding socioeconomic variables have been an obstacle in the speeding up process of convergence. Low income countries have the best convergence situation in model D but some \( \beta \) estimates are unstable.

In Table 6, with addition of more variables as one moves from models A to D, one sees that the speed of convergence increases (absolute value of \( \beta \)) when OLS for 144 countries for 1995-2012 is taken. However, this does not happen for low and lower middle-income countries with models C and D. Also for high-income countries models A and C show inconclusive results.
Table 6.
Cross section estimates of convergence parameter $\beta$ with models A to D: 1995 – 2012

<table>
<thead>
<tr>
<th>MODEL</th>
<th>Low income N = 28</th>
<th>Lower middle income N = 27</th>
<th>Upper middle income N = 35</th>
<th>High income N = 54</th>
<th>Total (median regression) N = 144</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>-0.352 (-9.05)</td>
<td>-0.473 (-13.19)</td>
<td>-0.253 (-10.54)</td>
<td>0.046 (2.97)</td>
<td>-0.058 (-5.55)</td>
</tr>
<tr>
<td>B</td>
<td>-0.479 (-18.12)</td>
<td>-0.441 (-11.43)</td>
<td>-0.379 (-17.87)</td>
<td>-0.076 (-4.21)</td>
<td>-0.246 (-17.79)</td>
</tr>
<tr>
<td>C</td>
<td>-0.181 (-1.15)</td>
<td>-0.055 (-0.57)</td>
<td>-0.299 (-3.08)</td>
<td>-0.134 (-1.59)</td>
<td>-0.201 (-3.81)</td>
</tr>
<tr>
<td>D</td>
<td>-0.281 (-1.16)</td>
<td>0.130 (1.33)</td>
<td>-0.496 (-3.89)</td>
<td>-0.159 (-1.97)</td>
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Quantile regression results (Figure 9) for model A in years 1970 – 2012 and 1995 – 2012 show only significant convergence for country growth rates in above 0.5 quantiles in years 1995-2012 - typical result for non-high-income countries (see Table 6: row A, Figure 8 and Appendix B). This does not strike against OLS results. However, the results with model D) show almost uniform $\beta$-convergence for all quintiles with significant 95% CI’s.
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Quantile regression results (Figure 9) for model A in years 1970 – 2012 and 1995 – 2012 show only significant convergence for country growth rates in above 0.5 quantiles in years 1995-2012 - typical result for non-high-income countries (see Table 6: row A, Figure 8 and Appendix B). This does not strike against OLS results. However, the results with model D show almost uniform $\beta$-convergence for all quintiles with significant 95% CI's.

Figure 9. Unconditional quantile process coefficients ($q = 0.1, 0.2, ..., 0.9$) in 1970 – 2012 and 1995 – 2012 respectively with model A)
Figure 10. Unconditional quantile process coefficients ($q = 0.1, 0.2, ..., 0.9$) in 1970 – 2012 and 1995 – 2012 respectively with model D)

DISCUSSION AND CONCLUSIONS

For decreasing TB and cancer mortality rates the trend growth estimates are the largest in absolute terms in case of low income and lower middle-income countries. Whereas in TB mortality one sees a speeding up of declining mortality rate for not high-income countries, just the opposite effect is valid for cancer mortality rate, as the speed of decreasing cancer mortality rates is speeding up only in high-income countries.

Whereas one sees a $\sigma$-convergence trends in upper middle and lower middle-income countries till the early 1990s for TB mortality, in case of cancer mortality there is no such convergence. Overall, there is more evidence of $\sigma$-convergence in case of TB mortality than in case of cancer between 1970 – 2012. However, one can argue that average TB and cancer mortality rates are going down for all country groupings in 1970 – 2005 but in the period after it went up to 2012 the declining process has halted especially for cancer in non-high-income countries.

Considering $\beta$-convergence (in terms of absolute values) for both cancer and TB, taking the cross-section growth between 1970–2012 in different income groups, one sees clear convergence with unconditional Barro-regressions for both TB and cancer mortality rates covering the period 1970 – 2012. For condition $\beta$-convergence, when health care technological and socioeconomic variables are taken into consideration, we find that for low income countries, the TB and cancer mortality rates respectively, have the fastest converge. However, when analyzing the period 1995-2012 having less erroneous measurements, TB in low-income countries still show the fastest $\beta$-convergence, but with cancer rates the convergence disappears when additional HCT and socio-economic initial variables are added in test models.
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results with quantile regressions give additional insights to convergence analysis that are not obtained with fixed effects panel regressions. β - convergence is more evident across the quantiles in conditional models.

The results indicate that the cancer mortality rates are very country specific and when conditioning the rates in different income groups the convergence is not present anymore. The global (conditional) convergence still seems to be missing for cancer mortality rates albeit the average rates have declined almost to the current period. Contrary to cancer rate results the TB mortality rate results give implication that a catching-up of declining of TB mortality takes place in poorer countries through diffusion of HCTs from the richer nations. This phenomenon is however not yet clearly observed in cancer mortality rates where diffusion and disease processes are more heterogeneous than with TB.

One can argue that cancer mortality is not responding as well as TB mortality to diffusion of HCTs. Here one seems to forget the fact that prevalence, diagnosis and treatment of TB has been present for a while, while both the diagnosis and treatment of cancer effectively are comparatively a newer phenomenon. Existing therapies like chemo-, surgical-, radio, precision medicine, and (controversial) stem cell transplant, and many newer treatments for cancer (e.g., molecular targeted therapies) are in their pre-clinical test phases. Also, new or rare types of cancer are being diagnosed (e.g. chronic myeloid leukemia). This all happens in large scale only in the richer countries. Note also that R & D in new cancer treatments are more expensive than for new TB treatments like multi-drug resistant ones or that with HIV. Thus, it could take some time for new cancer treatments to diffuse to poorer countries.
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REFERENCES


Appendix A
Data sources

The main TB data source has been WHO (2015b). TB mortality, TB incidence and prevalence data are available here. Individual country data has been collected in this database for many countries from their national databases. The main sources of OECD’s cancer mortality and case statistics have been from International Agency for Research on Cancer (IARC, 2015a), IARC Cancer Mondial (IARC, 2015b), GLOBOCAN (2015) and OECD (2015a). Further data sources have been WHO (2015f) and IARC (2015c). Data for Nordic countries have been taken from NORDCAN (2015). Other data sources have been EUREG (2015), EUCAN (2015), CI5 (2015), OECD (2015c) and SURVCAN (2015). HCT (radiotherapy equipment, MRI units, CT scanners, mammography machines) data for both TB and cancer have been obtained from eclectic databases. The most important one for OECD data has been from OECD (2015b). Global (WHO, 2015c), including not OECD European countries (WHO, 2015d) as well as Europe’s (Eurostat, 2015a) HCT data have been added to OECD data. BCG vaccination data (WHO, 2015e) has also been considered. This database includes WHO’s global summary of vaccine preventable diseases monitoring system. This data has been further supplemented by UNICEF data (UNICEF, 2015a; WHO, 2015m). Health expenditure data has been acquired from WHO (2015k,
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However, data for $TP_{kt}$ was available for 66 countries from 1970 to 2012. These countries cover the OECD countries, G20 countries, Western & Eastern European nations and former Soviet Republics. Data for San Marino and Macedonia were not available for the whole period. Hence, they are left out. Additionally, the data for the variables $THEXPC_{kt}$ and $AW_{kt}$ were available for the period 1995 – 2012 for all the 196 countries. Hence these two variables are added for the said period when doing the regressions for 1995 – 2012. For the socioeconomic variable $THEXPC_{kt}$ data is available for all countries only from 1995 – 2012. Further, data for $AW_{kt}$, which is a socioeconomic variable is available from 1990 onwards. Hence to keep parity with $THEXPC_{kt}$ variable, data from 1995 to 2012 has been taken for $AW_{kt}$. 
## Appendix B
Mean and Standard Deviation for TB and cancer growth rates in 2012-1970 and in 2012-1995

<table>
<thead>
<tr>
<th>Country groups</th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Obs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-1.323292</td>
<td>1.025381</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
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<td>1.004639</td>
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<tr>
<td>3</td>
<td>-0.951953</td>
<td>0.865850</td>
<td>55</td>
</tr>
<tr>
<td>4</td>
<td>-0.881951</td>
<td>0.586311</td>
<td>62</td>
</tr>
<tr>
<td>All</td>
<td>-1.065862</td>
<td>0.868855</td>
<td>196</td>
</tr>
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Figure 1. TB growth rates: 2012/1970

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<td>1</td>
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<td>2</td>
<td>0.511728</td>
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<td>3</td>
<td>0.460702</td>
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<tr>
<td>4</td>
<td>0.384494</td>
<td>0.387470</td>
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<tr>
<td>All</td>
<td>0.490787</td>
<td>0.637743</td>
<td>196</td>
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Figure 2. TB growth rates: 2012/1995

Author's Biography
Devdatta Ray holds a Master of Social Sciences and Master of Arts and is a researcher in the Department of Health and Social Management at the University of Eastern Finland.
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<td>3</td>
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<td>0.183233</td>
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ARTICLE IV
Health, inequality and income: a global study using simultaneous model

Devdatta Ray* and Mikael Linden

1 Background

Today, the health of most people in the world depends on their ability to locally adopt health knowledge and health technologies that have been discovered and developed elsewhere. Life expectancy and infant mortality are major health status determinants that impact economic growth. OECD countries with higher life expectancies and low infant mortality rates have more economic development and higher standards of living than all other countries. However, unfortunately, only in recent times economists have realized that health is an important part of human capital formation sustaining economic growth and improvements in health status can be justified on purely economic grounds. Good health raises levels of human capital and this has a positive effect on individual productivity and human capital returns. Better health increases workforce productivity by reducing incapacity and the number of days lost to sick leave besides increasing the opportunities of obtaining better paid work. Although good health may be considered a form of human capital that has a beneficial effect on productivity, income also influences

Abstract

A simultaneous three-equation model is specified between GDP per capita ($GDP_c$) level, infant mortality rate and health expenditures for 194 countries from 1990 to 2014. GMM-2SLS estimation results indicate that simultaneous decreasing infant mortality rate and increasing $GDP_c$ level effects are found in sample with three income level country groups. Health expenditures have larger than one elasticity when effects from $GDP_c$ level and number of doctors per capita are summed together. Increase in income inequality measured with $GINI$ coefficient increases infant mortality rate in non-poor countries. We test for Kuznets' hypothesis maintaining a positive $GDP_c$ level income inequality relationship in poor countries contrary to rich ones. Kuznets' hypothesis is not rejected for poor countries with proposed $∩$-shaped $GDP_c$ function on $GINI$ that also identifies the negative income inequality effects on $GDP_c$ growth. In poorest countries, the possible Kuznets' hypothesis and involved low-income high-inequality trap can be eliminated by raising health expenditures–$GDP$ ratio and with cost-effective health technology. Breaking the possible negative relationship between income inequality and health status in these countries makes health promotion policy and positive income–health path to develop smoothly.

