Universität St.Gallen

Health and Economic Development - Evidence from the Introduction of Public Health Care

Anthony Strittmatter, Uwe Sunde

August 2011 Discussion Paper no. 2011-32

School of Economics and Political Science,
Department of Economics

University of St. Gallen

Editor: Martina Flockerzi
University of St. Gallen
School of Economics and Political Science
Department of Economics
Varnbüelstrasse 19
CH-9000 St. Gallen
Phone +41 71 224 23 25
Fax +41 71 224 31 35
Email seps@unisg.ch

Publisher: School of Economics and Political Science
Department of Economics
University of St. Gallen
Varnbüelstrasse 19
CH-9000 St. Gallen
Phone +41 71 224 23 25

Electronic Publication: Fax +41 71 224 31 35
http://www.seps.unisg.ch

Health and Economic Development - Evidence from the Introduction of Public Health Care[1]

Anthony Strittmatter[2], Uwe Sunde[3]

Author's address:

Anthony Strittmatter
University of St.Gallen
Varnbüelstrasse 14
CH-9000 St.Gallen
Email anthony.strittmatter@student.unisg.ch

Uwe Sunde
University of St.Gallen, SEW-HSG
Varnbüelstrasse 14
CH-9000 St.Gallen
Phone +41 71 224 23 29
Email uwe.sunde@unisg.ch

[1] The authors would like to thank Davide Cantoni, Bernd Fitzenberger, Volker Grossmann, Michael Lechner, Jan Schuhmacher, Rudi Stracke, Thomas Vischer and seminar participants at the University of St.Gallen, and the annual meeting of the Swiss Economics Association for helpful comments.

[2] University of St.Gallen, and Albert-Ludwigs-University Freiburg.
[3] University of St. Gallen, IZA Bonn, and CEPR, London.

Abstract

This paper investigates the causal effect of changes in health on economic development using a long panel of European countries. Identification is based on the particular timing of the introduction of public health care systems in different countries, which is the random outcome of a political process. We document that the introduction of public health care systems had a significant immediate effect on the dynamics of infant mortality and crude death rates. The findings suggest that a reduction in infant mortality or crude death rates exhibited a positive effect on growth in income per capita and increased population growth.

Keywords

Mortality, Economic Development, Growth, Public Health Care.

JEL Classification

I10, J10, O11, N13

1 Introduction

The causal effect of general health conditions on economic performance is intensely debated in the literature. Arguably, health, reflected by mortality of infants and adults, affects economic performance through human capital investments, physical capital accumulation, population growth, productivity and female labor force participation. The main challenge for the identification of the total effect is the problem of reverse causality, since mortality is likely to be affected by economic development, for example, because rich countries can afford better health systems.

The existing literature has used two different instrumental variable strategies to circumvent the reverse causality problem and identify the causal effect of longevity on economic growth. Most of the literature, including recent work by Lorentzen, McMillan, and Wacziarg (2008) has used exogenous variation across countries, such as climatic factors, geographical features, or disease indices, as instruments for differences in life expectancy across countries, and has found positive effects of longevity on growth. Recent research, starting with the contribution of Acemoglu and Johnson (2007) has exploited within-country variation by applying time varying instruments to identify the causal effect of life expectancy on economic growth, and has found mixed or even negative effects.

In this paper, we aim to contribute to this literature by estimating the causal effect of mortality growth on economic growth using a novel identification strategy that exploits within country variation in a long-panel of 12 European countries over the period 1820 to 2010. In particular, we apply an instrumental variable approach that exploits variation in the introduction dates of universal public health care. Universal public health care systems in terms of the introduction of a public health insurance or the public payment of subsidies for health services imply (potential) coverage of the entire population. In the sample, we adopt a rather broad concept of universal public health care systems reflected by the introduction of access to health care for all people in need for health care, independent of their individual income. The novel feature of the identification strategy is its reliance on the particular timing of the implementation in each country, rather than on

the implementation per se. While the implementation of public health care might be related to the level of economic development, the particular year in which the implementation takes place is largely random, since the implementation is typically the outcome of a lengthy political process with substantial uncertainty. In light of this fact, we apply a timing of events methodology, in which identification is driven by within country variation in mortality around the period of implementation, which can be used to identify causal effects on economic development in that time period.

The empirical results indicate that the introduction of public health systems significantly reduced infant mortality and crude death rates, which are proxies for an improvement in overall health conditions. The second stage estimates provide evidence for a significant positive effect of overall health conditions on economic growth as well as on population growth.

These findings complement and qualify the existing estimates in the literature in several dimensions. Using cross country data from the World Health Organization (WHO), the United Nations (UN) Population Division and the World Bank, the empirical literature typically finds that an increase in adult mortality substantially reduces GDP per capita growth. Lorentzen et al. (2008) for example, find that an increase in adult mortality of one standard deviation reduces growth by 1.1 percentage points, mainly through the physical capital and fertility channel. This line of research cannot account for unobserved heterogeneity across countries or exploit health dynamics, since the instruments are constant over time, however. Noting this, Acemoglu and Johnson (2007) use panel data for 47 countries from the League of Nations, the WHO and the UN, and exploit the drop in mortality from specific infectious diseases due to the international epidemiological transition as instrument for the change in life expectancy. This identification makes use of the fact that the mortality rate from these diseases was exogenous in 1940, because no treatments, medication or vaccines were available before that time. By 1980, on the other hand, all these diseases could be treated or prevented in all countries due to medical advances and international organizations such as the WHO. The findings suggest a positive but insignificant

effect of life expectancy on aggregate GDP, and a positive significant effect on population growth. The total effect on GDP per capita is negative.

This finding led to controversial discussions about the identifying assumptions that drive the results. Bloom, Canning, and Fink (2009) argue that mortality from the specific diseases in the instrument by Acemoglu and Johnson was not exogenous in 1940 as countries like the United States had reduced their disease burden, e.g., from malaria, before 1940, and given the evidence that mortality from infectious diseases in the United States had peaked around 1900 as shown by Cutler, Deaton, and Lleras-Muney (2006). Acemoglu and Johnson (2009) clarify their identifying assumptions, leaving open whether the different results in the literature are driven by the different identification assumptions, or by the different sample compositions in terms of countries and observation period, as suggested by Angeles (2010) and Cervellati and Sunde (2011).

The findings presented in this paper indicate that the positive effect of life expectancy on growth found in cross-country studies is not necessarily due to the use of time-invariant instruments. Second, the findings support the evidence of Cervellati and Sunde (2011) that the different results in the literature might be driven by differences in sample composition. Third, the findings suggest that improving health conditions might have a substantial effect on economic performance, and that public health policy and the institutional environment might play an important role for economic development.

The remainder of the paper is structured as follows. Section 2 presents and discusses the identification strategy and describes the data. The main results are presented in Section 3, and results from additional robustness checks are discussed in Section 4. Section 5 concludes.

2 Identification and Data

2.1 Identification Strategy

The identification strategy uses the exact date of implementation of a universal public health system as exogenous variation. In particular, the identification

exploits the effect of the implementation of a public health system on within-country variation in health, to identify the effect of variation in health conditions on economic growth. Of course, the implementation and existence of a universal public health care system is influenced by the initial economic situation. Poor countries might not be able to afford the costs of a public health system, while rich countries can afford them. However, we argue that the *timing* of the introduction is exogenous and driven by many complex political processes that are unrelated to current economic performance. Due to the small sample size, it is possible to identify the driving political forces behind the implementation of a public health system and to investigate the plausibility of the identification assumption of the exogeneity of the implementation date in detail.

As mentioned in the introduction, we use a broad concept of universal public health systems. According to Mackenbach (1996), the introduction of universal public health systems improves life expectancy especially for children and less endowed individuals who, in contrast to rich individuals, cannot afford the contributions to a private insurer. Upon their introduction, many universal public health systems indeed only cover a small fraction of the population, often just children and very needy people. This implies that the introduction of a public health system can be expected to affect overall health in different ways. To capture this, we use infant mortality as well as the crude death rate as different proxy measures of longevity and health.

A first indication of exogeneity of the implementation date is the randomness of the implementation process. For example, in the Netherlands several attempts to introduce a universal public health care system failed. Finally, the first universal public health insurance system was introduced under the German occupation in 1941. Other countries which where also under German occupation did not introduce a universal public health care system during World War II. In Spain, the Franco Regime introduced the first universal public health care system in 1942. Earlier attempts to introduce a public health care system failed, because there was no majority in parliament. We therefore think that the implementation dates are driven by events which have a high degree of uncertainty and are difficult to

predict. In particular, we think that there are good reasons to assume that the implementation dates are exogenously determined and not driven by economic growth.[1]

A second indication for the validity of this identification strategy is evidence suggesting that the introduction of a universal public health care system took many years and that the timing was heavily influenced by the political regime. In an autocratic regime, like in Italy at the time when the health system was implemented, the government could decide about a public system by itself. In a democracy, a majority in the parliament or in a referendum is required. This takes typically much longer. For example, in Switzerland the first referendum was rejected in 1899. Only 12 years later the health insurance law was finally passed by another referendum. Evidence strongly suggests that the introduction dates depend on the type of government. Lindert (2004) distinguishes between elite democracies and full democracies.[2] He argues that elite-democracies are less willing to set up government financed social programs, in comparison to full democracies. In monarchies like Austria, social insurance systems were introduced relatively early in order to reduce the power of the socialists.

A third factor is the type of health care provider in place before the introduction of a universal public health system. Private insurers typically oppose a public insurance scheme. Especially in the Netherlands, they prevented the introduction of an early public health system for a long time. In other countries like Denmark or Finland, corporate groups or municipalities were responsible for early health care. They typically supported the introduction of a universal public health system, speeding up the implementation in an international comparison.

Another important factor is the type of public insurance which is introduced. We do not distinguish the introduction of a health system that covers the entire population from the beginning, from the introduction of subsidies for health services only for a specific subpopulation of needy people. When an adequate insurance scheme is already in place before public health care, the government can

[1] A historical review of public health insurance for all relevant countries can be found in the appendix.

[2] Elite democracies are political systems with property requirements for franchise, which excluded large parts of the population from the voting process.

simply pay subsidies to these institutions, as was the case in Belgium and Denmark. The payment of subsidies typically covers only very needy people and is therefore less expensive. Laws for such systems can pass the parliament more easily than a more comprehensive health law like in the UK, where the public health insurance scheme covers the entire population, from the first day of introduction. Not distinguishing among the different types of public health care systems might constitute a problem because it potentially weakens the link to health outcomes. On the other hand, the consideration of different types of public health care implies a large degree of randomness in the timing of the implementation.

Thus, the introduction dates of a universal public health care system are influenced by factors that can either be controlled for, or which are (mean) independent of the economic situation, so that the dates of implementation can be assumed to be plausibly exogenous for the purpose of this paper. (Note that this does *not* imply that the fact that a public health care system is eventually implemented is independent of the economic situation.) The exclusion restriction (that the implementation of a public health system affects economic growth in the intermediate aftermath of the implementation only through effects on public health conditions) appears plausible, in particular in light of the fact that we consider growth rates rather than levels in our outcome specifications. If anything, the identifying assumption is conservative, since less developed countries can also have high income or population growth rates (e.g., during a convergence process or the demographic transition). It is therefore unlikely, that the growth rate has an influence on the introduction date of a public health care system, especially when one takes into account the fact that such an introduction takes many years. Moreover, since under- and less-developed countries might simply not be able to afford the costs of a public health care system, we consider in our sample only relatively developed countries (see Table 23 in the Appendix). We argue that during the entire observation period, each of the countries in our sample could in principle afford the costs of a public health system.

2.2 Data

We use data for 12 Western European countries over the period 1820 until 2010. The data on GDP per capita, population size and GDP are collected from Maddison (2006). This data is available on a yearly basis and goes back to 1820. The infant mortality rate and the crude death rate are taken from Flora, Kraus, and Pfenning (1987). They provide data for 13 Western European countries from 1815 until 1975.[3] We do not consider Ireland and Germany, because of too many missing observations and major territorial changes.[4] Additional data for Spain is collected from Mitchell (1992). In case of missing observations we use data from Mitchell (1992) until 1988 and after that we use the mortality rates from the OECD Health Data (updated June 20, 2010). The infant mortality rate is defined as deaths under the age of one per 1,000 live births, i.e., stillbirths are excluded. The crude death rate is the number of deaths per 1,000 persons. The dates for the introduction of a public health system in the 12 countries are reported in Table 23. Detailed information about the history of universal public health care systems can be found, for each country separately, in the appendix. As additional controls, we use variables which approximate the political institutions in terms of democratization of a country, reflected by a dummy that takes value 1 after the first observation of election rules in a specific country, as well as a variable that reflects the age of these rules. Dates for the first year of election are collected from Persson and Tabellini (2003). Additionally, we use the political regime characteristics and transitions (1800-2009) from the Polity IV project. From this index we create indicators for autocracies, anocracies and democracies, as suggested by Gurr (1974). In order to account for the structural change we condition on the share of agriculture, industry (manufacturing) and services as fraction of total GDP from Flora (1983).[5] Moreover, we collect the government expenditure as share of total GDP, the number of labor disputes (per million workers), the number of workers involved in labor disputes (per million workers),

[3]For the UK we use own calculations of the mortality rates as weighted average of the mortality rates from England, Wales, Scotland and North Ireland.

[4]However, the main results do not change when we include these countries.

[5]Missing observations are from Mitchell (1992) and from OECD Annual National Accounts (Volume 2).

number of days lost in labor disputes (per million workers), and gross capital formation as share of total GDP from Mitchell (1992). Missing data for government expenditure and gross capital formation is completed with data from the OECD Annual National Accounts (Volume 2) and additional data about labor disputes is collected from the ILO Department of Statistics.

Using this information, we create an unbalanced data set with a 20-yearly frequency from 1850 until 2008 as described in the next section.[6] In the baseline specification we have 84 observations. Descriptive statistics can be found in Table 3. We use discrete growth rates in our estimation, because the mortality growth rates are typically negative.[7] The infant mortality growth is between -75% and 37%. On average the growth rate is -36%. The growth in the crude death rate exhibits less variation. It is on average -11% and lies between -50% and 33%. GDP per capita growth is on average 49% over a 20-year period, which implies an annual growth rate of about 2%.[8] The population growth is on average 14% and the average aggregate GDP growth amounts to 70% during 20 years.

2.3 Estimation Strategy

In order to exploit the exact introduction dates, we construct the panel data as follows. In addition to calendar time τ we create a synthetic time variable t, which is normalized to be equal to zero in the 20-year period after the introduction of a universal public health care system. We then calculate the growth rates for each country and 20-year time period on the basis of the synthetic time frame. Figure 1 illustrates that the exact years for which the growth rates are calculated are

[6] In earlier specifications we also used a balanced data set for which the number of observations is much smaller. The main results are identical.

[7] The growth rate of variable $x_{i,t}$ is calculated as

$$\Delta x_{i,t} = \frac{x_{i,t} - x_{i,t-1}}{x_{i,t-1}},$$

where i denotes the country and t the time dimension. The results for log-differences are qualitatively equal and quantitatively larger, but the calculation in log-growth rates would potentially overestimate the true values as it delivers negative growth rates exceeding -100%. The results also do not change when we use average yearly growth rates.

[8] There are some observations of very high growth rates, in particular for Austria, which exhibits growth of up to 283% during one 20-year window (almost 7% per year). In robustness checks we excluded Austria. The results remain robust and qualitatively unchanged.

different for each country. For example, in Switzerland, where the public health system was implemented in 1911, we compute the growth rates from 1891 to 1911, from 1911 to 1931, and so on. In the Netherlands, where the public health system was implemented in 1941, we calculate the growth rates from 1921 to 1941 and from 1941 to 1961 and so on. Accordingly, the introduction dates correspond to time $t = -1$ (minus 20 years) of this synthetic time index. As consequence, for each country the exact years over which the growth rates are computed are different. This procedure has two advantages. First, this approach calculates the exact growth rates directly after the introduction of the public system on a yearly basis. Second, it helps to disentangle common time factors, since we do not calculate the growth rates for exactly the same years, even though there is some overlap. In the next step we construct an instrument for the introduction of a universal public health care system, which is equal to 1 at time $t = 0$ of the synthetic time frame, and 0 otherwise (see Figure 2). This means that the instrument is only equal to 1 immediately after the introduction of the public health care system. This is a precise way to identify the timing of the introduction, and hence its effect on health. This construction is conservative, as long run effects are not captured by this strategy. Likewise, this identification strategy avoids that the results take up long run time trends or shocks, which are unrelated to the introduction of public health insurance. Overall, exploiting a one-off variation in public health systems and constructing the data around the implementation date suggests that the exclusion restriction that the implementation of a health system affects growth only through improvements in health (conditional on other control variables) is plausibly satisfied.

We are interested in estimating the causal effect of changes in mortality, measured by infant mortality or crude death rates, on GDP per capita growth, population growth and aggregate GDP growth, respectively. While all variables are observed in calendar time τ, they are coded in terms of the synthetic time index t. Consequently, all variables are indexed by the subscript i indicating the country, subscript τ indicating the year of observation, and t indicating the 20-year period in terms of the synthetic time index. Since the empirical analysis is conducted on

the basis of a 20-year frequency, τ and t essentially contain the same information, such that we can limit the notation to the synthetic time index t. The respective outcome variables (GDP per capita growth, population growth, and aggregate GDP growth) are denoted by $\Delta y_{i,t}$ for $i = 1, ..., N$ and $t = 1, ..., T$. The variable $\Delta m_{i,t}$ corresponds to the change in mortality, either in terms if the infant mortality or the crude death rate. Additional lagged control variables are denoted by the vectors $x_{1,i,t-1}$ and $x_{2,i,t-1}$. Let $s_{i,t}$ be a selection indicator, where $s_{i,t} = 1$ if $\{\Delta y_{i,t}, \Delta m_{i,t}, x_{1,i,t-1}, x_{2,i,t-1}\}$ are jointly observed in a particular country and observation period, and zero otherwise.[9] Then Δy and Δm are two $1 \times S$ vectors, where number of observations are indicated by S, with $S = \sum_{i=1}^{N} \sum_{t=1}^{T} s_{i,t}$. X_1 and X_2 are two $k_j \times S$ matrices (with $j \in 1, 2$), involving k_j lagged control variables, including deterministic time patterns and country specific intercepts.[10] The regression model is specified as

$$\Delta y = \widehat{\alpha} \Delta m + \widehat{\beta}_1' X_1 + \widehat{\beta}_2' X_2 + \widehat{u},$$

where the residual vector $\widehat{u}$ has an expected value $E[\widehat{u}] = 0$. The parameter $\widehat{\beta}_j$ is a $k_j \times 1$ vector (with $j \in 1, 2$) and the coefficient $\widehat{\alpha}$ is a scalar. The estimated parameter of interest is $\widehat{\alpha}$, which has no causal interpretation in this simple setting in light of the potential problem of reverse causality.

In order to identify causal effects, we use the introduction of a universal public health care system ($z_{i,t} \equiv \Delta Ins_{i,t-1}$) as instrument for infant mortality growth and crude death growth as illustrated in Figure 2. The identification of causal effects is based on assumptions about the instrument, which hold jointly conditional on the control variables X_1 and X_2. First, we assume that the outcome Δy has no systematic influence on the instrument z. Second, the instrument has only an indirect effect on the outcome variables, i.e., the introduction of universal health services improves the health status Δm and via this channel the expected out-

[9] Only periods which are observed in the unbalanced panel are considered in the following.

[10] Deterministic time patterns, such as trends or period dummies, are coded in synthetic time t but exploit variation in calendar time τ. For example, period dummies are coded as 1 if the respective period t falls in the corresponding decade of calendar time, and zero otherwise. Likewise, period trends are coded according to the respective calendar time period that corresponds to the particular t of a given country-year observation.

come, alternative channels do not exist. These two assumptions are not testable, and we can only use economic arguments and plausibility tests to justify that they are satisfied. The third assumption, which states that the instrument has sufficient power to influence the endogenous variable, can be tested. In light of modest unconditional effects of the introduction of an universal public health care system on mortality growth (see Figure 3), this instrument could potentially have little power. In order to avoid weak instrument problems in the identification of the effect of interest, we use interactions between the instrument and other control variables, involved in the matrix X_1, as additional instruments.[11] This allows for more exogenous variation, since the introduction of a public health care system can have heterogenous effects with respect to the initial economic situation. The matrix X_2 is not interacted in order to avoid problems with too many instruments providing little additional explanatory power. The first stage regression is given by

$$\Delta m = \widehat{\gamma}_1 z + \widehat{\gamma}_2'(X_1 \cdot diag(z'z)) + \widehat{\delta}_1' X_1 + \widehat{\delta}_2' X_2 + \widehat{v},$$

where the instrument z (dimension $1 \times S$) is equal to one when a public health insurance is introduced in $t-1$ and zero otherwise. The estimated residual vector is denoted by $\widehat{v}$, with $E[\widehat{v}] = 0$. The parameter $\widehat{\gamma}_2$ is a $k_1 \times 1$ vector, the parameter $\widehat{\delta}_j$ is a $k_j \times 1$ vector (with $j \in 1, 2$) and the coefficient $\widehat{\gamma}_1$ is a scalar.

In the second stage we use the predicted mortality growth ($\widehat{\Delta m}$) as regressor,

$$\Delta y = \widetilde{\alpha}\widehat{\Delta m} + \widetilde{\beta}_1' X_1 + \widetilde{\beta}_2' X_2 + \widetilde{\varepsilon},$$

where the residual vector is represented by $\widetilde{\varepsilon}$, with $E[\widetilde{\varepsilon}] = 0$. The parameter $\widetilde{\beta}_j$ is a $k_j \times 1$ vector (with $j \in 1, 2$) and the coefficient $\widetilde{\alpha}$ is a scalar. The coefficient $\widetilde{\alpha}$ represents the estimated causal effect of interest. This setup makes implicit functional form assumptions, in terms of assuming linearity. Even though we do not think this parametric assumption reflects the reality exactly, we think it is a

[11] Since we condition on X_1, the identifying assumptions are also valid for the interaction between the instrument z and X_1, if z is exogenous conditional on X_1 and X_2 (see e.g., Angrist and Pischke, 2008).

good approximation for reality. Moreover, the specifications estimated correspond to the canonical empirical growth model as used by, e.g., Barro (1991), Durlauf, Johnson, and Temple (2005) and Acemoglu and Johnson (2007).