Keywords: Income–health relationship, Health expenditures, Income inequality, Kuznets' hypothesis

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Keywords: Income–health relationship, Health expenditures, Income inequality, Kuznets’ hypothesis
health in a positive way. The capacity to generate higher earnings facilitates an increase in the consumption of health-related goods such as adequate food, medicine and health care, which provide longer longevity. However, the income effects on health are not equally distributed. Wealthier people can provide higher investments in health capital although the marginal benefits are largest among the poorest. Thus, the simultaneous health–income relationship needs to be analyzed in a framework where income inequality also affects the health outcomes.

In response to this, we specify a simultaneous three-equation model between level GDP per capita \((GDP_{c})\), health status \((HS)\) and health expenditures per capita \((HEc)\) for a data set of 194 countries in years 1990–2014. \(GDP_{c}\) and \(HS\) determinations are also affected by income inequality, i.e., we propose that income inequality effects on \(GDP_{c}\) \((level\ and\ growth)\) and \(HS\) are depending on the income level of country. To obtain a compact approach on health and inequality effects on \(GDP_{c}\), the analysis is cast in the framework of Kuznets’ hypothesis maintaining a positive income inequality relationship for poor countries contrary to the rich ones. We argue that so-called low-income high-inequality trap can be escaped in the presence of Kuznets’ hypothesis by raising the health expenditures–GDP ratio and with cost-effective health technology. The argument is that the poorest countries can quite quickly do this, if improvements in their health status and inequalities create a push effect in the growth direction. This can happen easily when negative relationship between income inequality and health status is not present. However, often inequalities, low levels of health expenditures, high unemployment, low levels of education and labor productivity and detrimental health-related behavior in non-rich countries—all slow down or even hinder this important and urgently needed catch-up. Estimation results show that Kuznets’ hypothesis is still relevant for countries with low \(GDP_{c}\) levels, i.e., low-income high-inequality trap is still present in the poorest countries blocking the catch-up. However, the modern version of hypothesis maintaining that income inequality has negative effect on the growth of \(GDP_{c}\) is not rejected for countries with different levels of \(GDP_{c}\). Generally we show that a positive bidirectional health–income relationship is not rejected and that more equal income distribution decreases infant mortality rate.

The structure of paper is as follows. Sections 2 and 3 focus on the studies on health and inequality effects on economic growth. Section 4 analyzes the Kuznets' hypothesis and gives the model to be estimated. In Sect. 5 the estimation results are given and discussed. Section 6 ends the paper with conclusion. Subsequently, appendices give the study variables with data resources, review of the relevant econometric methods and detailed estimation results.

2 Health and economic growth

In the second part of the twentieth century, child mortality rates and life expectancy improved throughout poor countries. Gwatkin (1980) and Deaton (2013) labeled this as the third of three great waves of mortality decline. Here the first, starting at the end of the nineteenth century, began in North and Western Europe and was then transmitted to North America. The second wave, beginning in the nineteen twenties, was in South and Eastern Europe. The rate of gain in life expectancy was even more rapid than in the first wave. By the middle of the twentieth century, life expectancies in the South
Gains in income were important for improving nutrition and funding along with better water and sanitation schemes. However, some countries made progress in reducing infant mortality even in the absence of such economic growth (Reinhart 1999). This health improvement came from the globalization of health and health care knowledge. More recently, the global infant mortality decline has depended on transmission of both conventional and advanced health care technology. Although they may be expensive, medical techniques diffuse more rapidly than changes in behavior, which respond slowly and unevenly to changes in knowledge about health risks (Aguayo-Rico et al. 2005). However, if one accepts the argument that health is largely determined by the transfer of technology and knowledge, the current state of mortality from any epidemic in any part of the world is evidence of the failure of globalization to transfer effective drug based technology and treatment from the rich countries to the poorer ones (Shastry and Weil 2002).

Partly as response to these new aspects of human capital a new family of theories of economic growth emerged in the 1980s that were better equipped to explain long-term growth (Romer 1986; Lucas 1988). Central to these models was the idea that technology was endogenous to the growth process. In the neoclassical growth models, the notion of growth as increased stocks of capital goods was codified in the Solow–Swan growth model. In contrast to these Lucas (1988) and Romer (1986) considered technology as endogenous and incorporated a new concept of human capital which had increasing rates of return. The focus shifted on increased human capital, learning and level of R&D activity. However, one saw still large variations in health between rich and poor countries contributed to differences in income and vice versa. Better health of workers meant higher productivity and less disparities of income which in their turn meant more spending causing improved health and longevity. So, there was a multiplier effect with better health. Similarly, the effects of exogenous health improvements like a vaccine that made workers healthier lead to a multiplier effect as healthier workers produced more output (Weil 2005, 2009). This “health view” assumed that income differences between the countries were mainly caused by different health environments. The “income view”, on the other hand, assumed that most differences between the countries had their roots in aspects of production that were unrelated to health, e.g., in physical capital accumulation or technology. Weil (2005, 2009) further showed that there was a link between poverty reduction and long-term economic growth that was impacted by health. From the early 1990s, the role of human capital (including health) was almost universally regarded as being indispensable for economic growth. Sustained growth depended on levels of human capital whose stocks increased because of better health and education, effective learning and training procedures. Without a labor force with some minimal levels of education and health, a country was incapable of maintaining a state of continuous growth (Rivera and Currais 2003).

The analysis and comparison of mortality patterns in several economies led Morand (2004) to suggest the theory of epidemiological transition. There were three basic patterns of transition in this theory, namely the classical or western model, the
accelerated model, and the delayed one. These three patterns corresponded to three scenarios of i) endogenous transition during the neoclassical growth regime, ii) endogenous transition during the modern growth regime, and iii) transition triggered by exogenous factors. In an endogenous growth model output was produced by combining physical and human capital inputs, where agents could invest in health and education resulting in improvement of their human assets (Galor 2011; Van Zon and Muysken 2001). This, in turn, affected their lifetime utility positively. So, economic growth had a direct effect on health status and longevity which in turn impacted economic growth.

Empirical growth model estimates have generally showed the importance of health status on growth. A study by Lorentzen et al. (2008) regressed GDPc on the average child and adult mortality rates over the period 1960–2000. The study found a strong effect of mortality rates on income growth. Aghion et al. (2010) analyzed the relationship between health and growth across OECD countries using cross-country panel regressions. They found a significant and positive impact of health on growth and vice versa between 1940 and 1980. This study showed that between 1940 and 2000 average GDPc and average life expectancy among high-income countries achieved larger gains in GDPc but smaller increases in life expectancy than they did in low and middle-income countries. By combining both so-called Lucas and Nelson–Phelps approaches, they could show that achieving higher life expectancy had a positive significant effect on GDPc growth. It improved health standards and increased current productivity growth (“Lucas effect”), while higher health standards improved future productivity growth (“Nelson–Phelps effect”).

Bloom et al. (2004) showed that improvements in health increased output not only through labor productivity, but also through capital accumulation. There was a positive impact of health and health expenditures on income growth. There were four ways by which health impacted economic growth. It enhanced labor productivity, created a greater labor supply, acted as a catalyst for education and training that fostered higher skills, and called for more savings leading to more investment in physical and intellectual capital. Bloom and Canning (2008) further showed that health also affected prospective life spans and life cycle behaviors. To the extent that income was a consequence of health, investments in health were to become a priority. This argument for health as an investment good was particularly relevant since there were cheap and easily implementable health policies that could improve health dramatically.

According to Sachs (2001), rising health status drives economic growth. The lag between declines in mortality and fertility resulted in a baby boom generation that started a period of economic growth as they entered the workforce. Sachs (2001) called this “demographic dividend”. Health, (income) inequality and economic developments were endemically interrelated. Policies with regard to health spending influenced welfare of contemporary and next period’s generations which then influenced economic growth. The long-term demographic and economic data with regard to developed OECD countries showed that increase in general human capital during transition periods influenced the rhythm of economic growth permanently. As per capita income went up, the longevity of population increased. As longevity went up,
savings and investment for education and retirement increased. This was supported by higher rates of private and public investments for health. This leads to capital accumulation, which in turn leads to the economies’ aggregate efficiency and levels of economic activities to go up (see also Casasnovas et al. 2005; Cervellati and Sunde 2009; Aisa and Pueyo 2006).

De la Croix and Licandro (1999) focused on an overlapping generation model with uncertain lifetime and endogenous growth. The model gave the positive effect of life expectancy on growth for economies with a relatively low life expectancy. However, this was negative in more advanced economies. The positive effect of a longer life on growth could be offset by an increase in the average age of the workers. Life expectancy affected growth directly, namely when the probability of dying young was high, the discount rate was also high making it optimal for people to start working early in their life and not to stay at school too long (Fogel 1994). When households had to decide the moment at which they would leave school to work, life expectancy became a central factor that affected the optimal length of education and hence the growth rate of the economy. So, the positive effect of a longer life on growth could be offset by an increase of the average age of the working population.

Bhargava et al. (2001) investigated the effects of health indicators such as adult survival rates (ASR) on GDP growth rates at 5-year intervals in several countries. The models for growth rates were estimated considering the interaction between ASR and lagged GDPc level. Endogeneity and reverse causality were considered. Average life expectancy in many developing countries was only 40 years in 1950 but increased to 63 years by 1990. Many factors like improved nutrition, better sanitation, innovations in medical technologies, and public health infrastructure increased life span. Further, life expectancy was strongly influenced by child mortality and low-cost interventions. Bhargava et al. showed that for the poorest countries, a 1% change in ASR was associated with an approximate 0.05% increase in economic growth rate. While the magnitude of this coefficient was small, a similar increase of 1% in investment/GDP ratio was associated with a 0.014% increase in growth rate. One could see ASR in poor countries reflected the levels of nutrition, smoking prevalence rates, infectious diseases, health infrastructure and factors like accidents leading to premature deaths. By contrast, differences in ASR in middle- and high-income countries were influenced by genetic factors and by access to and costs of preventive and curative health care. Because investments in skill acquisition in poor countries depended on the ASR, the years for which skilled labor remained productive were important for explaining economic productivity.

The empirical literature on the effect of health on economic development (Bloom et al. 2004; Webber 2002; Acemoglu 2011) focused on the labor productivity effects of health on economic growth where improvements in health lead to an increase in per capita income directly, as each individual was able to produce more per unit of labor input. On the other hand, the significance of the demographic variables in growth regressions was asserted by many other authors (Bloom et al. 2004; Sala-i-Martin et al. 2004). The fertility equation was found, e.g., by Schultz (1997). He considered the determinants of fertility to be education, income, employment, religion, nutrition, family planning and child mortality. Bloom et al. (2004) provided a summary of results of various studies that used life expectancy as a proxy for health in the analysis of the direct effect of health
on education and economic growth (e.g., Barro and Lee 1984; Bhargava et al. 2001; Barro and Sala-i-Martin 2004; Sachs and Warner 1997; Blackburn and Cipriani 2002; Chakraborty 2004; Ehrlich and Lui 1991).