2.4 Preliminary Analysis and Reduced Form Effects

In Table 1, we show descriptive statistics for periods when the public health systems are unchanged and for periods when a universal public health care system is introduced, separately. The lagged level of GDP per capita, which is an indicator for the initial economic situation, is lower on average in periods when the public health system has changed. Accordingly, we do not find a systematic positive correlation between the introduction of public health systems and the level of economic development. This is also confirmed when considering the economic and demographic growth rates. Only population growth is on average slightly higher in periods when a public health care system is introduced. We do not find evidence that public health care systems are introduced systematically in periods of fast economic or demographic growth. This supports the argument that the exact timing of the introduction of a universal public health care system is determined exogenously and not driven systematically by initial economic or demographic growth rates.

Table 1: Summary statistics separated for periods when public health care system is implemented.

Summary Statistics					
Periods when Public Health Care System is unchanged ($\Delta Ins_{i,t} = 0$)					
	20-Year Frequency				
Variable	Obs	Mean	Std. Dev.	Min	Max
$gdpc_{i,t}$	72	8430.211	6952.132	1206.913	23912.280
$\Delta gdpc_{i,t}$	72	0.508	0.428	-0.382	2.830
$\Delta pop_{i,t}$	72	0.138	0.080	0.022	0.336
$\Delta gdp_{i,t}$	72	0.717	0.496	-0.349	3.053
Periods when Public Health Care System is Implemented ($\Delta Ins_{i,t} = 1$)					
	20-Year Frequency				
Variable	Obs	Mean	Std. Dev.	Min	Max
$gdpc_{i,t}$	12	3875.220	1433.479	2126.270	6745.636
$\Delta gdpc_{i,t}$	12	0.345	0.225	-0.069	0.657
$\Delta pop_{i,t}$	12	0.175	0.082	0.009	0.295
$\Delta gdp_{i,t}$	12	0.571	0.229	0.127	0.891

Table 2: Logit regression of the dummy for the introduction of a universal public health care system on GDP per capita growth, population growth, GDP growth and control variables in levels.

	Instrument (ΔIns$_{i,t}$)					
	20-Year Frequency					
	(1)	(2)	(3)	(4)	(5)	(6)
$\Delta gdpc_{i,t}$	-1.563	-0.835				
	[0.958]	[1.023]				
$\Delta pop_{i,t}$			4.811*	4.315		
			[2.747]	[3.465]		
$\Delta gdp_{i,t}$					-0.962	-0.481
					[0.608]	[0.627]
$lgdpc_{i,t}$		-0.508**		-0.655***		-0.577**
		[0.243]		[0.220]		[0.236]
$lpop_{i,t}$		-0.057		0.103		-0.068
		[0.068]		[0.115]		[0.074]
Observations	84	84	84	84	84	84

Results from Logit regressions the dependent variable is the instrument, $\Delta Ins_{i,t}$. Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

As plausibility test, we regress a dummy for the present introduction of an universal public health care system ($\Delta Ins_{i,t}$) on GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively.[12] In order to account for deviations from the long-run equilibrium we condition on log GDP per capita ($lgdpc_{i,t}$) and log population size ($lpop_{i,t}$), following the suggestions of Durlauf, Johnson, and Temple (2005). The results in Table 2 show insignificant coefficients for the economic growth rates. Only for population growth we find a weak influence on the instrument. This influence disappears as soon as we condition on the initial economic situation. All other growth rates have an insignificant influence and rather high standard errors. Accordingly, the economic and demographic growth rates have no predictive power for the introduction of a universal public health care system. As expected, the initial GDP per capita has some predictive power on the introduction dates. In the final specifications we do not use the present but the lagged introduction of an universal public health care system as instrument ($z_{i,t} = \Delta Ins_{i,t-1}$). Therefore, we are confident that our identification strategy is not prone to reverse causality.

In Figure 3 we plot the growth rates in the different dimensions (mortality, income, population) against the time before and after the introduction of a uni-

[12]Since the instrument is a binary outcome variable, we use a non-linear Logit specification.

versal public health system. The introduction of a universal public health care system occurs at time $t = -5$ years.[13] In this figure we calculate average five-year growth rates, which are indicated by the dots. We regress the growth rates on a constant, a time trend and allow for different coefficients before and after the introduction dates. The predicted growth rates are indicated by the solid line, the 90% confidence interval is within the dashed lines. Figure 3(a) depicts the growth in infant mortality. After the introduction of a universal public health system, the predicted infant mortality growth is 5.7 percentage points lower. This difference is only significant at the 5%-level, however. The predicted growth of crude death rates is not significantly different after the introduction compared to before as indicated by Figure 3(b). However, by looking at the average observed values, we find that the absolute change in crude death rates is substantially lower in the first decade after the introduction and increases only thereafter. Figure 3(c) plots the predicted growth in GDP per capita; this series shows a large discontinuity after time zero. After the introduction, the predicted growth rate is 4.6 percentage points higher. There is no significant difference in the predicted population growth rates as depicted in Figure 3(d). After the introduction, the dynamics of population growth change strongly, however. Aggregate GDP growth increases significantly after the introduction of a public health care system, as shown by Figure 3(e). The predicted value is 5.5 percentage points higher after the introduction.

3 Empirical Findings

3.1 Baseline Specifications

As first step of the analysis, we present the correlations between economic and demographic development and mortality growth that are obtained from OLS regressions. Tables 4 and 5 present results from regressions of the outcomes (GDP per capita growth, population growth and aggregate GDP growth) on the

[13] In order to have more observations when displaying the reduced form effects, we use a shorter time period than 20 years in this plot.

measures of mortality change (infant mortality and crude death rates, respectively). As suggested by Durlauf, Johnson, and Temple (2005) we condition in all specifications on the lagged log GDP per capita ($lgdpc_{i,t-1}$) and the lagged log population size ($lpop_{i,t-1}$). This specification allows us to account for deviations from long-run equilibrium. Moreover, we condition on a lagged dummy for countries that have completed their demographic transition in the respective period ($PostTrans_{i,t-1}$), based on evidence by Cervellati and Sunde (2011) that the level of demographic development is an important factor for the effect of life expectancy on growth.[14] We distinguish between four specifications. In the first specification we include a linear year trend. In the second specification, we allow for country specific linear year trends. In the third specification, we include period fixed effects. In the fourth specification, we include a linear time trend as well as country-specific intercepts (fixed effects).[15] We find that a one percentage point reduction in infant mortality growth ($\Delta imr_{i,t}$) increases GDP per capita growth ($\Delta gdpc_{i,t}$) by about 0.6 percentage points, has no significant effect on population growth ($\Delta pop_{i,t}$), and reduces GDP growth ($\Delta gdp_{i,t}$) by about 0.8 percentage points. According to Table 5, a one percentage point reduction in crude death growth ($\Delta cdr_{i,t}$) has no or only a marginally significant positive effect on GDP per capita growth, increases population growth by about 0.1-0.2 percentage points, and has a positive effect on GDP growth in the specification with period fixed effects. The effects vary substantially across the different model specifications. Since we expect reverse causality and a negative relationship between economic and demographic growth and mortality growth, we suspect that the coefficients have a positive bias.[16] The causal coefficients are expected to be

[14] Following Chesnais (1992), we define a country to be post-transitional when the crude death rate is lower than a certain threshold,

$$PostTrans_{i,t-1} = \begin{cases} 1 & \text{when } cdr_{i,t-1} \leq \overline{cdr}, \\ 0 & \text{when } cdr_{i,t-1} > \overline{cdr}, \end{cases}$$

where $\overline{cdr} = \frac{1}{S} \sum_{i=1}^{N} \sum_{i=t}^{T} s_{i,t} \cdot cdr_{i,t}$.

[15] In unreported specifications, we also included a quadratic year trend and war dummies. Since the general results do not change, we dropped these variables from the final specifications.

[16] The exogenous change in mortality growth is expected to be smaller than observed, since interdependencies with economic and demographic growth increase the observed change. We expect a negative effect of mortality growth on income and population growth, as well as a negative effect of income growth on mortality growth. Therefore, a small exogenous change in

smaller (more negative).

In the next step we apply the instrumental variable approach described in the last subsection. In Tables 6 and 7, we report the first stage estimation results for infant mortality and crude death rate growth, respectively. As suspected, we find that the interaction effects play an important role in predicting the health variables. The instrument and most of the interactions have a significant influence on infant mortality growth and crude death growth, respectively. F-tests for the joint influence of all coefficients which involve the instrument suggest a highly significant joint influence in all specifications.[17] Also, the first stage explains a substantial portion of the variation of the health variables. The first stage statistics suggest that the instruments have sufficient power to identify exogenous variation in the crude death growth.

Table 8 presents estimation results of the causal effect of changes in mortality, in terms of infant mortality growth, from the second stage of 2SLS estimations. The results in the first panel suggests a significant negative effect of infant mortality growth on growth in GDP per capita. We find that a one percentage point increase in infant mortality growth, leads to a reduction in GDP per capita growth by about 1.5 percentage points. In the specification (4a), where we include country dummies and a linear year trend, the effect is 1.5 percentage points. In the second panel of Table 8, we estimate the causal effect of infant mortality growth on population growth. The absolute size of coefficients of infant mortality growth increase only slightly compared to the OLS results and are still insignificant, which suggests that infant mortality growth has little influence on population growth. The last panel of Table 8 contains the the results regarding the causal effect of infant mortality growth for GDP growth. A one percentage point increase in infant mortality growth reduces GDP growth by 1.7-1.9 percentage points.

mortality growth is multiplied to a larger observed change.

[17] Because of the high number of interaction terms and the one-off variation in the instrument, there might be a problem of multicollinearity. This is no reason for concern, however, since the first stage is only a prediction model and we do not aim to draw any causal conclusions from the coefficient estimates, such that multicollinearity does not matter. In contrast, multicollinearity can even help, because it increases the predictive power of the model (see e.g., Angrist and Pischke, 2008). As sensitivity analysis for the first stage we applied an outlier analysis. We could not find any outlier with a strong impact on the estimates. The results of the outlier analysis are available upon request.

In Table 9 we present the corresponding IV results for crude death rate growth as measure for variation in health conditions. The effect on GDP per capita growth turns negative, but is still insignificant except for the specification with period fixed effects. On the other hand, the effect on population growth is significant and even stronger (with a coefficient between -0.18 and -0.34). The effect on GDP growth are all negative, but only significant in the specification with period fixed effects.

The bottom of Tables 8 and 9 presents some first stage statistics. The Shea's R^2 is always higher than 0.15 which indicates that the instruments have substantial explanatory power. The same conclusion can be drawn from the first stage F-Statistic, where the null hypothesis of weak identification can be rejected. We also report the Hansen's J Statistic, because we use a large number of interactions as instrument. The null hypothesis can generally not be rejected, suggesting that we do not face a considerable problem of too many instruments with little additional explanatory power. In the lower part of each panel of Tables 8 and 9, we also present the results of a Hausman specification test. We regress the outcome variable (GDP per capita growth, population growth or aggregate GDP growth) on the observed change in mortality (infant mortality growth and crude death growth, respectively) and the estimated error from the first stage. Significant coefficients for the error term would indicate problems of endogeneity in the OLS regressions, otherwise, the problem of reverse causality is less severe in the growth rates. We find significant endogeneity between infant mortality growth and GDP per capita growth and GDP growth, respectively. However, the effect of infant mortality growth on population growth does not differ significantly between OLS and 2SLS estimates. Also for the crude death growth we do not find any significant indication for endogeneity at all. This indicates that the use of growth rates instead of levels in the outcome equation, as well as the sample construction already solves large parts of the endogeneity issues of the crude death rate.

Overall, the results suggest that the effect of infant mortality growth on GDP per capita growth and GDP growth is negative, and we find no effect on population growth. Crude death growth, in turn, has zero effect on GDP per capita

growth and GDP growth, and a negative effect on population growth.[18]

Despite the evidence from the first stage statistics, the 2SLS results might be biased due to the relatively large number of instruments, some of which might be weak. In order to investigate the robustness of our results, we replicate our analysis using limited information maximum likelihood (LIML) methods, which imply a lower bias than 2SLS estimates, in particular in small samples (see, e.g., Flores-Lagunes, 2007, and Angrist and Pischke, 2008). Tables 10 and 11 present the results for infant mortality and crude death rate growth, respectively. The results are qualitatively as well as quantitatively very similar to those obtained with 2SLS, even though the standard errors are larger as is to be expected with LIML. Taken together, however, the results suggests that the bias of the IV results is at best modest.

3.2 Influence of Institutions

As discussed above, institutions can also have an important influence on economic and demographic growth (see e.g., Acemoglu, Johnson, and Robinson, 2005). In order to account for this influence, we use three different measures for institutions. We use a dummy for democratization, which is equal to one in the period after election rules are introduced and zero otherwise ($\Delta ElectRules_{i,t-1}$). Introduction dates of election rules are collected from Persson and Tabellini (2003). Another measure is the age of these election rules ($AgeElectRules_{i,t-1}$). This variable is equal to zero before the introduction of election rules and afterwards corresponds to the difference between the respective observation year and the introduction year. Finally, we use political regime dummies from the Polity IV Project. We distinguish between autocracies, democracies and anocracies. The omitted category are anocracies, which are mixed or incoherent authority regimes.

Regression results are presented in Table 12. In all models we use our first baseline specification, where we include a year trend.[19] The differences in the coef-

[18] The results are essentially unchanged when adding lags of Δm as additional controls. The respective coefficients are not significantly different from zero.

[19] We do not find strong differences in the other three specifications used in the baseline results. Thus we concentrate here on the most simple specifications. However, the results are robust in the other specifications.

ficients compared to the benchmark results are marginal. We apply a Durbin-Wu-Hausman (DWH) test to check for significant differences between the coefficient of mortality growth, relative to the baseline model.[20] We do not find a significant difference in any of the models. Accordingly, the institution variables cannot account for the influence of mortality growth on economic and demographic growth, i.e., the effect of mortality changes on economic development does not appear to be just an indirect effect of institutions, and accounting for institutions does also not damage our identification strategy.[21] However, especially for population growth, institutions have some additional explanatory power.

4 Robustness Checks

4.1 Additional Controls

Up to this point, the focus was mainly on the problem of reverse causality, since this is the most serve endogeneity problem in empirical growth models. Another potential problem could be a direct effect of the instrument on the outcome variable. Eventually, this would imply that there exists an omitted variable that is jointly correlated with the instrument, the change in mortality, and economic or demographic development. In this case, the results obtained with our identification strategy would still be biased.

Such an omitted variable could be the share of people working in the health service sector, or, more broadly, the service sector in general. The introduction of a universal public health care system could increase the number of doctors, nurses and chemists. People who work in the service sector have lower mortality rates, since the probability of an accident is much lower in such occupations. Finally, the fraction of people working in the health sector could be correlated with economic and demographic growth, since it is an indicator for structural change. In order to account for potential biases, we would ideally condition on the fraction of people working in the service sector. This variable is not available,

[20] We calculate a heteroscedasticity robust DWH statistic following Baum and Schaffer (2003).
[21] The results for crude death rate growth are also qualitatively unchanged and are available upon request.

but we have as proxy the fraction of GDP by sector of origin. We distinguish between the agricultural, industrial and service sectors. Since the shares sum up to one, we use the service sector as omitted category. Unfortunately, we do not have the fraction of GDP by sector for all countries in all time periods under observation. It is for example difficult to get these fractions for Switzerland. In order to receive comparable results, we first repeat our baseline specification with the smaller sample. Then we include the additional control variables and use a DWH test to control for significant differences in the coefficient of interest. Because of the low number of observations, we use the most parsimonious baseline specification, where we include a year trend. Also, in light of the finding that the change in crude death rates appears to be affected less by reverse causality, we concentrate on infant mortality growth in the following.[22] Results can be found in Table 13. The coefficients of interest remain robust when including control variables for structural change, we do not find any significant difference to the baseline specification.[23]

Additionally, one might argue that it is important to condition for government expenditure. The introduction of a universal public health system could be rather expensive and could have a direct effect on economic growth and via the larger income also on mortality growth. However, Lindert (2004) argues that developed countries show much care in choosing the design of taxes and transfers so as to avoid compromising growth. We can observe public consumption as share of total GDP ($\%PubCons_{i,t-1}$) in the data. Results including public consumption as additional control are reported in Table 14. We do not find any significant difference in the coefficient of infant mortality growth in comparison to the baseline model with this smaller sample. Additionally, one could argue,

[22] A complete analysis was also done for crude death growth. We do not find any incidence for additional endogeneity which would affect our results. In some specifications we find even significant negative effects of crude death growth on GDP per capita growth and GDP growth, respectively. These could be driven by the smaller sample size. All robustness checks are available upon request.

[23] The first stage F-Statistic takes often low values in the robustness checks. The reason is that in many instances we do not observe the additional control variables in the period after the introduction of a universal public health care system. Therefore, we do not only have a lower number of observations in the sample, but even less observations in the period after the introduction. In light of this, we find it worth noting that the F-Statistic is in all specifications still significant at the 5%-level and the Shea's R^2 is considerably high.

that not the share of public consumption has an influence on the coefficients of interest, but the change in public consumption, following an argument that only changes in fiscal policy influence economic development. In Table 15 we condition on the lagged relative public consumption growth ($\Delta\%PubCons_{i,t-1}$). The coefficients of infant mortality growth are not significantly different. The effect of infant mortality growth on population growth even becomes significant, but this can be explained by the different sample composition.

It could also be that individuals have a higher work-life satisfaction after the introduction of a universal public health care system, because they have the feeling that the government takes care of them. Higher satisfaction could have an influence on health as well as on economic development, since more satisfied individuals could have a higher productivity. We use the lagged number of labor disputes (per million workers) ($\#Disp_{i,t-1}$) and the lagged number of workers involved in labor disputes (per million workers) ($\#Work_{i,t-1}$) as proxy for work-life satisfaction. Since we have a number of autocratic (terror) regimes in our sample, where the population is potentially very unsatisfied, but is not allowed to demonstrate, we additionally condition on democracies. Results can be found in Table 16. According to the DWH test, the coefficient of interest do not change significantly. In order to have also a different proxy for work-life satisfaction we include the lagged number of days lost in labor disputes (per million workers) ($DaysLost_{i,t-1}$) in the regressions of Table 17. Again the coefficient of interest does not change according to the DWH test.

Finally, one could argue that our estimation model is misspecified since one needs to condition on the gross capital formation according to the prediction of the Solow growth model. For a small number of observations we have data on the gross capital formation as share of total GDP ($\%GCF_{i,t-1}$). In Table 18 we find that the effect of infant mortality growth on GDP per capita growth differ significantly at the 10%-level when we condition on gross capital formation. The effect is even more negative. If anything, our baseline specifications would underestimate the true absolute effect of infant mortality growth on GDP per capita growth. The effects on population growth and GDP growth are unaffected

by the inclusion of gross capital formation.

4.2 Placebo Treatments

As another robustness check, we apply a placebo treatment test. As placebo treatment, we artificially set the instrument equal to one one period 'too early'.[24] Accordingly, the instrument is equal to one in the period of introduction and not one period after the introduction of a universal public health care system. Since there is not enough adjustment time for the mechanisms that affect changes in mortality, we expect zero effect at the first stage. This raises the problem of weak identification. In Table 19 we display the first stage of the placebo treatment test for the baseline specifications. As expected, the instrument and the interaction have virtually no effect on mortality growth. Without a single exception, all coefficients in all specifications are insignificant. We perform a test for joint significance. We cannot reject the null hypothesis of no joint influence in all specifications at the 1%-level. In Table 19 we also show the first stage statistics. The Shea's R^2 and the F-Statistic are rather low, which indicates that the placebo-instrument has nearly no power despite a comparably large number of observations as in the main analysis. In the second stage, the coefficients of interest are insignificant and have high standard errors, as shown in Table 20. Nearly all coefficients change sign and become positive. We interpret this as indication that our identification strategy passes the placebo treatment test.[25] This also suggests that it it unlikely that the previous results are driven by common time trends or random shocks. Moreover, this supports our initial assumptions about the causal channel, namely that first the universal public health care systems were introduced and then the effects on economic and demographic development unfolded, and not vice versa.

[24] A delayed placebo makes less sense since the placebo might pick up delayed treatment effects as the spread of health system coverage. Similarly, indirect effects like demographic change and education might become active with a delay.

[25] Similar results are obtained for crude death growth.

4.3 Different Data Frequencies

One important parameter in our empirical analysis is the choice of the data frequency. The choice of a 20-year data frequency is to some extent arbitrary. In our identification approach, the data frequency has two important implications. On the one hand, we want a fairly short data frequency, because otherwise the instrument has no power on mortality growth. In the long run, the effect of the introduction of a universal public health care system diminishes. On the other hand, we need a sufficiently long data frequency, because we expect that the effects of mortality change on economic and demographic development have a rather long run character and need time to unfold. If the data frequency is too short, mortality growth has no influence on the outcome variables. In this section, we therefore investigate the sensitivity of our results for alternative data frequencies of 15- and 30-year periods.

Table 21 shows the estimation results with 15-year data frequency. The main results remain robust. Infant mortality growth has a negative effect on GDP per capita growth and GDP growth and no significant effect on population growth. The coefficients are smaller, which indicates that infant mortality has a smaller effect on economic and demographic growth in the short run.