The role of health in economic development was analyzed via two channels in the paper by Finlay (2007), namely the direct labor productivity effect and the indirect incentive effect. The labor productivity hypothesis asserted that individuals who were healthier had higher returns to labor input. The incentive effect said that individuals who were healthier and had a greater life expectancy had the incentive to invest in education as the time horizon over which returns could be earned was extended. Education was the driver of economic growth, and thus, health played an indirect role in the model. Finlay’s results showed that the indirect effect of health was positive and significant.

Generally, the above results are, however, affected by income distribution—both directly and indirectly via its health effects. The relevant literature (see the reviews by Galor 2009; Aghion et al. 1999) shows that the inequality effects on economic growth could be substantial but diverse (Voitchovsky 2009). As different forms of inequality have a major impact on health status—on both infant mortality and life expectancy—the GDPc level and growth relationship between health status and must pay attention also to (income) in equalities.

### 3 Inequality and economic growth

Rising inequality is a concern of today. In advanced economies, the gap between the rich and poor is at its highest level in decades (Stiglitz 2013). Pervasive inequalities exist also with reference to access to education, health care and finance. Countries with higher levels of income inequality tend to have lower levels of mobility between generations, with parent’s earnings being a more important determinant of children’s earnings (Corak 2013). Redistribution has played an important role in cushioning market income inequality in mostly advanced economies. Many studies suggest that growing wealth inequality in advanced economies is largely driven by rising wealth concentration at the top (Piketty 2014; Saez 2014). An increase in income inequality can have both growth-promoting and growth-dampening effects. In their calculations, using data from 73 countries, Cornia and Court (2001) came to the conclusion that GINI coefficient value between 0.25 and 0.40 had a growth-promoting effect. At GINI coefficient value of 0.45 and above, an increase in income inequality had a growth-dampening effect.

A high level of income inequality impairs economy’s human capital insofar as low-income people do not have sufficient access to capital formation, health care and education. Typically, high level of income concentration leads to a situation where economic power is used to exert political influence to reduce taxes (Bernstein 2013). Decline in state revenues can cause reduction in investments in public health infrastructure and education. The resulting undersupply of public services dampens economic growth through a lack of public infrastructure and low productivity because of low expenditures (Galor 2011). On the demand side, a high degree of income inequality weakens the demand for goods and services. When increasing share of income goes to high-income households, the consequent savings and capital outflow causes less demand (Bernstein 2013). In less developed economies, savings can be available for investment, but sustaining consumer demand is missing. In highly developed economies, the level of capital
stock is already high. If there is then a decline in consumer demand, there is no incentive for additional investment. Thus, growth in the economy’s overall capital stock weakens along with long-term growth potential. In the medium term, this trend leads to stagnation or even economic contraction. So, in sum, whether an increasing level of income inequality dampens future economic growth—weakening both the supply side (human and real capital) and the demand side—largely depends also on the economy’s $\text{GDPc}$ level (Petersen and Schoof 2015).

Income and wealth distributions are systematically, albeit in nonlinear fashion, affected by the level of economic development. A lucid review of the “Kuznets school” is given by Piketty (1997), Deininger and Squire (1998), and Kanbur (2012). At a low level of $\text{GDPc}$, income and wealth distributions are wide but they change to become less wide when the economy reaches higher $\text{GDPc}$ level (Kuznets 1955). The modern condensed version of this hypothesis says that if the income or wealth distribution is unequal, the rate of economic growth is low. However, this version abstracts from the fact that the relationship suggested by Kuznets is path dependent. Kuznets himself used pre-World War 2 time-series data for USA, UK and Germany and argued that the level of $\text{GDPc}$ determined when the inequality–growth relationship was positive and when it was negative. Typically, at a low level of $\text{GDPc}$, one observed positive relationship between inequality and growth, while negative relationship prevailed at higher levels of $\text{GDPc}$. The latter results were expected when there were no obstacles for equal opportunity to human capital investment and productivity gains.

Generally, if capital had decreasing returns and capital markets were imperfect, the distribution of wealth mattered for $\text{GDPc}$ level and growth. In such a situation, only redistribution and public intervention to capital markets supported higher economic growth because income and wealth redistribution created many new investment projects with higher marginal returns and effort supply (Aghion et al. 1999), Benabou (1996) and Lee and Roemer (1998) concluded that generally private (human capital) investment and inequality did not show monotone negative relationship. Benhabib (2003) showed that the hump-shaped relationship comparable to Kuznets’ hypothesis could be found for inequality–growth relationship. Typically, with more unequal distribution and high capital taxes growth was hampered.

There exists also the perspective of sociopolitical theories (Gupta 1990; Alesina and Perotti 1996; Benhabib and Rustichini 1996). One version maintains that a greater degree of inequality in wealth and income could raise the likelihood of poor people participating in highly destructive activities such as crime, rioting and revolution. The resulting instability and distrust in the entire economic system could then lead to a decline in investment incentives, which could hamper long-run economic growth. The studies by Perotti (1994, 1996), Alesina and Rodrik (1994), Persson and Tabellini (1994) and De Mello and Tiongson 2006 based on the median voter and mean income model, i.e., economic growth would be improved by a middle class that is sufficiently wealthy to vote for a low level of redistribution, found evidence of a negative relationship between inequality and economic growth. Moreover, the link between redistribution (e.g., the amount of taxes) and growth is found to be weakly negative or even positive. Forbes (2000) showed that a positive relationship between inequality and growth could be possible in the short- or medium-term when one was using high quality data for income
inequality provided by Deininger and Squire (1996). She argued that it was more likely to be negative in the long-run while being significantly positive in the short-run (see also Li and Zou 1998; Halter et al. 2014).

Barro (2000) examined the relationship through three-stage least squares using the same inequality data as in Forbes (2000) with an extensive set of control variables to reflect the sociopolitical status of each country. Unlike Forbes (2000), however, his estimation results showed that the effect of inequality was insignificant once the equation included various explanatory variables representing the correlation between the degree of economic development and the inequality level. Barro showed that the effects of inequality on growth differed, depending upon stages of economic development across countries. The paper by Barro (2008) updated and extended his earlier work. International data confirmed the presence of the Kuznets' curve that was relatively stable from the 1960s to the 2000s. A cross-country growth equation showed a negative effect of income inequality on economic growth, holding fixed a familiar set of other explanatory variables. This effect diminished as GDPc level rose and was even positive for the richest countries (see also Castello-Climent 2010). Neves et al. (2016) showed that the direction of growth effects also followed a certain time pattern: in the 1990s, most of the published studies found negative effects while at the beginning of this century, this tendency was reversed and empirical studies increasingly documented positive results. Cingano (2014) investigated the relationship between economic growth and inequality for data covering most of the OECD countries over the past 30 years. Different inequality measures in the growth equation were negative and statistically significant. He also evaluated the human capital accumulation theory and delivered evidence for human capital being a channel through which inequality could affect economic growth. For additional results on inequality effects on growth see, e.g., Herzer and Vollmer (2012), Kurita and Kuroiwasaki (2011), Ostry et al. (2014), De Gregorio and Lee (2004), Lee and Son (2016), Baur et al. (2015) and Petersen and Schoof (2015).

Available evidence on the links between inequality and social mobility is also largely based on cross-country correlations showing a negative relationship between inequality and inter-generational earnings mobility in a subset of OECD countries (D’Addio 2007; Corak 2013). Recent work by Chetty et al. (2014) based on millions of administrative data on income mobility in the USA finds that (upward) mobility is robustly negatively correlated with income inequality (and positively with school quality).

A common drawback of most empirical studies analyzing the growth–inequality relationship lies in a possible misspecification of the model. They do not account for the hypothesis that the effect of inequality on economic growth could be nonlinear depending on the stage of development (i.e., the level of GDPc) and the initial level of inequality. The analysis by Kolev and Niehues (2016) delivers evidence in favor of this hypothesis. Economic growth is negatively correlated with net income inequality for countries with low initial level of GDPc. However, the effect becomes weaker with increased GDPc level and even positive for the case of developed countries. In addition to Kuznets' hypotheses, the human capital accumulation theory can motivate nonlinear inequality effects on economic growth.

Banerjee and Duflo (2003) stress the fact that the relationship between change in income inequality and GDPc growth is highly nonlinear and find empirical support
for their conjuncture by using partial linear models. This has naturally implications for Kuznets type analysis although Banerjee and Duflo (2003) anchor their analysis mostly to a political economy model. Their results on partially linear models imply that there exists nonlinearity, and for mostly data points negative relationship, between growth and changes in GINI coefficients for 45 non-poor countries in years 1965–1995 with non-overlapping five-year period panels. They use controls taken from Barro (2000) and Perotti (1996). They sum up that changes in the inequality variable are associated with lower growth in the short run, independent of the direction of these changes. Accordingly, they argue that this nonlinearity can explain why the results of empirical research have been so different under the constraint of linear specification. Chen (2003) even finds a statistically significant quadratic term in the regression analysis pointing toward an inverted U-shaped relationship between inequality and economic growth.

The study by Voitchovsky (2005) has the central hypothesis that top-end inequality encourages growth while bottom-end inequality retards growth. This is explored using a standard growth model and a set of explanatory variables to control for inequality at the top and the bottom ends of the income distribution simultaneously. A panel GMM estimation is undertaken on a sample of industrialized countries, using data from the Luxembourg Income Study (2003) which indicates that inequality at different parts of the distribution does have different implications for growth, i.e., the profile of inequality is also an important determinant of economic growth. Top-end inequality appears to have a positive effect on growth, while inequality further downs the income distribution appears to be inversely related to subsequent growth. These findings highlight possible limitations of an exploration of the impact of income distribution on growth using a single inequality statistic.

Dominica et al. (2008) and Neves et al. (2016) conduct a meta-analytic reassessment of the effects of inequality on growth. The former points out that the magnitude of the estimated effect of inequality on growth in the literature depends crucially on the estimation method, data quality and sample coverage. Studies using panel fixed effects estimators seem to report stronger effect of inequality on economic growth than cross-sectional results. Overall, the results of the meta-analysis by Dominica et al. (2008) show that the inequality effect on growth tends to be negative and more pronounced in less developed countries. Neves et al. (2016) extend the meta-analytic reassessment to more recent studies and show that the empirical literature on the inequality–growth nexus is biased toward statistically significant results. As the authors stress, this makes the empirical effect of inequality on economic growth seem larger in absolute terms than what it is actually.

From policy perspective, the current level of results is too mixed. There is no one-size-fits-all approach to tackling inequality effects on GDPc. One neglected issue is that growth rate of GDPc and income inequality affects each other simultaneously (Baumol 2007; Lundborg and Squire 2003; Huang et al. 2009). It is not only the level of GDPc that conditions the income inequality but the growth rate also matters. In the following these questions are cast back in the framework of Kuznets’ hypothesis to have a systematic approach to build simultaneous model between GDPc, health level and health expenditure where inequality plays an important role.
4 Models, data and methods

4.1 Kuznets approach

In following we propose a simultaneous three-equation model for GDP per capita level (or GDPc growth), health status (HS) measured with infant mortality rate (IM) and health expenditures per capita (HEc) for data set of 194 countries in years 1990–2014. Income level and inequality (INEQ) determine IM that subsequently affects the level of HEc with income level. GDPc is also determined by typical labor or population, educational and technological input variables. In order to analyze GDPc growth effects we propose a similar simultaneous model GDP per capita growth rate (\(\Delta\ln\text{GDPc}\)). Models are given in details in Sect. 4.3.

Nonlinear Kuznets’ GDPc effects with income inequality depending on the level of GDPc must be specified into the model. This happens by estimating the model for three different GDPc level country groups. The nonlinear income level effects on inequality allow us to analyze the Kuznets’ hypothesis in details that is often neglected in the literature. Because our system model includes also linked equations for health status and health expenditures we argue that low-income high-inequality trap implied by Kuznets’ hypothesis can be escaped under suitable conditions by rising the health expenditures–GDP ratio and with cost-effective health technology. This happens even if the negative inequality–health status relationship is still valid in the poorer countries. In practice we test for Kuznets’ hypothesis maintaining a positive income inequality relationship for poor countries contrary to rich countries in the model where we estimate also the empirical linkages between GDPc, HS and HEc variables.

4.2 Health-poverty trap and Kuznets hypothesis in income–health model

Main argument behind our empirical model is the following stylized figure (see also Shin 2012). It shows that

1. if a country is in the low-GDPc high-inequality trap (R1), it can escape from it by increasing inequality (INEQ) that sustains higher GDPc, and
2. after some high level of inequality GDPc increases only if inequality starts to decrease, but
3. if the country increases its relative investment in health (i.e., HE/GDP – ratio increases: \(A \to A^*\)), and the new level of health expenditures (HE) is utilized efficiently to raise country’s health status (HS), then the country will escape from the low-GDPc high-inequality trap (R2) (see Fig. 1).

Note that negative and monotone health status and inequality relationship (HS–INEQ) with large given inequality is the main part of our argument. It can be supported with many arguments, but empirically it is still valid for the majority of poor and low-income countries (see, e.g., Linden and Ray 2017; Deaton 2003, 2013). Thus, we exclude from the analysis the case of poor country with equal income distribution. Note also that Kuznets’ hypothesis is not necessary for our policy alternative (increases in HE/GDP and HS/HE ratios) to work out successfully. Hypothesis makes it only harder way to happen as the way out of trap is not a direct one. The results
with Kuznets’ hypothesis speak for big jump—health policy alternative with large exogenous productive investments in health services and technology with some short-run GDPc reductions to find the low inequality but later on a higher GDPc position. If GDPc-INEQ curve is monotonic and negative (less INEQ and more GDPc), the small-step policy will work out smoothly without rising inequality. If we have monotonic positive GDPc-INEQ curve (more INEQ and more GDPc) it leads in progress to cyclical GDPc long-run solution (see “Appendix 1”) that the active health promoting does not make less volatile.

Note that the above model with related policy options and arguments was based on the modern reading of Kuznets’ hypothesis, i.e., possible causation goes from inequality to level and to growth of GDP per capita. The original hypothesis was more the opposite one noticing that first the level of GDPc determines the income and wealth inequalities that must increase to give the way to the growth, and later on the growth is hampered if the inequalities are too large. In more formal way this means that inequality is \( \cap \)-shaped function of GDPc like

\[
INEQ = a + b \cdot GDPc + d \cdot GDPc^2
\]

with \( b > 0 \) and \( d < 0 \). To express this as inverse function of \( INEQ \) is a difficult task and we don’t pursuit in that direction here. Instead, we analyze what can be obtained from the function

\[
GDPc(t) = a + b \cdot INEQ(t) + d \cdot INEQ(t)^2.
\]

We have added time to variables to get the growth presentation of equation, i.e.,

\[
dGDPc = \beta \cdot dINEQ + 2\delta \cdot INEQ \cdot dINEQ = dINEQ \cdot (\beta + 2 \delta \cdot INEQ).
\]

The negative inequality–growth relation favored by modern literature is determined by conditions: \( \beta > 0 \) and \( \delta < 0 \) for given (positive) value of \( dINEQ/dt \) and large values of \( INEQ \). However, note the complex nonlinear relationship between the time change of \( GDPc \), time change in \( INEQ \), and the level of \( INEQ \). Thus, with time-series observations...
the linear growth regressions on the level of INEQ are biased if the true relationship is nonlinear (Banerjee and Dufo 2003).

The first derivate of GDPc with respect to INEQ (or \[ \frac{dGDPc}{dINEQ} \]) gives result:

\[
\frac{dGDPc}{dINEQ} = \beta + 2\delta \cdot INEQ,
\]

i.e., when \( \beta > 0 \) and \( \delta < 0 \) change in GDPc (with respect to positive change in INEQ) is positive when INEQ is not above \( IE^* \) (Fig. 2, right panel). When we compare this outcome to “classical” Kuznets’ proposition, we observe similarities between the models as long as \( IE < IE^* \) with low values of GDPc (see Fig. 2, left panel).

Both models imply that inequality must rise to sustain the GDPc level first (the classical Kuznets hypothesis), but for Kuznets’ curve a radical change happens at some level of GDPc (and INEQ) that turns GDPc–INEQ relationship negative. In our model this is also found at high values of INEQ but level of GDPc decreases after it, i.e., any low INEQ–high GDPc solutions are not found in our model. The difference between the models comes more evident when we look at models in derivate terms \( dGDPc/dINEQ \). In Kuznets’ model the derivate is infinite at the turning point value \( IE^* \) (sometimes called also as a catastrophe point) implying that derivate is \( \rightarrow +\infty \) and \( \rightarrow -\infty \) very close to it. Empirically this is not a reasonable outcome. However, in our model there exists a well-determined region not identified by original Kuznets’ curve below \( IE^* \) level where inequality rises but positive (time) change of GDPc decreases to zero giving negative relation between positive growth rate of GDPc and INEQ. However, the GDPc level and INEQ relationship is still positive up to point \( IE^{**} \). In practice this means that some implications of Kuznets’ hypothesis and curve can be estimated with our \( \cap \)-shaped function

\[
GDPc(t) = \alpha + \beta INEQ(t) + \delta INEQ(t)^2.
\]

Case of \( \beta > 0 \) and \( \delta < 0 \) is here relevant—before the maximum point of our \( \cap \)-shaped function is obtained—for the modern GDPc growth literature. Note that some features of the upper arm of Kuznets’ curve is obtained also with \( \beta > 0 \) and \( \delta < 0 \) values when

![Fig. 2 Kuznets curve and \( \cap \)-shaped GDPc function of inequality](image-url)
INEQ is large, i.e., negative relationship between INEQ and GDPc level. To sum up we
argue that our GDPc–INEQ model gives an efficient way to test Kuznets–hypothesis for
countries at different GDPc levels. Next this is done with simultaneous model in the con-
text of health status and expenditure relationships with GDPc.

4.3 Health–income simultaneous equations model
As inequality is only one of the factors of economic growth, we can see empirically quite
different inequality–growth correlations with, e.g., income GINI coefficients and GDPc
growth rates when the other growth factors are neglected. Thus, without paying atten-
tion to the starting level of GDPc, human capital, and other growth inputs these corre-
lations are misleading. We focus next on simultaneous health expenditure and status
relationships with level and growth of GDPc where income distribution determines both
health status and GDPc.

The following three-equation model captures the interlinked health and growth effects
that are conditioned with prevailing income inequality in economy. The first equation is
a typical empirical GDPc level equation based on production function that is augmented
with economy’s health status (HS) and second-order polynomial in income inequality
to measure its nonlinear GDPc effects. Population and educational variables (POP and
EDUC) measure the effects of labor and human capital inputs on income. Variable
TECH stands for the capital input measured as the change in nation’s technological
advance. The second equation gives the determination of nation’s health status related
to per capita GDP level, inequality and other health improving (exogenous) variables X.
Finally, health expenditures per capita (HEc) are determined by GDPc level, health status
and some health expenditure-related (exogenous) variables X. Thus, in general terms we
argue that the income level (and growth) of economy is determined simultaneously with
its health status and health expenditures. This means that in the model at least variables
lnGDPc, lnGDPc, lnHS, and lnHEc are endogenously determined. The structure of the
model is identifiable and needs instrumental variable (IV) estimation approach.

\[
\begin{align*}
\ln\text{GDPci} &= \alpha_0 + \alpha_1 \ln\text{HSi} + \alpha_2 \ln\text{INEQi} + \alpha_3 \ln\text{POP} + \alpha_4 \ln\text{EUCi} + \alpha_6 \ln\text{TECHi} + \epsilon_{1i}, \\
\ln\text{HSi} &= \beta_0 + \beta_1 \ln\text{GDPci} + \beta_2 \ln\text{INEQi} + \beta_3 X_{1i} + \epsilon_{2i}, \\
\ln\text{HEci} &= \alpha_0 + \alpha_1 \ln\text{GDPci} + \alpha_2 \ln\text{HSi} + \alpha_3 X_{2i} + \epsilon_{3i}.
\end{align*}
\]

Figure 2 and the above model are based on Kuznets’ hypothesis arguing that the relation
between the GDPc level and inequality is hump-shaped. However, the more recent
modeling (see Sect. 3) has focused on the growth rate and inequality. Albeit this growth
formulation was easily derived in the preceding section in Kuznets framework, the mod-
ern theory and empirical literature have neglected the Kuznets’ approach too often.
Thus, we follow partly here the Kuznets’ formulation prosed by Shin (2012) that the level
of GDPc conditions also the inequality effects on GDPc growth rate, i.e., in poor coun-
tries inequality growth effects can be positive but in more advances countries they are
negative. To get also more direct results comparable with modern literature, the first
equation in our simultaneous model has now the following form

\[
\Delta\ln\text{GDPci} = d_0 + d_1 \ln\text{HSi} + d_2 \ln\text{INEQi} + d_3 \Delta\ln\text{POP} + d_4 \Delta\ln\text{EUCi} + d_5 \Delta\ln\text{TECHi} + \epsilon_{4i},
\]
where we expect have positive sign for \( d \) poor countries, and negative sign is found in rich countries. To test this and the general nonlinear Kuznets’ approach, we divide our data (194 countries) in three income groups in 1995–2014 period:

S1: countries that average GDPc level was less than 1000US$,
S2: group countries with GDPc level in margins 1000–10,000US$, and
S3: group countries with GDPc level in the above 10,000US$.