Table 22 shows the results for 30-year data frequency. The number of observations is very small. The size of the coefficients is even larger than for the 20-year data frequency, but the coefficients are insignificant because of the high standard errors. Only in specification 3, where we include period dummies, the effect of interest is smaller. When we increase the data frequency further to 40 years, the coefficients of interest get even larger. However, the first stage has less power and the number of observations decreases to 35. For crude death growth we find that the instrument has strong power independent of the data frequency. The coefficient estimates are qualitatively and quantitatively unaffected by the data frequency.[26] In summary, we conclude that our estimates might potentially not capture the total effect of mortality growth on economic and demographic growth. Our findings might nevertheless be valid for the medium term. Long run

[26] Results are available upon request.

effects of mortality growth on economic and demographic growth could be even stronger, but are difficult to identify with our identification strategy.

5 Conclusion

This paper has applied a novel identification strategy based on the timing of the implementation of a universal public health system to estimate the causal effect of mortality changes on economic growth and population growth. The results indicate that a reduction in mortality accelerates growth of income per capita and population size. The results reconcile earlier findings in the literature by documenting a positive effect of mortality reductions on growth based on an identification strategy that exploits within-country over-time variation, suggesting that the discrepancies in earlier findings might be the result of differences in sample composition, rather than identification method. Moreover, our results suggest that public health policy plays a potentially important role for economic development.

Naturally, there are caveats to our analysis that need to be taken into account when interpreting our results. First, the findings are based on a small sample, with the identifying variation stemming from European countries in the late 19th and early 20th Century. As in previous studies, sample composition might affect the generality and external validity of our results. Nevertheless, given the particular sample, the results can be seen as a complement to studies using exclusively cross-country variation in geo-climatological conditions (as in Lorentzen et al., 2008) or using within country variation during a very particular period of global development (as the global epidemiological transition exploited by Acemoglu and Johnson, 2007).

References

Acemoglu, D., and S. Johnson (2007): "Disease and Development: The Effect of Life Expectancy on Economic Growth," *Journal of Political Economy*, 115(6), 925–985.

——— (2009): "Disease and Development: A Reply to Bloom, Canning and Fink," *Working Paper*.

Acemoglu, D., S. Johnson, and J. Robinson (2005): "Institutions as the Fundamental Cause of Long-Run Growth," in *Handbook of Economic Growth*, ed. by P. Aghion, and S. Durlauf, vol. 1, pp. 385–472. Elsevier.

Angeles, L. (2010): "Demographic Transitions: Analyzing the effects of Mortality on Fertility," *Journal of Population Economics*, 23, 99–120.

Angrist, J. D., and J. Pischke (2008): *Mostly Harmless Econometrics*. Princeton University Press.

Barro, R. (1991): "Economic Growth in a Cross Section of Countries," *Quarterly Journal of Economics*, 106(2), 407–443.

Baum, C., and M. Schaffer (2003): "Instrumental Variables and GMM: Estimation and Testing," *Stata Journal*, 3(1), 1–31.

Bekker, P. (1994): "Alternative Approximations to the Distributions of Instrumental Variables Estimators," *Econometrica*, 62(3), 657–681.

Bloom, D., D. Canning, and G. Fink (2009): "Disease and Development Revisited," *NBER Working Paper*, No. 15137.

Cervellati, M., and U. Sunde (2011): "The Effect of Life Expectancy on Economic Growth Reconsidered," *Journal of Economic Growth*, 16(2), 99–133.

Chesnais, J. (1992): *The Demographic Transition: Stages, Patterns and Economic Implications. A Longitudinal Study of Sixty-Seven Countries Covering the Period 1720-1984*. Clarendon Press, Oxford.

Corens, D. (2007): "Health System Review: Belgium," *Health Systems in Transition*, 9(2), 1–172.

Cutler, D., A. Deaton, and A. Lleras-Muney (2006): "The Determinants of Mortality," *Journal of Economic Perspectives*, 20, 97–120.

Durlauf, S., P. Johnson, and J. Temple (2005): "Growth Econometrics," in *Handbook of Economic Growth*, ed. by P. Aghion, and S. Durlauf, vol. 1, pp. 555–677. Elsevier.

Durán, A., J. Lara, and M. Van Waveren (2006): "Health System Review: Spain," *Health Systems in Transition*, 8(4), 1–208.

Flora, P. (1983): *State, Economy, and Society in Western Europe, 1815-1975, I.* St. James Press, Chicago.

Flora, P., F. Kraus, and W. Pfenning (1987): *State, Economy, and Society in Western Europe, 1815-1975, II.* St. James Press, Chicago.

Flores-Lagunes, A. (2007): "Finite Sample Evidence of IV Estimators under Weak Instruments," *Journal of Applied Econometrics*, 22(3), 677–694.

Glenngård, A., F. Hjalte, M. Svensson, A. Anell, and V. Bankauskaite (2005): "Health System Review: Sweden," *Health Systems in Transition*, 7(4), 1–128.

Gurr, T. R. (1974): "Persistence and Change in Political Systems, 1800-1971," *American Political Science Review*, 68(4), 1482–1504.

Hofmarcher, M., and H. Rack (2001): "Health System Review: Austria," *Health Systems in Transition*, 3(5), 1–120.

Johnsen, J. (2006): "Norway," *Health Systems in Transition*, 8(1), 1–167.

Lindert, P. (2004): *Growing Public*. Cambridge University Press.

Lo Scalzo, A., A. Donatini, L. Orzella, A. Cicchetti, S. Profili, and A. Maresso (2009): "Italy," *Health Systems Review*, 11(6), 1–216.

Lorentzen, P., J. McMillan, and R. Wacziarg (2008): "Death and Development," *Journal of Economic Growth*, 13, 81–124.

Mackenbach, J. (1996): "The Contribution of Medical Care to Mortality Decline: McKeown Revisted," *Journal of Clinical Epidemiology*, 49, 1207–1213.

Maddison, A. (2006): *The World Economy, Volume 1: A Millenial Perspective*. OECD Publishing, Paris.

Minder, A., H. Schoenholzer, and M. Amiet (2000): "Switzerland," *Health Care Systems in Transition*, pp. 1–82.

Mitchell, B. (1992): *European Historical Statistics, 1750-1988*. Stockston Press, New York, 3 edn.

Persson, T., and G. Tabellini (2003): *The Economic Effects of Constitutions*. MIT Press, Cambridge, Mass.

Robinson, R., and A. Dixon (1999): "United Kingdom," *Health Systems in Transition*.

Sandier, S., V. Paris, and D. Polton (2004): "Health System Review: France," *Health Systems in Transition*, 6(2), 1–145.

Schäfer, W., M. Kroneman, W. Boerma, M. Van den Berg, G. Westert, W. Devillé, and E. van Ginneken (2010): "Health System Review: The Netherlands," *Health Systems in Transition*, 12(1), 1–229.

Strandberg-Larsen, M., M. Nielsen, S. Vallgårda, A. Krasnik, and K. Vrangbæk (2007): "Health System Review: Denmark," *Health Systems Review*, 9(6), 1–164.

Vuorenkoski, L. (2008): "Health System Review: Finland," *Health Systems in Transition*, 10(4), 1–168.

Figure 1: Timing of Events: Data Preparation

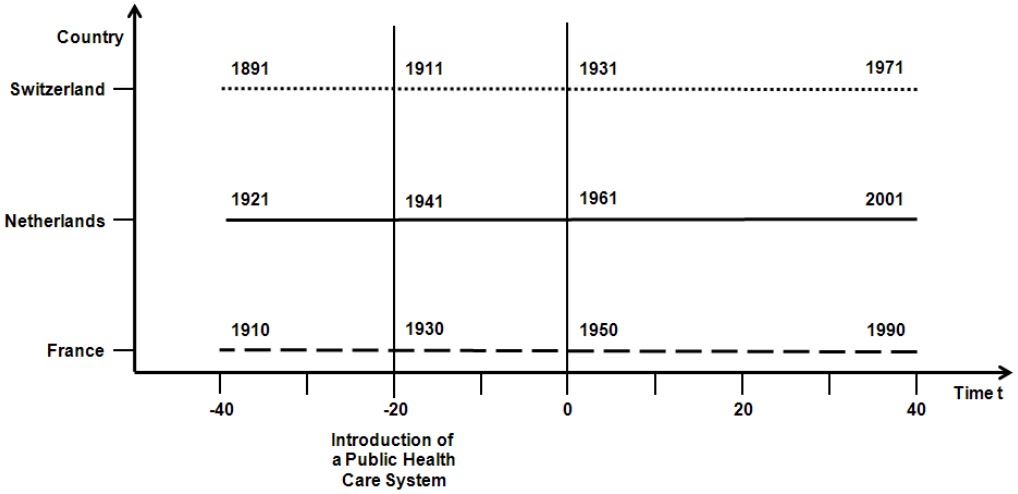


Figure 2: Timing of Events: Construction of the Instrument z_i, t.

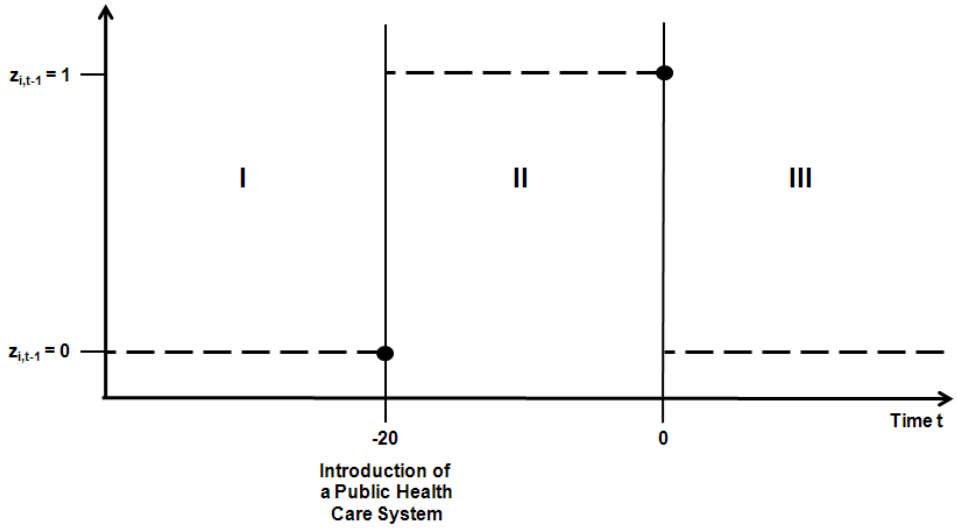
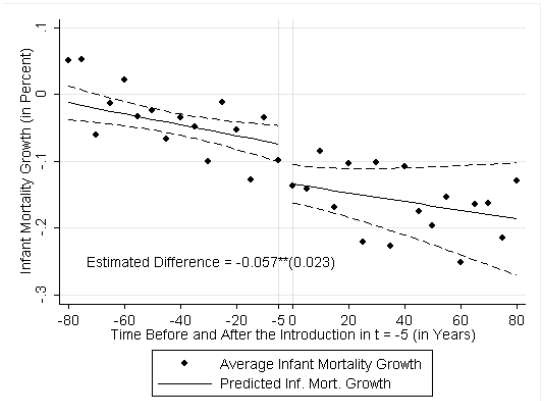
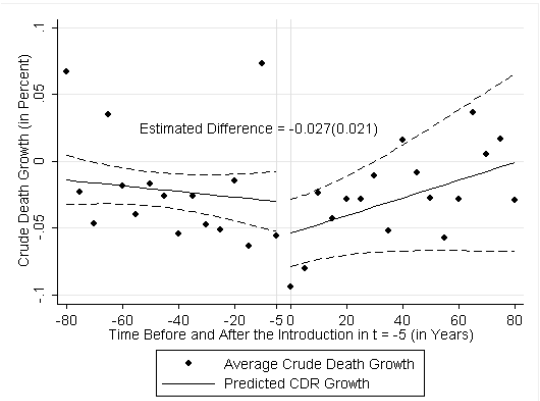