### 4.4 Estimation strategy

To avoid the evident endogeneity of almost all variables in growth process modeling, we used the following approach. First, all our modeling variables are measured with the mean values of country specific variable values in period 1995–2014 (20 years: \( \bar{Y}_i, \bar{X}_i \)).

This makes the measurements independent of cyclical effects, and we can focus on average the long-run results. Second, we use means of variable measurements from period 1990–1994 as additional and instrument variables in model estimation. Thus, our instrumentation leans to pre-determination of variable values in relation to model estimation period. Variables dated at the beginning of sample period 1990–1994 reduce the problem of endogeneity. Note that if the variables are highly persistent endogeneity may still persist. Thus, we argue that (a) variable set (\( \bar{Y}_i, \bar{X}_{i-1} \)) from period 1990–1994 does not correlate with model errors (\( \varepsilon_{1i}, \varepsilon_{2i}, \varepsilon_{3i} \)) in period 1995–2014, and (b) our additional instrument variables (\( \bar{Z}_i, \bar{Z}_{i-1} \)) do not correlate with model errors and not with left-hand-side endogenous variables, i.e., we have \( E[(\varepsilon_{1i}, \varepsilon_{2i}, \varepsilon_{3i})|\bar{Y}_i, \bar{Z}_{i-1}] = 0 \) and \( E[\bar{Y}_i|\bar{Z}_i, \bar{Z}_{i-1}] = 0 \).

These conditions put high pressure for finding proper instrument variables that are not ill-conditioned by weak instruments and endogeneity. “Appendix 2” gives a more detailed description of methods used. However, we note that our \( \bar{Z}_i, \bar{Z}_{i-1} \) set compromises of values of alcohol consumption per capita (AC), percentage of population using improved drinking water source (\( AW \)), geographic area of country in km²'s (\( AREA \)) and means of GDPc level and growth of countries having the same level of development as country \( i \) (\( GDPc_{iv} \)). The last one is advocated by Pritchett and Summers (1993) as a valid instrument in this context. Thus, we argue that our endogenous left-hand-side variables \( \ln GPDc, \Delta \ln GPDc, \ln HS \) measured with infant mortality (\( \ln IM \)), and \( \ln HEc \) measured with total health expenditures per capita are not necessarily affected by values of variables \( AC, AW, AREA \) and \( GDPc_{iv} \) in period 1995–2014 (\( Z_i \)) and by their period 1990–1994 values (\( Z_{i-1} \)).

For example, in the above GDPc level equation the values of chosen instruments are independent of variable GDPc values but they affect the HS (infant mortality rate) values. Thus, e.g., using pre-determined period 1990–1994 mean values of the percentage of population using improved drinking water source (\( AW_{-1} \) or \( \ln AW_{-1} \)) as an instrument for \( IM \) is a natural choice in this equation but we also argue that it will not directly affect the value of GDPc.

### 5 Results

Table 1 reports the GDPc level GMM-2SLS estimation results for the model variables with main interest of this study. More detailed information on data used and estimation results is found in “Appendices 3 and 4.”
variables to be valid instrument in this context. Thus, we argue that our endogenous
variables need to be measured with the help of proper instrument variables. To avoid the evident
endogeneity of almost all variables in growth process modeling, we used the following approach. First,
all our modeling variables are measured with the means of variable measurements from period 1990–1994.
Variables dated at the beginning of sample period 1990–1994 reduce the quality of long-run results. Second,
we use means of variable measurements from period 1995–2014.

4.4 Estimation strategy

Table 1 reports the estimation results for structural parameters for GDPc level equation in income
level groups S1, S2 and S3 (GMM-2SLS, estimated values in parenthesis if their significance level is above 5% level)

<table>
<thead>
<tr>
<th>Right-hand-side variables</th>
<th>S1: &lt;1.000s</th>
<th>S2: 1.000s–10.000s</th>
<th>S3: &gt;10.000s</th>
</tr>
</thead>
<tbody>
<tr>
<td>lnGDPc equation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnIM</td>
<td>−1.320</td>
<td>−0.756</td>
<td>−0.361</td>
</tr>
<tr>
<td>GNY</td>
<td>0.161</td>
<td>(−0.030)</td>
<td>(0.047)</td>
</tr>
<tr>
<td>GNY²</td>
<td>−0.0015</td>
<td>(0.0005)</td>
<td>(−0.0003)</td>
</tr>
<tr>
<td>lnIM equation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnGDPc</td>
<td>−0.178</td>
<td>−0.555</td>
<td>−0.641</td>
</tr>
<tr>
<td>GNY</td>
<td>(−0.003)</td>
<td>0.014</td>
<td>0.061</td>
</tr>
<tr>
<td>lnHEc equation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnGDPc</td>
<td>0.777</td>
<td>0.967</td>
<td>0.725</td>
</tr>
<tr>
<td>lnIM</td>
<td>(0.894)</td>
<td>(0.075)</td>
<td>0.164</td>
</tr>
<tr>
<td>lnDOC</td>
<td>0.239</td>
<td>0.182</td>
<td>0.315</td>
</tr>
</tbody>
</table>

5.1 Health status (IM) effects on GDPc and HEc

Health status measured with infant mortality (lnIM) has a negative effect on the level GDPc. Largest
effects are found in the poor countries with less than 1000US$ per capita income with coefficient value of −1.32. Interestingly we found statistically negative effects also in rich countries (−0.361). In the middle-income countries 10% increase in infant mortality rate decreases the level of GDPc with 7.56%. Contrary to these significant results, in both statistical and economic terms, we found only health status (IM) effects on health expenditures (HEc) in rich countries. A 10% increase in infant mortality rate increases health expenditures by 1.64%.

5.2 Income level (GDPc) effects on HS and HEc

In lnIM equation the level of GDPc decreases most effectively infant mortality in rich
countries. A 10% increase of GDPc reduces the infant mortality rate by 6.4%. In poor
countries the effect is only 1.78%. In lnHEc equation income effects on health expenditures are largest in the middle-income countries (0.967). However, if we pay attention to the number of doctors per population (lnDOC) as part of health expenditures, the sum of these health expenditure elasticities is smallest in poor countries. Note, however, that in all country groups the sum of elasticities of GDPc and DOC on HEc is numerically above one.

From the policy perspective these results mean that (marginally) healthier people (i.e.,
reduced IM in lnGDPc equation) generates more income in poor countries compared to
rich countries but when the level of GDPc is large enough, the wealth effects (GDPc in
lnIM equation) increase health more effectively in richer countries than in poor coun-
tries. Health policy to reduce infant mortality seems to rise health expenditures (HEc)
only in rich countries. However, GDPc level together with number of doctors per capita
generate health expenditures in same magnitudes with respect to given level of GDPc in
all income levels. These results may be an indication of poor efficiency and different
targets for health expenditures in non-rich countries. In sum, if only one policy option
is allowed then policy to reduce infant mortality is still a cost-effective policy alternative.
For example, in the poor economies the net effect between \( GDPc \) and \( IM \) in \( lnGDPc \) and \( lnIM \) equations is large. A 10% reduction in \( IM \) means (ceteris paribus) a 13.2% increase in \( GDPc \) that sustains a lower \( IM \) rate with an estimated value of 2.34% \((13.2 \times 0.178)\). However, the (income) inequality effects on \( GDPc \) and \( IM \) may make this less evident.

5.3 Income inequality (GINI) effects on \( GDPc \) and HS

The \( GINI \) coefficients result in \( lnGDPc \) equation shows that nonlinear inequality effects (Kuznets’ hypothesis) on \( GDPc \) are found only in poor countries. We obtain \( \cap \)-shaped \( GINI \) outcome on \( GDPc \) level. As the \( GINI \) coefficients have values in range of 40–70 in poor countries, this means that our estimate for \( GDPc–INEQ \) relationship is \( \text{hump-shaped} \) (see Fig. 3). Estimation results for infant mortality rate (\( lnIM \) equation) show that in middle- and high-income countries increase in income inequality affects health status adversely, i.e., higher \( IM \) rate. Surprisingly in the poor economies (\( S1 \) group countries) this is not found. In these countries there is an indirect inequality effect on infant mortality rate: increase in \( GDPc \) level happens most likely with increasing inequality but only the \( GDPc \) increase lowers infant mortality rate that is not directly affected by \( GINI \).

Thus, the problem of infant mortality in poor economies is not so much caused by the income inequality but by the low level of \( GDPc \).

From the policy perspective this result is interesting. Now for a poor country with large inequality increases in \( HE/GDPc \) and \( HS/HE \) ratios lead to higher level health status. However, this can also happen with less inequality (compare points \( B \) and \( B^* \) in Fig. 3). Between them we find an optimum value of inequality \( B^{**} \) that provides the highest level of \( GDPc \). Note that if \( HS–INEQ \) relation with typical shape as depicted in Fig. 3 would be valid here this would mean that quite moderate policy increase in \( HE/GDP \) or \( HS/HE \) ratio would move us from \( B \) to \( B^* \) or to \( B^{**} \). However, the \( \cap \)-shaped \( GDPc–INEQ \) relationship allows not for a permanent trajectory with less income inequality and higher \( GDPc \) level. Only way to escape this trap is to hold to the health policy that raises health status and sustains eventually for higher \( GDPc \) level that \( \text{breaks} \) the \( \cap \)-shaped \( GDPc–INEQ \) relationship and allows for less inequality.

![Fig. 3 Determination of health status (HS) in poor countries with Kuznets’ curve identified with \( \cap \)-shaped function and health expenditures (HE)](image-url)
higher relationship allows not for a permanent trajectory with less income inequality and ratio would move us from or HS/HE est level of Fig. 3). Between them we find an optimum value of inequality status. However, this can also happen with less inequality (compare points large inequality increases in tries) this is tus adversely, i.e., higher in middle- and high-income countries increase in income inequality affects health sta-

shaped
GINI outcome on (Kuznets’ hypothesis) on

The 5.3 Income inequality (GINI) effects on GDPc and HS

5.4 IM and income inequality (GINI) effects on GDPc growth

ΔlnGDPc equation estimation results with linear effects of the GINI coefficients are given in Table 2. First, we observe that infant mortality rate has a positive but statistically insignificant effect on economic growth in all income groups. However, we find for all income level groups that larger income inequality (GINI) predicts less GDPc growth rate. The growth-reducing effect is largest in rich countries. For the poor countries significant negative estimate value of − 0.0009 means that these countries can be located on upper Kuznets’ curve arm and also left from the point IE** in Fig. 2.

When comparing the above GMM-2SLS results with SURE and GMM system results (obtained upon request), we observe that there exist larger differences between GMM-2SLS and SURE results than between GMM-2SLS and GMM system results in terms of point estimates and their SEs. As expected GMM system estimates are more efficient than GMM-2SLS estimates. However, the estimate and efficiency differences are not large enough in most cases to make above inferences with GMM-2SLS redundant. Also according to tests found in “Appendix 4” it is shown that our choice of instruments is valid one.