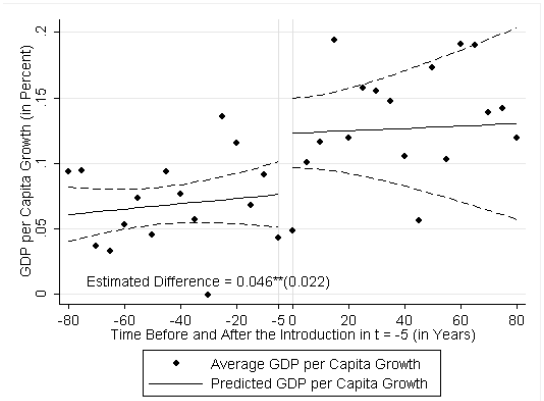
Figure 3: Growth rates before and after the introduction of an universal public health care system (t=-5).



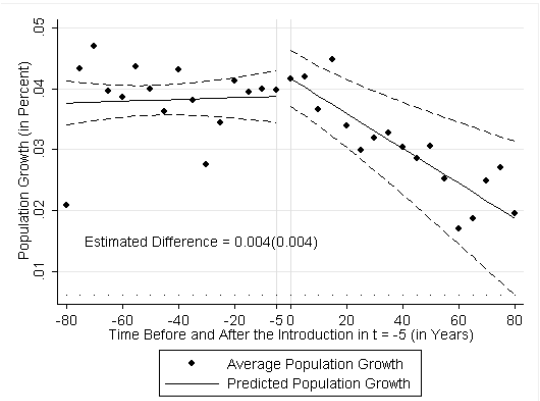
(a) infant mortality growth



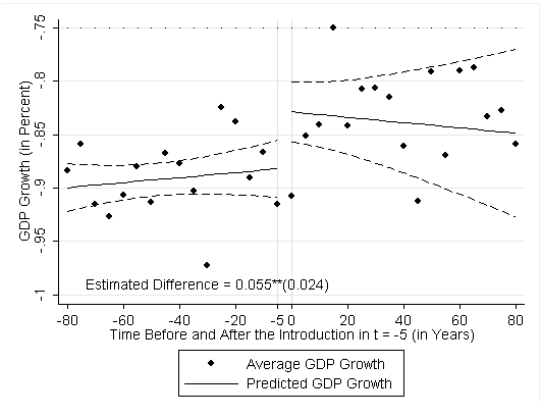
(b) crude death rate growth



(c) GDP per capita growth



(d) population growth



(e) GDP growth

Table 3: Summary statistics.

	Summary Statistics				
	20-Year Frequency				
Variables	Obs	Mean	Std. Dev.	Min	Max
$\Delta imr_{i,t}$	84	-0.358	0.232	-0.752	0.370
$\Delta cdr_{i,t}$	84	-0.110	0.146	-0.508	0.327
$\Delta gdpc_{i,t}$	84	0.485	0.408	-0.382	2.830
$\Delta pop_{i,t}$	84	0.143	0.081	0.009	0.336
$\Delta gdp_{i,t}$	84	0.696	0.469	-0.349	3.053
$lgdpc_{i,t-1}$	84	8.238	0.761	6.829	9.763
$lpop_{i,t-1}$	84	9.151	1.099	7.262	10.954
$PostTrans_{i,t-1}$	84	0.488	0.503	0	1
$\Delta ElectRules_{i,t-1}$	84	0.119	0.326	0	1
$AgeElectRules_{i,t-1}$	84	20.095	31.755	0	151
$Autocracies_{i,t-1}$	82	0.073	0.262	0	1
$Democracies_{i,t-1}$	82	0.622	0.488	0	1
$\%Industry_{i,t-1}$	52	34.173	8.915	18.800	50.600
$\%Agri_{i,t-1}$	52	22.362	16.933	2.000	55.000
$\%GovExp_{i,t-1}$	65	13.411	9.439	0.765	44.193
$\Delta\%GovExp_{i,t-1}$	50	0.483	0.650	-0.446	2.157
$\#Disp_{i,t-1}$	51	2.440	5.619	0.002	23.780
$\#Work_{i,t-1}$	54	25.624	57.109	0.010	311.870
$DaysLost_{i,t-1}$	51	0.230	0.331	0.000	1.186
$\%GCF_{i,t-1}$	54	16.321	6.628	5.657	29.200
$Year$	84	1938.476	41.930	1850	2008

Table 4: OLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth and other controls.

Dependent Variable:	**GDP per capita Growth (Δgdpc$_{i,t}$)**			
	20-Year-Frequency			
	(1a)	(2a)	(3a)	(4a)
$\Delta imr_{i,t}$	-0.683***	-0.663***	-0.719***	-0.502*
	[0.172]	[0.199]	[0.155]	[0.264]
$lgdpc_{i,t-1}$	-0.414**	-0.416*	-0.512**	-0.567
	[0.181]	[0.214]	[0.168]	[0.331]
$lpop_{i,t-1}$	-0.006	0.010	-0.008	-0.118
	[0.017]	[0.026]	[0.019]	[0.221]
$PostTrans_{i,t-1}$	0.342*	0.384	0.143	0.404
	[0.189]	[0.214]	[0.114]	[0.231]
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
R-squared	0.478	0.516	0.671	0.515
Observations	84	84	84	84
Dependent Variable:	**Population Growth (Δpop$_{i,t}$)**			
	20-Year-Frequency			
	(1b)	(2b)	(3b)	(4b)
$\Delta imr_{i,t}$	-0.043	-0.051	-0.019	-0.018
	[0.065]	[0.089]	[0.051]	[0.057]
$lgdpc_{i,t-1}$	-0.016	-0.011	-0.020	-0.052
	[0.027]	[0.031]	[0.021]	[0.037]
$lpop_{i,t-1}$	-0.028***	-0.033***	-0.028***	-0.163**
	[0.007]	[0.008]	[0.007]	[0.067]
$PostTrans_{i,t-1}$	0.020	0.017	0.017	0.020
	[0.026]	[0.034]	[0.026]	[0.033]
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
R-squared	0.257	0.448	0.279	0.556
Observations	84	84	84	84
Dependent Variable:	**GDP Growth (Δgdp$_{i,t}$)**			
	20-Year-Frequency			
	(1c)	(2c)	(3c)	(4c)
$\Delta imr_{i,t}$	-0.869***	-0.865***	-0.851***	-0.636*
	[0.195]	[0.227]	[0.163]	[0.317]
$lgdpc_{i,t-1}$	-0.483**	-0.482**	-0.599***	-0.702**
	[0.174]	[0.211]	[0.159]	[0.318]
$lpop_{i,t-1}$	-0.044**	-0.032	-0.045*	-0.312
	[0.018]	[0.028]	[0.024]	[0.267]
$PostTrans_{i,t-1}$	0.490*	0.531*	0.244	0.499*
	[0.228]	[0.259]	[0.139]	[0.264]
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
R-squared	0.464	0.501	0.648	0.505
Observations	84	84	84	84

Results from OLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 5: OLS regression of GDP per capita growth, population growth and GDP growth on crude death rate growth and other controls.

Dependent Variable:	**GDP per capita Growth (Δgdpc$_{i,t}$)**			
	20-Year-Frequency			
	(1a)	(2a)	(3a)	(4a)
$\Delta cdr_{i,t}$	0.169	0.169	-0.425*	0.379
	[0.247]	[0.290]	[0.201]	[0.406]
$lgdpc_{i,t-1}$	-0.498**	-0.484*	-0.537**	-0.739**
	[0.190]	[0.226]	[0.178]	[0.310]
$lpop_{i,t-1}$	-0.010	0.006	-0.016	-0.216
	[0.023]	[0.033]	[0.019]	[0.248]
$PostTrans_{i,t-1}$	0.385*	0.431*	0.188	0.403*
	[0.187]	[0.213]	[0.134]	[0.207]
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
R-squared	0.437	0.481	0.638	0.509
Observations	84	84	84	84
Dependent Variable:	**Population Growth (Δpop$_{i,t}$)**			
	20-Year-Frequency			
	(1b)	(2b)	(3b)	(4b)
$\Delta cdr_{i,t}$	-0.161***	-0.134***	-0.192***	-0.111***
	[0.040]	[0.040]	[0.048]	[0.029]
$lgdpc_{i,t-1}$	-0.011	-0.008	-0.030	-0.037
	[0.022]	[0.023]	[0.019]	[0.035]
$lpop_{i,t-1}$	-0.028***	-0.033***	-0.030***	-0.146**
	[0.007]	[0.007]	[0.007]	[0.059]
$PostTrans_{i,t-1}$	0.040	0.035	0.032	
	[0.029]	[0.033]	[0.026]	[0.031]
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
R-squared	0.329	0.492	0.358	0.586
Observations	84	84	84	84
Dependent Variable:	**GDP Growth (Δgdp$_{i,t}$)**			
	20-Year-Frequency			
	(1c)	(2c)	(3c)	(4c)
$\Delta cdr_{i,t}$	-0.073	-0.035	-0.765***	0.216
	[0.288]	[0.331]	[0.207]	[0.430]
lgdpc_i,t-1	-0.571**	-0.556**	-0.641***	-0.875**
	[0.188]	[0.229]	[0.165]	[0.303]
lpop_i,t-1	-0.049*	-0.038	-0.057**	-0.394
	[0.024]	[0.037]	[0.025]	[0.273]
PostTrans_i,t-1	0.490*	0.531*	0.244	0.499*
	[0.223]	[0.252]	[0.155]	[0.241]
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
R-squared	0.410	0.451	0.634	0.484
Observations	84	84	84	84

Results from OLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 6: First stage: 2SLS regression of infant mortality growth on the instruments and other controls.