5.5 Endogenous income inequality effects

Basically, the Kuznets’ hypothesis says that income level determines income distribution. The endogeneity of GINI coefficient is evident in this case. Next we do not propose an additional equation in the model for GINI variable but we estimated the above models treating GINI also as endogenous variable. Results in Tables 6, 7 and 8 (in “Appendix 4”) show that significant results with endogenous GINI were hard to obtain. Some results are still noteworthy. GMM C test rejects for poor countries the null alternative of exogenous GINI (see Table 6 in “Appendix 4”) for dlnGDPc equation, and the sign of GINI variable coefficient estimate is positive with 5% level. A clear increasing inequality effect on infant mortality is now also found for the poor countries in lnIM equation. However, in lnGDPc equation the estimates for second-order polynomial of GINI are very imprecise for all country groups. We take this as evidence of difficulty of finding proper instruments for endogenous GINI variable. The first-stage equation results in lnGDPc equation for GINI and GINI² show the evidence of weak instruments. We leave the further analysis with endogenous GINI variable for future research.

6 Conclusions

Income–health relationship is in the core of health economic analysis—at both micro- and macro-levels. We analyzed the simultaneous relationships between health status measured with infant mortality rate and GDPc level and GDPc growth in the
presence of income inequality variable ($GINI$) in infant mortality and income equations. System model included also equation for determination of health expenditures ($HEc$). Our sample consisted of data set of 194 countries in years 1990–2014. Cross section of country mean values of relevant variables in period 1995–2014 was used as estimation sample. Year 1990–1994 mean values were used as instrument variables with addition to some other variables. The cross-sectional data were divided into three country groups based on the mean $GDPc$ level values during the sample period 1995–2014. This was done in order to test for Kuznets’ hypothesis maintaining a positive nonlinear relationship between $GDPc$ level and income inequality in poor countries contrary to the rich countries.

Our GMM-2SLS estimation results indicated that the simultaneous positive infant mortality rate and $GDPc$ level effects are found in all country groups sustaining decrease in mortality rate with an increase in $GDPc$ level with positive feedback effect from lower mortality rate to $GDPc$. Increasing income inequality measured increased infant mortality rate. However, positive $GDPc$ growth effects from infant mortality rates were not found. Health expenditures had larger than one elasticity when effects from $GDPc$ level and number of doctors per capita were summed together.

We argued that for poor countries the low-income high-inequality trap can be also escaped in the presence of Kuznets’ hypothesis by rising the health expenditures–$GDPc$ ratio and with cost-effective health technology. This happens easily if the negative health status–inequality relationship is not valid or if the countries’ have positive nonlinear $GDPc$ level dependence on income inequality with decreasing curvature. This was identified with $\cap$-shaped $GDPc$ function on $GINI$ coefficient. Both of these conditions were found in our sample only for the poorest countries and not rejecting the Kuznets’ hypothesis. However, the negative income inequality effects on growth of $GDPc$ were found in all income level country groups. Weak tentative IV results with endogenous $GINI$ did not support the Kuznets’ hypothesis for any country income level group and only significant, albeit positive, $GINI$ effect on growth of $GDPc$ was found for the poor countries.

From the economic growth policy perspective, our results mean that the policy to combine exogenously determined fight against infant mortality and income inequality means higher $GDPc$ level in long run. In the poorest countries Kuznets’ hypothesis and low-income high-inequality trap may still be present but these can be avoided by breaking the possible negative relationship between income inequality and raising health status with the suggested policy. If this is not possible the positive income–health relationship will eventually make income inequality as obstacle to growth as the higher $GDPc$ levels are reached.

Authors’ contributions
The first author wrote the following parts: introduction, health and economic growth, inequality and economic growth, data, results and conclusions. The second author formulated the models, methods, added to results and appendices. Both authors read and approved the final manuscript.

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Competing interests
The authors declare that they have no competing interests.
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GDP ratio and with cost-effective health technology. This happens easily if the
escaped in the presence of Kuznets’ hypothesis by rising the health expenditures–
infant mortality rate. However, positive
from lower mortality rate to
decrease in mortality rate with an increase in
mortality rate and
increase in

Appendix 1: Cyclical GDPc solution
Assume that GDPc is determined by INEQ in the following way

\[ GDPc_t = a + bINEQ_t \] (b > 0),

and this has also a negative effect on following period’s health status HS_{t+1}

\[ HS_{t+1} = c + dINEQ_t \] (d < 0).

Next, if we have a positive relationship between GDPc_{t+2} and t + 1 period’s health status,

\[ GDPc_{t+2} = e + fHS_{t+1} \] (f > 0),

then all this sums up to a second-order difference equation (DE) for GDPc:

\[ GDPc_{t+2} = e + f \cdot (c + d \cdot INEQ_t) = e + f \cdot (c + d \cdot (\frac{-\phi}{b} + \frac{1}{b}GDPc_t)) \]
\[ \Rightarrow GDPc_{t+2} - \phi GDPc_t = \tau (\phi = f \cdot d \cdot (\frac{1}{b}) < 0). \]

The solution for this DE equation is cyclical, i.e., roots are \( \pm \sqrt{\phi} \).

Appendix 2: GMM-2SLS
The first equation in our model has the form of \( y_i = x_i' \beta + \epsilon_i \) where \( x_i' = y_{i,2} \) indicates the right-hand-side endogenous variables. The vector of instruments \( z_i' = x_{i,2} \) corresponds to exogenous variables. The (orthogonal) moment condition says that \( E[z_i'(y_i - x_i' \beta)] = 0 \). This is the general starting point to all IV estimators. When the number of instruments exceeds the number of right-hand-side endogenous variables, like here, we have the over-identified case. Note that all IV estimators are prone to finite-sample bias even they are consistent in infinite samples. The question is how much bias we tolerate for our IV estimators with chosen instruments in finite samples.

The weak instrument analysis can give us some guidelines to follow. Before these options it is good thing to conduct, e.g., correlation and partial correlation analysis between endogenous and their instrument variables. Likewise the tests for regressor endogeneity and for over-identifying restrictions (i.e., tests for instrument orthogonality) must be performed. The former tests, like the Durbin–Wu–Hausman test, assume that endogenous variables are independent (exogenous) and compare ordinary OLS results to IV results. If the differences are large in the coefficient values, it indicates that OLS approach is wrong. Alternatively, we regress endogenous variables \( y_{i,2} \) on all exogenous variables including the instruments and use residuals of these OLS models as explanatory in OLS model for \( y_i \) without \( y_{i,2} \). This gives the robust regression \( F \) test by Hausman (1978). Rejections with tests for over-identifying restrictions imply that at least one of
instruments is not valid. However, rejection has also implications concerning the model misspecification, i.e., some instruments should be considered as model variables.

Weak instrument analyses rest on the assumption that instruments are valid. However, this consistency does not guarantee that the instruments can’t be weak ones. Thus, instruments can well be uncorrelated with model errors terms but they do not correlate enough strongly with the endogenous variables. Then the OLS model for single endogenous variable \( y_{ij} \) on \( z'_{ij} = x'_{ij} \) will provide low \( R^2 \), \( t \) and \( F \) test values. Now the asymptotic theory of IV estimators may provide a poor guide in finite samples and large biases are expected in estimates. The question is how large biases we tolerate depends on the number of endogenous variables and instruments (i.e., over-identifying restrictions). Stock and Yogo (2005) provide some tests to analyze the severity of weak instruments with 2SLS, GMM and LIML estimators. The results are based on minimum eigenvalue of a matrix analog of \( F \) statistics. To use tests one has to first decide the size of bias tolerance compared to biased OLS results. If we take bias to be less than 10% of OLS results with two endogenous variables and five instruments, we have critical value of 8.78 for 5% \( F \) test. If less than 20% bias is allowed with four instruments, the critical value is 5.57. Obtaining test values larger than these, e.g., over 10, we reject the null hypothesis of weak instruments indicating reliable IV results.

**Appendix 3: Variables and data sources**

\[ GDP_{c_{it}} = GNI_{PC_{it}}, \text{ gross national income per capita in current US$ calculated using World Bank Atlas method. Sources: World Bank (2016), kushnirs.org (2016).} \]

\[ IM_{it} = \text{Infant mortality rate is the number of infants dying before reaching 1 year of age, per 1000 live births in a given year. Sources: World Development Indicators (World Bank 2016), Global Health Observatory Data (WHO 2016), UN Data (UN.org 2016) and UNICEF statistics (2016).} \]

\[ INEQ_{it} = GINI_{it}, \text{ Gini Index. Source: SWIID Version 4.0 (Solt 2009). The "gini}_\text{market" data are taken, which is the estimate of Gini index of inequality. This is equivalent to (square root scale) household market (pre-tax, pre-transfer) income. Here Luxembourg Income Study data are used as the standard. Further, data are obtained from OECD (OECD.StatExtracts 2014). UNU WIDER (2015) data are also collected. Data from the Inequality project hosted by the University of Texas (2016) complement the other data. Additionally data from “All the Ginis Dataset” (World Bank 2016) are also retrieved.} \]

\[ AC_{it} = \text{alcohol consumption/adult(15+) in liters of pure alcohol per person per year. Source: WHO (2016) and Quandl.com (2016).} \]

\[ HEces_{it} = TOTHEXPces, \text{ total health expenditure. Derived as } \% \text{ of } GDP_c. \text{ Sources: Global Health Expenditure Database (WHO 2016). Other sources include OECD.org (2016), World Bank (World Bank 2016) and Gapminder.org (2016).} \]

\[ POP_{it} = \text{total population, both sexes, combined in thousands. The following population age structure variables are used: age cohorts 0-14 (POP1_{it}), 15-64 (POP2_{it}) and 65+(POP3_{it}). Sources: UNPD (2015 and 2016) UN.org (2016), World Bank (2016) with reference to the total and the three age cohorts.} \]

\[ POPLU_{it} = \text{urban population as } \% \text{ of total population. Sources: World Bank (2016), and Gapminder.org (2016).} \]
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Obtaining test values larger than these, e.g., over 10, we reject the null hypothesis of

with two endogenous variables and five instruments, we have critical value of 8.78 for

ance compared to biased OLS results. If we take bias to be less than 10% of OLS results

matrix analog of 2SLS, GMM and LIML estimators. The results are based on minimum eigenvalue of a

berg compared to endogenous variables. Then the OLS model for single endoge-

instruments can well be uncorrelated with model errors terms but they do not correlate

ever, this consistency does not guarantee that the instruments can’t be weak ones. Thus,

misspecification, i.e., some instruments should be considered as model variables.

Weak instrument analyses rest on the assumption that instruments are valid. How-

ulner population as % of total population. Sources: World Bank (2016), and

Other sources include OECD.org (2016), UN Data (UN.org 2016) and

Income Study data are used as the standard. Further, data are obtained from OECD

(data are taken, which is the estimate of Gini index of inequality. This is equivalent to

Appendix 3: Variables and data sources

Table 3 GMM-2SLS estimation results for country $group = 1$ (robust $t$ values in parenthesis, $N=50$, 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnM</th>
<th>lnHEc</th>
<th>dlnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>8.714 (2.69)</td>
<td>6.233 (8.65)</td>
<td>-3.747 (1.29)</td>
<td>-0.283 (1.60)</td>
</tr>
<tr>
<td>lnIM</td>
<td>-1.320 (-1.97)</td>
<td>0.894 (1.50)</td>
<td>0.052 (1.54)</td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>0.162 (2.57)</td>
<td>-0.003 (-0.58)</td>
<td>-0.0009 (-1.90)</td>
<td></td>
</tr>
<tr>
<td>GINI_1</td>
<td>-0.0014 (-2.46)</td>
<td>0.009 (0.03)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP</td>
<td>-0.267 (-1.23)</td>
<td>0.036 (2.79)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP_1</td>
<td>5.295 (2.13)</td>
<td>0.0006 (0.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnPCR</td>
<td>0.088 (2.09)</td>
<td>0.302 (6.40)</td>
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<td></td>
</tr>
<tr>
<td>lnRD_E</td>
<td>-0.178 (-2.06)</td>
<td>0.777 (2.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnAW</td>
<td>-0.178 (-2.06)</td>
<td>0.777 (2.02)</td>
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<td></td>
</tr>
<tr>
<td>lnPOPU</td>
<td>0.150 (1.89)</td>
<td>0.239 (2.36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.081</td>
<td>0.243</td>
<td>0.309</td>
<td>0.364</td>
</tr>
<tr>
<td>GMW C test for exogeneity</td>
<td>lnIM</td>
<td>lnlnGNIPc</td>
<td>lnIM, lnGNIPc</td>
<td>lnIM, lnGNIPc</td>
</tr>
<tr>
<td>Robust $F$ test for first-stage regressions</td>
<td>lnIM</td>
<td>lnlnGNIPc</td>
<td>lnIM, lnGNIPc</td>
<td>lnIM, lnGNIPc</td>
</tr>
<tr>
<td>Hansen’s $J$ test for over-identifying restrictions</td>
<td>chi$^2$(1) = 2.19</td>
<td>chi$^2$(1) = 2.07</td>
<td>chi$^2$(1) = 1.86</td>
<td></td>
</tr>
<tr>
<td>Additional instruments</td>
<td>AC, AC_1, lnAREA, AW_1</td>
<td>lnGDP_i, lnGDP_iv_1, lnGDP_iv_1</td>
<td>AC_1, AW_1</td>
<td>AC_1, AW_1</td>
</tr>
</tbody>
</table>

Appendix 4: estimation results

See Tables 3, 4, 5, 6, 7 and 8.
### Table 4 GMM-2SLS estimation results for country group = 2 (robust t values in parenthesis, N=94, 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnHEc</th>
<th>dlnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>9.718 (6.45)</td>
<td>12.302 (10.30)</td>
<td>−2.038 (−1.48)</td>
<td>0.086 (3.17)</td>
</tr>
<tr>
<td>InIM</td>
<td>−0.756 (−3.42)</td>
<td>0.075 (0.50)</td>
<td>−0.002 (−0.17)</td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>−0.030 (−0.58)</td>
<td>0.014 (2.18)</td>
<td>−0.001 (−2.73)</td>
<td></td>
</tr>
<tr>
<td>GINI²</td>
<td>0.0001 (0.31)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP</td>
<td>11.40 (2.69)</td>
<td></td>
<td></td>
<td>0.003 (3.22)</td>
</tr>
<tr>
<td>lnIMm, InMPCm</td>
<td>0.122 (1.03)</td>
<td></td>
<td>0.314 (1.55)</td>
<td></td>
</tr>
<tr>
<td>lnIM</td>
<td>−0.361 (−2.34)</td>
<td>0.164 (2.72)</td>
<td>0.023 (1.61)</td>
<td></td>
</tr>
<tr>
<td>lnMPC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>InRD_E</td>
<td>0.144 (3.42)</td>
<td></td>
<td></td>
<td>0.120 (2.42)</td>
</tr>
<tr>
<td>dlnRD_E</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&quot;R²&quot;</td>
<td>0.437</td>
<td>0.528</td>
<td>0.744</td>
<td>0.218</td>
</tr>
<tr>
<td>GMM C test for exogeneity</td>
<td>$\chi^2(1) = 3.84^*$</td>
<td>$\chi^2(1) = 4.20^*$</td>
<td>$\chi^2(2) = 3.70$</td>
<td>$\chi^2(1) = 0.04$</td>
</tr>
<tr>
<td>Robust F test for first-stage regressions</td>
<td>$F(4,83) = 8.85^*$</td>
<td>$F(2,88) = 41.67^*$</td>
<td>$F(5,86) = 13.63^*$</td>
<td>$F(5,86) = 5.57^*$</td>
</tr>
<tr>
<td>Hansen's J test for over-identifying restrictions</td>
<td>$\chi^2(3) = 4.98$</td>
<td>$\chi^2(1) = 0.01$</td>
<td>$\chi^2(3) = 1.80$</td>
<td>$\chi^2(2) = 2.16$</td>
</tr>
<tr>
<td>Additional instruments</td>
<td>AC, AC_1, InAREA, AW_1</td>
<td>AC_1 InRD_E_1</td>
<td>AC_1, AW_1</td>
<td>InAREA</td>
</tr>
</tbody>
</table>
Table 4 GMM-2SLS estimation results for country group = 2 (robust \( t \) values in parenthesis, \( N = 94 \), 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnHEc</th>
<th>dlnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>9.718 (6.45)</td>
<td>12.302 (10.30)</td>
<td>-2.038 (−1.48)</td>
<td>0.086 (3.17)</td>
</tr>
<tr>
<td>lnIM</td>
<td></td>
<td>-0.756 (−3.42)</td>
<td>0.075 (0.50)</td>
<td>-0.002 (−0.17)</td>
</tr>
<tr>
<td>GINI</td>
<td></td>
<td>-0.030 (−0.58)</td>
<td>0.014 (2.18)</td>
<td>-0.001 (−2.73)</td>
</tr>
<tr>
<td>GINI²</td>
<td></td>
<td>0.0001 (0.31)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP</td>
<td></td>
<td>11.40 (2.69)</td>
<td>0.003 (3.22)</td>
<td></td>
</tr>
<tr>
<td>lnPCRm_1</td>
<td>0.122 (1.03)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPRC</td>
<td>0.314 (1.55)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnRD_E_1</td>
<td>0.144 (3.42)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnRD_E</td>
<td>0.120 (2.42)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnGNIPc</td>
<td>-0.551 (−4.49)</td>
<td>0.967 (7.30)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnAW_1</td>
<td>-1.244 (−3.76)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnDOC</td>
<td>0.182 (3.30)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[ R^2 \] 0.437 0.528 0.744 0.218

GMM C test for exogeneity

\[ \text{lnIM} \chi^2(1) = 3.84^* \]
\[ \text{lnGNIPc} \chi^2(1) = 4.20^* \]
\[ \text{lnIM, lnGNIPc} \chi^2(2) = 3.70 \]
\[ \text{lnIM} \chi^2(1) = 0.04 \]

Robust \( F \) test for first-stage regressions

\[ \text{lnIM} F(4,83) = 8.85^* \]
\[ \text{lnGNIPc} F(2,88) = 41.67^* \]
\[ \text{lnIM} F(5,86) = 13.63^* \]
\[ \text{lnIMPCm} F(5,86) = 5.57^* \]
\[ \text{lnIM} F(3,86) = 6.68^* \]

Hansen's \( J \) test for over-identifying restrictions

\[ \chi^2(3) = 4.98 \]
\[ \chi^2(1) = 0.01 \]
\[ \chi^2(3) = 1.80 \]
\[ \chi^2(2) = 2.16 \]

Additional instruments AC, AC_1, lnAREA, lnRD_E_1, AC_1, lnRD_E_1

---

Table 5 GMM-2SLS estimation results for country group = 3 (robust \( t \) values in parenthesis, \( N = 50 \), 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnHEc</th>
<th>dlnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>8.081 (5.19)</td>
<td>5.632 (5.18)</td>
<td>-0.228 (−0.27)</td>
<td>0.019 (0.12)</td>
</tr>
<tr>
<td>lnIM</td>
<td>-0.361 (−2.34)</td>
<td></td>
<td>0.164 (2.72)</td>
<td>0.023 (1.61)</td>
</tr>
<tr>
<td>GINI</td>
<td>0.047 (0.67)</td>
<td>0.061 (5.09)</td>
<td></td>
<td>-0.002 (−2.59)</td>
</tr>
<tr>
<td>GINI²</td>
<td>-0.0003 (−0.37)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP</td>
<td>9.412 (6.13)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP3_1</td>
<td></td>
<td>-0.313 (−2.30)</td>
<td>0.015 (0.43)</td>
<td></td>
</tr>
<tr>
<td>lnPCR_1</td>
<td>3.062 (1.09)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnRD_E</td>
<td>0.193 (4.03)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnRD_E</td>
<td></td>
<td></td>
<td></td>
<td>0.224 (1.03)</td>
</tr>
<tr>
<td>lnGNIPc</td>
<td></td>
<td>-0.642 (−5.68)</td>
<td>0.725 (9.21)</td>
<td></td>
</tr>
<tr>
<td>lnDOC</td>
<td>0.315 (5.00)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\[ R^2 \] 0.719 0.526 0.315 (5.00) 0.714

GMM C test for exogeneity

\[ \text{lnIM} \chi^2(1) = 0.002 \]
\[ \text{lnGNIPc} \chi^2(1) = 3.83^* \]
\[ \text{lnIM, lnGNIPc} \chi^2(2) = 3.96 \]
\[ \text{lnIM} \chi^2(1) = 3.98^* \]

Robust \( F \) test for first-stage regressions

\[ \text{lnIM} F(3,41) = 1.64 \]
\[ \text{lnGNIPc} F(7,43) = 395.81^* \]
\[ \text{lnIM} F(5,43) = 3.66^* \]
\[ \text{lnIMPCm} F(4,44) = 0.79 \]

Hansen's \( J \) test for over-identifying restrictions

\[ \chi^2(2) = 4.57 \]
\[ \chi^2(4) = 5.64 \]
\[ \chi^2(2) = 1.52 \]
\[ \chi^2(2) = 1.04 \]