Dependent Variable:	**Infant Mortality Growth (Δimr$_{i,t}$)**			
	20-Year Frequency			
	(1a)	(2a)	(3a)	(4a)
$\Delta Ins_{i,t-1}$	-0.436	-0.239	1.356	-0.970
	[1.086]	[1.187]	[0.989]	[1.188]
$\Delta Ins_{i,t-1} \times lgdpc_{i,t-1}$	-0.079	-0.118	-0.166	-0.024
	[0.164]	[0.177]	[0.127]	[0.175]
$\Delta Ins_{i,t-1} \times lpop_{i,t-1}$	0.117**	0.132**	0.013	0.131**
	[0.043]	[0.044]	[0.043]	[0.045]
$\Delta Ins_{i,t-1} \times PostTrans_{i,t-1}$	0.208**	0.191**	0.208***	0.208**
	[0.074]	[0.085]	[0.063]	[0.086]
$\Delta Ins_{i,t-1} \times YearTrend$	-0.006***	-0.006***		-0.007***
	[0.002]	[0.002]		[0.001]
$\Delta Ins_{i,t-1} \times D_{(Year \leq 1920)}$			-0.268**	
			[0.108]	
$\Delta Ins_{i,t-1} \times D_{(1920<Year \leq 1935)}$			-0.372**	
			[0.130]	
$\Delta Ins_{i,t-1} \times D_{(1950<Year \leq 1965)}$			-0.472***	
			[0.131]	
$\Delta Ins_{i,t-1} \times D_{(1965<Year)}$			-0.163	
			[0.163]	
$lgdpc_{i,t-1}$	0.110***	0.082*	0.035	0.202***
	[0.032]	[0.042]	[0.024]	[0.047]
$lpop_{i,t-1}$	-0.006	-0.003	-0.006	0.104
	[0.011]	[0.013]	[0.013]	[0.163]
$PostTrans_{i,t-1}$	-0.144***	-0.142***	-0.083	-0.132***
	[0.042]	[0.040]	[0.052]	[0.040]
Year Trend	Yes	No	No	Yes
Country Specific Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Dummies	No	No	No	Yes
Joint F-Test ($\Delta Ins\_i, t-1$)	18.96	16.95	9.743	62.40
p-value	0.000	0.000	0.001	0.000
R-squared	0.754	0.784	0.766	0.792
Observations	84	84	84	84

Results from 2SLS regressions (first stage), the dependent variables is infant mortality growth ($\Delta imr_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 7: First stage: 2SLS regression of crude death rate growth on the instruments and other controls.

Dependent Variable:	**Crude Death Rate Growth (Δcdr$_{i,t}$)**			
	20-Year Frequency			
	(1a)	(2a)	(3a)	(4a)
$\Delta Ins_{i,t-1}$	-3.334***	-3.888***	-2.145**	-3.965***
	[0.690]	[0.759]	[0.756]	[0.707]
$\Delta Ins_{i,t-1} \times lgdpc_{i,t-1}$	0.295**	0.344**	0.180	0.350***
	[0.103]	[0.113]	[0.102]	[0.106]
$\Delta Ins_{i,t-1} \times lpop_{i,t-1}$	0.097**	0.117***	0.060**	0.120***
	[0.032]	[0.030]	[0.024]	[0.031]
$\Delta Ins_{i,t-1} \times PostTrans\_i, t-1$	0.123	0.153	0.108	0.148
	[0.094]	[0.098]	[0.077]	[0.108]
$\Delta Ins_{i,t-1} \times YearTrend$	-0.006***	-0.008***		-0.007***
	[0.002]	[0.002]		[0.002]
$\Delta Ins_{i,t-1} \times D_{(Year \leq 1920)}$			0.067	
			[0.139]	
$\Delta Ins_{i,t-1} \times D_{(1920<Year \leq 1935)}$			0.128	
			[0.111]	
$\Delta Ins_{i,t-1} \times D_{(1950<Year \leq 1965)}$			-0.153	
			[0.092]	
$\Delta Ins_{i,t-1} \times D_{(1965<Year)}$			-0.099	
			[0.142]	
$lgdpc_{i,t-1}$	0.021	0.003	-0.044	0.127
	[0.051]	[0.062]	[0.027]	[0.098]
$lpop_{i,t-1}$	-0.013	-0.011	-0.015	0.205**
	[0.015]	[0.017]	[0.014]	[0.090]
$PostTrans_{i,t-1}$	0.062	0.055	0.029	0.074
	[0.074]	[0.075]	[0.070]	[0.088]
Year Trend	Yes	No	No	Yes
Country Specific Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Dummies	No	No	No	Yes
Joint F-Test ($\Delta Ins\_i, t-1$)	11.74	17.55	9.053	22.90
p-value	0.000	0.000	0.001	0.000
R-squared	0.275	0.344	0.440	0.429
Observations	84	84	84	84

Results from 2SLS regressions (first stage), the dependent variables is crude death rate growth ($\Delta cdr_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 8: Second stage: 2SLS regression of GDP per capita growth, population growth, and GDP growth on infant mortality growth and other controls.

Dependent Variable:	**GDP per capita Growth (Δgdpc$_{i,t}$)**			
	(1a)	(2a)	(3a)	(4a)
$\widehat{\Delta imr}_{i,t}$	-1.529***	-1.337***	-1.358***	-1.543***
	[0.481]	[0.372]	[0.490]	[0.446]
$lgdpc_{i,t-1}$	-0.324**	-0.357**	-0.509***	-0.346
	[0.165]	[0.180]	[0.149]	[0.281]
$lpop_{i,t-1}$	-0.002	0.014	-0.004	-0.040
	[0.014]	[0.020]	[0.019]	[0.244]
$PostTrans_{i,t-1}$	0.267	0.319*	0.132	0.319*
	[0.163]	[0.177]	[0.099]	[0.181]
Hansen J Statistic	2.423	2.218	8.984	3.040
p-value	0.658	0.696	0.254	0.551
Hausman Test (T-Stat)	2.564	2.359	1.586	2.365
p-value	0.026	0.038	0.141	0.037
Dependent Variable:	**Population Growth (Δpop$_{i,t}$)**			
	(1b)	(2b)	(3b)	(4b)
$\widehat{\Delta imr}_{i,t}$	-0.094	-0.118	-0.117	-0.069
	[0.134]	[0.137]	[0.130]	[0.147]
$lgdpc_{i,t-1}$	-0.011	-0.005	-0.020	-0.041
	[0.031]	[0.031]	[0.020]	[0.039]
$lpop_{i,t-1}$	-0.028***	-0.032***	-0.027***	-0.159***
	[0.007]	[0.007]	[0.006]	[0.060]
$PostTrans_{i,t-1}$	0.015	0.011	0.015	0.016
	[0.029]	[0.034]	[0.026]	[0.032]
Hansen J Statistic	6.692	7.248	9.686	4.243
p-value	0.153	0.123	0.207	0.374
Hausman Test (T-Stat)	0.396	0.554	0.983	0.372
p-value	0.700	0.590	0.347	0.717
Dependent Variable:	**GDP Growth (Δgdp$_{i,t}$)**			
	(1c)	(2c)	(3c)	(4c)
$\widehat{\Delta imr}_{i,t}$	-1.990***	-1.795***	-1.745***	-1.943***
	[0.635]	[0.529]	[0.665]	[0.582]
$lgdpc_{i,t-1}$	-0.364**	-0.400**	-0.595***	-0.424
	[0.155]	[0.180]	[0.140]	[0.301]
$lpop_{i,t-1}$	-0.038**	-0.027	-0.040	-0.214
	[0.015]	[0.022]	[0.024]	[0.336]
$PostTrans_{i,t-1}$	0.307	0.356	0.154	0.364*
	[0.207]	[0.221]	[0.127]	[0.221]
Hansen J Statistic	0.730	0.913	10.93	1.073
p-value	0.948	0.923	0.142	0.898
Hausman Test (T-Stat)	2.706	2.451	1.629	3.103
p-value	0.020	0.032	0.132	0.010
	Specification (all panels)			
	20-Year-Frequency			
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
	First Stage Statistics (all panels)			
Shea's R-Squared	0.158	0.207	0.195	0.172
F-Statistic	18.962	16.949	9.743	62.395
p-value	0.000	0.000	0.001	0.000
Observations	84	84	84	84

Results from 2SLS regressions, the dependent variables are GDP growth ($\Delta gdp_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. Each panel represents a separate set of regressions. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). First stages are as in Table 6 and are identical for each specification. Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 9: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on crude death rate growth and other controls.

Dependent Variable:	**GDP per capita Growth (Δgdpc$_{i,t}$)**			
	(1a)	(2a)	(3a)	(4a)
$\widehat{\Delta cdr}_{i,t}$	-0.230	-0.203	-1.141**	-0.341
	[0.558]	[0.513]	[0.476]	[0.541]
$lgdpc_{i,t-1}$	-0.472***	-0.464**	-0.572***	-0.616**
	[0.183]	[0.197]	[0.177]	[0.282]
$lpop_{i,t-1}$	-0.010	0.006	-0.022	-0.101
	[0.021]	[0.028]	[0.019]	[0.223]
$PostTrans_{i,t-1}$	0.425**	0.466**	0.244*	0.483**
	[0.174]	[0.190]	[0.133]	[0.199]
p-value	0.000	0.000	0.000	0.000
Hansen J Statistic	5.523	5.352	8.670	6.308
Hausman Test (T-Stat)	0.701	0.657	1.364	1.225
p-value	0.498	0.525	0.200	0.246
p-value	0.238	0.253	0.277	0.177
Dependent Variable:	**Population Growth (Δpop$_{i,t}$)**			
	(1b)	(2b)	(3b)	(4b)
$\widehat{\Delta cdr}_{i,t}$	-0.227**	-0.177**	-0.339***	-0.183**
	[0.103]	[0.073]	[0.107]	[0.072]
$lgdpc_{i,t-1}$	-0.006	-0.006	-0.037***	-0.025
	[0.022]	[0.020]	[0.014]	[0.031]
$lpop_{i,t-1}$	-0.028***	-0.033***	-0.031***	-0.135***
	[0.006]	[0.006]	[0.007]	[0.046]
$PostTrans_{i,t-1}$	0.046	0.039	0.043	0.042
	[0.032]	[0.031]	[0.029]	[0.030]
Hansen J Statistic	2.414	0.854	6.542	1.506
p-value	0.660	0.931	0.478	0.826
Hausman Test (T-Stat)	0.573	0.434	0.215	0.914
p-value	0.578	0.673	0.834	0.380
Dependent Variable:	**GDP Growth (Δgdp$_{i,t}$)**			
	(1c)	(2c)	(3c)	(4c)
$\widehat{\Delta cdr}_{i,t}$	-0.692	-0.567	-1.845***	-0.727
	[0.634]	[0.637]	[0.546]	[0.654]
$lgdpc_{i,t-1}$	-0.531***	-0.527***	-0.695***	-0.714***
	[0.173]	[0.197]	[0.171]	[0.274]
$lpop_{i,t-1}$	-0.049**	-0.038	-0.066***	-0.244
	[0.022]	[0.031]	[0.025]	[0.225]
$PostTrans_{i,t-1}$	0.551**	0.580**	0.328**	0.604**
	[0.220]	[0.228]	[0.163]	[0.235]
Hansen J Statistic	5.225	5.221	7.683	4.222
p-value	0.265	0.265	0.361	0.377
Hausman Test (T-Stat)	0.901	0.735	1.424	1.321
p-value	0.387	0.477	0.182	0.213
	Specification (all panels)			
	20-Year-Frequency			
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
	First Stage Statistics (all panels)			
Shea's R-Squared	0.193	0.234	0.155	0.253
F-Statistic	11.74	17.554	10.411	22.904
p-value	0.000	0.000	0.000	0.000
Observations	84	84	84	84

Results from 2SLS regressions, the dependent variables are GDP growth ($\Delta gdp_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. Each panel represents a separate set of regressions. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). First stages are as in Table 7 and are identical for each specification. Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 10: LIML estimation results for GDP per capita growth, population growth and GDP growth on infant mortality growth and other controls.