Additional instruments AC, AC_1, AREA

---
### Table 6 GMM-2SLS estimation results for country group = 1 with endogenous GINI and GINI² (robust t values in parentheses, N=50, 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnHEc</th>
<th>dinGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>7.071 (0.97)</td>
<td>4.577 (2.60)</td>
<td>−3.747 (−1.29)</td>
<td>−0.616 (−2.67)</td>
</tr>
<tr>
<td>lnIM</td>
<td>−1.221 (−1.82)</td>
<td>0.894 (1.50)</td>
<td>0.097 (2.50)</td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>0.178 (0.67)</td>
<td>0.049 (1.75)</td>
<td>0.004 (2.50)</td>
<td></td>
</tr>
<tr>
<td>GINI²</td>
<td>−0.002 (−0.63)</td>
<td>4.030 (1.38)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dPOP</td>
<td>0.024 (−1.06)</td>
<td>0.028 (1.82)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnPCR_1</td>
<td>0.079 (1.68)</td>
<td>0.777 (2.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnRD_E1</td>
<td>−0.411 (−1.64)</td>
<td>0.777 (2.02)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnDOC</td>
<td>−0.155 (−0.45)</td>
<td>0.166 (0.85)</td>
<td>0.239 (2.36)</td>
<td></td>
</tr>
<tr>
<td>&quot;P&quot;</td>
<td>0.141</td>
<td>.</td>
<td>0.309</td>
<td>.</td>
</tr>
<tr>
<td>GMM C test for exogeneity</td>
<td>lnIM</td>
<td>lnGNIPc</td>
<td>lnIM, lnGNIPc</td>
<td>lnIM</td>
</tr>
<tr>
<td></td>
<td>chi²(1) = 1.11</td>
<td>chi²(1) = 1.88</td>
<td>chi²(2) = 2.24</td>
<td>chi²(1) = 4.35*</td>
</tr>
<tr>
<td></td>
<td>GINI, GINI²</td>
<td>GINI</td>
<td>GINI</td>
<td>GINI</td>
</tr>
<tr>
<td></td>
<td>chi²(2) = 0.04</td>
<td>chi²(1) = 3.44</td>
<td>.</td>
<td>chi²(1) = 7.46*</td>
</tr>
<tr>
<td>Robust F test for first-stage regressions</td>
<td>lnIM</td>
<td>lnGNIPc</td>
<td>lnIM, lnGNIPc</td>
<td>lnIM</td>
</tr>
<tr>
<td></td>
<td>F(4,42) = 1.83</td>
<td>F(7,40) = 43.17*</td>
<td>F(4,44) = 3.19</td>
<td>F(2,42) = 1.87</td>
</tr>
<tr>
<td></td>
<td>GINI</td>
<td>GINI</td>
<td>GINI</td>
<td>GINI</td>
</tr>
<tr>
<td></td>
<td>F(4,42) = 0.99</td>
<td>F(7,40) = 1.08</td>
<td>F(4,44) = 5.32</td>
<td>F(4,42) = 3.63*</td>
</tr>
<tr>
<td></td>
<td>GINI²</td>
<td>F(4,42) = 1.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hansen’s J test for over-identifying restrictions</td>
<td>lnIM</td>
<td>lnGNIPc</td>
<td>lnIM, lnGNIPc</td>
<td>lnIM</td>
</tr>
<tr>
<td></td>
<td>chi²(1) = 3.11</td>
<td>chi²(2) = 2.33</td>
<td>chi²(2) = 5.24</td>
<td>chi²(2) = 0.83</td>
</tr>
<tr>
<td></td>
<td>.</td>
<td>.</td>
<td>.</td>
<td>.</td>
</tr>
<tr>
<td>Additional instruments</td>
<td>AC, AC_1, lnAREA, AW_1</td>
<td>AC, AC_1, lnGDP_iv, lnGDP_iv_1, dinGDP_iv_1, lnAREA, dinRD_E_1</td>
<td>AC_1, AW_1</td>
<td>AC, AC_1, AW_1, lnAREA</td>
</tr>
</tbody>
</table>
Table 7 GMM-2SLS estimation results for country $\text{group} = 2$ with endogenous $\text{GINI}$ and $\text{GINI}^2$ (robust $t$ values in parenthesis, $N = 93$, 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnHEc</th>
<th>dlnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>8.084 (1.90)</td>
<td>10.877 (5.43)</td>
<td>-2.038 (-1.48)</td>
<td>0.059 (0.62)</td>
</tr>
<tr>
<td>lnIM</td>
<td>-0.611 (-2.13)</td>
<td>0.075 (0.50)</td>
<td>0.001 (0.01)</td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>-0.019 (-0.11)</td>
<td>0.050 (2.39)</td>
<td>-0.001 (-0.29)</td>
<td></td>
</tr>
<tr>
<td>GINI$^2$</td>
<td>0.000 (0.47)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPOP</td>
<td>5.978 (1.07)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnPOP1</td>
<td>0.097 (0.62)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnPCR_1</td>
<td>0.097 (0.62)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnPCR</td>
<td>0.184 (3.13)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnRD_E_1</td>
<td>0.277 (1.29)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dlnRD_E</td>
<td>0.125 (1.94)</td>
<td></td>
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</tr>
<tr>
<td>lnGNIPc</td>
<td>-0.427 (-3.92)</td>
<td>0.967 (7.30)</td>
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</tr>
<tr>
<td>lnAW_1</td>
<td>-1.517 (-3.94)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnDOC</td>
<td>0.182 (3.30)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*R²*

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnHEc</th>
<th>dlnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>0.157</td>
<td>0.336</td>
<td>0.744</td>
<td>0.211</td>
</tr>
<tr>
<td>lnIM</td>
<td>0.000 (2.81)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>0.050 (2.39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GINI$^2$</td>
<td>0.000 (0.01)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnIM, lnGNIPc</td>
<td>0.000 (2.39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnIM, lnGNIPc</td>
<td>0.000 (0.01)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>lnIM</td>
<td>0.000 (2.39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>0.000 (2.39)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Robust $F$ test for first-stage regressions

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnIM</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>lnIM</td>
<td>F(4,85) = 8.78*</td>
<td>F(5,86) = 72.00*</td>
<td>F(5,86) = 13.63*</td>
<td>F(4,85) = 19.82*</td>
</tr>
<tr>
<td>lnGNIPc</td>
<td>F(4,85) = 1.10</td>
<td>F(5,86) = 2.57*</td>
<td>F(5,86) = 5.58*</td>
<td>F(4,85) = 1.41</td>
</tr>
</tbody>
</table>

Hansen’s $J$ test for over-identifying restrictions

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnIM</th>
<th>lnGNIPc</th>
<th>lnIM</th>
<th>lnGNIPc</th>
</tr>
</thead>
<tbody>
<tr>
<td>lnIM</td>
<td>chi$^2$(1) = 1.82</td>
<td>chi$^2$(3) = 4.16</td>
<td>chi$^2$(3) = 1.80</td>
<td>chi$^2$(2) = 2.08</td>
</tr>
<tr>
<td>lnGNIPc</td>
<td>chi$^2$(1) = 2.29</td>
<td>chi$^2$(3) = 4.16</td>
<td>chi$^2$(3) = 1.80</td>
<td>chi$^2$(2) = 2.08</td>
</tr>
</tbody>
</table>

Additional instruments

| AC, AC_1, lnAW_1, lnAREA | lnAREA, lnAC_1, lnRD_E_1, lnGDP_iv_1, dlnGDP_iv_1, lnPOPU, AC, ACm_1, AW_1 | AC, AC_1, lnAW_1, lnAREA | AC, AC_1, lnAW_1, lnAREA |
Table 8 GMM-2SLS estimation results for country group = 3 with endogenous GINI and GINI² (robust t values in parenthesis, N=50, 5% or less significance level results with italics, 5% or less significance level test values are indicated by *)

<table>
<thead>
<tr>
<th>Variable</th>
<th>lnGNIpc</th>
<th>lnIM</th>
<th>lnHec</th>
<th>dlnGNIpc</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>−63.463 (−0.17)</td>
<td>4.898 (3.69)</td>
<td>−0.228 (−0.27)</td>
<td>0.049 (0.40)</td>
</tr>
<tr>
<td>lnIM</td>
<td>−3.368 (−0.22)</td>
<td>0.164 (2.72)</td>
<td>0.007 (0.38)</td>
<td></td>
</tr>
<tr>
<td>GINI</td>
<td>3.409 (0.19)</td>
<td>0.077 (4.32)</td>
<td>−0.000 (−0.08)</td>
<td></td>
</tr>
<tr>
<td>GINI²</td>
<td>−0.034 (−0.19)</td>
<td>3.624 (0.14)</td>
<td>0.246 (−2.10)</td>
<td></td>
</tr>
<tr>
<td>dlnPOP</td>
<td>−3.624 (−0.14)</td>
<td>2.127 (0.08)</td>
<td>0.002 (−0.07)</td>
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<tr>
<td>dlnPOP3_1</td>
<td></td>
<td></td>
<td>0.130 (1.03)</td>
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</tr>
<tr>
<td>lnPCR_1</td>
<td></td>
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<td>0.246 (−2.10)</td>
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<td>lnPCR_1</td>
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<td>−0.002 (−0.07)</td>
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<td>lnRD_E</td>
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<td>0.130 (1.03)</td>
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<td>lnRD_E</td>
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<td>0.246 (−2.10)</td>
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<tr>
<td>lnGNIpc</td>
<td>−0.637 (−5.23)</td>
<td>0.725 (9.21)</td>
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<td>lnAW</td>
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<td>lnPOP_U</td>
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<td>lnDOC</td>
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<td><em>p</em>²</td>
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<td>0.149</td>
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<td>GAW C test for exogeneity</td>
<td>lnIM</td>
<td>lnGNIpc</td>
<td>lnIM, lnGNIpc</td>
<td>lnIM, lnGNIpc</td>
</tr>
<tr>
<td>GINI, GINI²</td>
<td>χ²(1) = 2.19</td>
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<td>Robust F test for first-stage regressions</td>
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<td>lnGNIpc</td>
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<tr>
<td>lnIM, lnGNIpc</td>
<td>F(4,42) = 1.29</td>
<td>F(4,42) = 1.72</td>
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<td>F(4,42) = 3.66</td>
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<td>lnIM, lnGNIpc</td>
<td>F(4,42) = 3.66</td>
<td>F(4,42) = 3.66</td>
<td>F(4,42) = 3.66</td>
<td>F(4,42) = 3.66</td>
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<td>Hansen’s J test for over-identifying restrictions</td>
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<td>χ²(1) = 0.04</td>
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<td>χ²(1) = 0.04</td>
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<td>Additional instruments</td>
<td>AC, AC_1, lnAW_1, lnAREA</td>
<td>dlnGDP, lnGDP_1, AC, AC_1, lnPOP_1, lnPOPU_1</td>
<td>dlnGDP, lnGDP_1, AC, AC_1, lnPOP_1, lnPOPU_1</td>
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Thesis analyzes the relationships between health status, health expenditures, health care technology, economic growth, and inequality on the global scale using econometric methods. Results show that at present poorest countries' income gradient is still high, public health expenditures are more health promoting than private spending, the Kuznets' hypothesis is valid in poor countries, and cancer mortality is less responsive than tuberculosis to global diffusion of health care technologies.