Dependent Variable:	**GDP per capita Growth (Δgdpc$_{i,t}$)**			
	(1a)	(2a)	(3a)	(4a)
$\Delta imr_{i,t}$	-1.754**	-1.510*	-1.791**	-1.774*
	[0.885]	[0.797]	[0.836]	[0.941]
Dependent Variable:	**Population Growth (Δpop$_{i,t}$)**			
	(1b)	(2b)	(3b)	(4b)
$\Delta imr_{i,t}$	-.119	-.132	-.185	-.119
	[0.226]	[0.156]	[0.233]	[0.254]
Dependent Variable:	**GDP Growth (Δgdp$_{i,t}$)**			
	(1c)	(2c)	(3c)	(4c)
$\Delta imr_{i,t}$	-2.079**	-1.895**	-2.274**	-2.098**
	[0.904]	[0.846]	[0.971]	[1.020]
	Specification (all panels)			
	20-Year-Frequency			
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
Observations	84	84	84	84

Results from LIML estimations with Bekker (1994) adjustment, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 11: LIML estimation results for GDP per capita growth, population growth and GDP growth on crude death rate growth and other controls.

Dependent Variable:	**GDP per Capita Growth (Δgdpc$_{i,t}$)**			
	(1a)	(2a)	(3a)	(4a)
$\Delta cdr_{i,t}$	-.582	-.477	-2.407	-.712
	[1.024]	[0.898]	[1.786]	[0.819]
Dependent Variable:	**Population Growth (Δpop$_{i,t}$)**			
	(1b)	(2b)	(3b)	(4b)
$\Delta cdr_{i,t}$	-.234*	-.179	-.391*	-.198*
	[0.135]	[0.112]	[0.218]	[0.116]
Dependent Variable:	**GDP Growth (Δgdp$_{i,t}$)**			
	(1c)	(2c)	(3c)	(4c)
$\Delta cdr_{i,t}$	-1.035	-.869	-2.951*	-1.074
	[1.023]	[0.975]	[1.576]	[0.891]
	Specification (all panels)			
	20-Year-Frequency			
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
Observations	84	84	84	84

Results from LIML estimations with Bekker (1994) adjustment, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 12: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth, democratization variables and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-1.456***	-1.519***	-0.102	-0.132	-1.922***	-2.063***
	[0.420]	[0.560]	[0.136]	[0.159]	[0.576]	[0.733]
$\Delta ElectRules_{i,t-1}$	-0.014		-0.027**		-0.092	
	[0.155]		[0.013]		[0.163]	
$AgeElectRules_{i,t-1}$	0.001		-0.000		-0.000	
	[0.001]		[0.000]		[0.002]	
$Autocracies_{i,t-1}$		0.012		0.011		0.060
		[0.147]		[0.024]		[0.160]
$Democracies_{i,t-1}$		0.064		-0.035*		0.014
		[0.093]		[0.020]		[0.118]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
Missing Dummy	No	Yes	No	Yes	No	Yes
DWH-Statistic	0.034	0.000	0.003	0.060	0.015	0.011
P-value	0.854	0.984	0.954	0.806	0.903	0.918
Shea's R-Squared	0.161	0.147	0.161	0.147	0.161	0.147
F-Statistic	16.852	13.423	16.852	13.423	16.852	13.423
p-value	0.000	0.000	0.000	0.000	0.000	0.000
Hansen J Statistic	2.652	2.302	5.561	8.855	0.977	0.697
p-value	0.618	0.680	0.234	0.0648	0.913	0.952
Observations	84	84	84	84	84	84

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 13: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth, sector of origin as percentage of total GDP and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-1.536***	-1.655***	-0.197	-0.210	-2.151***	-2.296***
	[0.238]	[0.261]	[0.135]	[0.164]	[0.373]	[0.383]
$\%Agriculture_{i,t-1}$		0.015**		0.001		0.018***
		[0.007]		[0.002]		[0.007]
$\%Industry_{i,t-1}$		0.016**		-0.003*		0.012
		[0.007]		[0.002]		[0.007]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
DWH-Statistic	.	0.251	.	0.007	.	0.148
P-value	.	0.617	.	0.936	.	0.701
Shea's R-Squared	0.273	0.269	0.273	0.269	0.273	0.269
F-Statistic	5.179	5.215	5.179	5.215	5.179	5.215
p-value	0.013	0.013	0.013	0.013	0.013	0.013
Hansen J Statistic	3.576	5.586	6.286	5.604	2.998	2.490
p-value	0.466	0.232	0.179	0.231	0.558	0.646
Observations	52	52	52	52	52	52

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 14: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth, government expenditure as percentage of total GDP and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-2.076***	-2.183***	-0.172**	-0.156	-2.727***	-2.826***
	[0.606]	[0.693]	[0.082]	[0.098]	[0.705]	[0.806]
$\%PubCons_{i,t-1}$		0.004		-0.001		0.004
		[0.005]		[0.001]		[0.007]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
DWH-Statistic	.	0.025	.	0.027	.	0.016
P-value	.	0.874	.	0.870	.	0.899
Shea's R-Squared	0.141	0.126	0.141	0.126	0.141	0.126
F-Statistic	8.396	3.858	8.396	3.858	8.396	3.858
p-value	0.002	0.029	0.002	0.029	0.002	0.029
Hansen J Statistic	1.139	1.025	1.886	1.762	0.581	0.523
p-value	0.888	0.906	0.757	0.779	0.965	0.971
Observations	65	65	65	65	65	65

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 15: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth, relative government expenditure growth and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-1.873**	-1.967**	-0.223**	-0.227**	-2.578***	-2.698***
	[0.744]	[0.814]	[0.095]	[0.109]	[0.873]	[0.960]
$\Delta\%PubCons_{i,t-1}$		-0.032		-0.002		-0.042
		[0.072]		[0.018]		[0.103]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
DWH-Statistic	.	0.014	.	0.001	.	0.016
P-value	.	0.906	.	0.972	.	0.899
Shea's R-Squared	0.176	0.162	0.176	0.162	0.176	0.162
F-Statistic	4.882	3.197	4.882	3.197	4.882	3.197
p-value	0.013	0.050	0.013	0.050	0.013	0.050
Hansen J Statistic	4.084	3.658	2.404	2.396	1.129	1.019
p-value	0.395	0.454	0.662	0.663	0.890	0.907
Observations	50	50	50	50	50	50

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 16: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth, number of labor disputes, number of workers involved in labor disputes and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-1.402***	-1.405***	0.132	-0.034	-1.425***	-1.718**
	[0.375]	[0.494]	[0.082]	[0.159]	[0.523]	[0.763]
$Democracies_{i,t-1}$	-0.112*	-0.111	-0.021	-0.032	-0.163*	-0.180*
	[0.063]	[0.074]	[0.037]	[0.034]	[0.087]	[0.102]
$\#Disp_{i,t-1}$		-0.005		-0.002*		-0.010
		[0.007]		[0.001]		[0.009]
$\#Work_{i,t-1}$		-0.000		-0.000		-0.000
		[0.001]		[0.000]		[0.001]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
DWH-Statistic	.	0.000	.	1.169	.	0.154
P-value	.	0.994	.	0.280	.	0.695
Shea's R-Squared	0.246	0.213	0.246	0.213	0.246	0.213
F-Statistic	19.815	16.221	19.815	16.221	19.815	16.221
p-value	0.000	0.000	0.000	0.000	0.000	0.000
Hansen J Statistic	3.479	1.628	5.903	6.154	2.112	0.855
p-value	0.481	0.804	0.207	0.188	0.715	0.931
Observations	51	51	51	51	51	51

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 17: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth, number of days lost in labor disputes and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-2.244***	-1.633***	0.074	-0.009	-2.437***	-1.911***
	[0.536]	[0.430]	[0.168]	[0.145]	[0.714]	[0.431]
$DaysLost_{i,t-1}$		-0.320**		-0.023		-0.368**
		[0.157]		[0.026]		[0.183]
$Democracies_{i,t-1}$	-0.096	-0.045	-0.049	-0.050	-0.187**	-0.135*
	[0.097]	[0.088]	[0.036]	[0.033]	[0.082]	[0.082]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
DWH-Statistic	.	2.458	.	0.373	.	1.674
P-value	.	0.117	.	0.542	.	0.196
Shea's R-Squared	0.158	0.210	0.158	0.210	0.158	.210
F-Statistic	29.303	13.202	29.303	13.202	29.303	13.202
p-value	0.000	0.000	0.000	0.000	0.000	0.000
Hansen J Statistic	0.720	3.225	4.542	3.709	0.454	2.492
p-value	0.949	0.521	0.338	0.447	0.978	0.646
Observations	51	51	51	51	51	51

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 18: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth gross capital formation and other controls.

	$\mathbf{\Delta gdpc_{i,t}}$		$\mathbf{\Delta pop_{i,t}}$		$\mathbf{\Delta gdp_{i,t}}$	
	20-Year Frequency		20-Year Frequency		20-Year Frequency	
	(1)	(2)	(3)	(4)	(5)	(6)
$\widehat{\Delta imr}_{i,t}$	-1.685***	-2.117***	-0.200	-0.163	-2.282***	-2.728***
	[0.317]	[0.419]	[0.137]	[0.139]	[0.418]	[0.597]
$\%GCF_{i,t-1}$		0.022**		-0.002		0.023**
		[0.009]		[0.001]		[0.011]
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes	Yes	Yes
Year Trend	Yes	Yes	Yes	Yes	Yes	Yes
DWH-Statistic	.	3.187	.	0.625	.	1.467
P-value	.	0.074	.	0.429	.	0.226
Shea's R-Squared	0.367	0.340	0.367	0.340	0.367	0.340
F-Statistic	11.123	6.635	11.123	6.635	11.123	6.635
p-value	0.001	0.004	0.001	0.004	0.001	0.004
Hansen J Statistic	1.127	4.984	2.568	2.608	2.454	6.103
p-value	0.890	0.289	0.632	0.625	0.653	0.192
Observations	54	54	54	54	54	54

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 19: First stage of placebo treatment test: Regression of infant mortality growth on the placebo instruments and other controls. The instrument is set equal to one always one period too early.

	Infant Mortality Growth (Δimr$_{i,t}$)			
	20-Year Frequency			
	(1a)	(2a)	(3a)	(4a)
$\Delta Ins_{i,t}$	4.282	3.255	-1.000	3.179
	[4.024]	[4.200]	[2.228]	[4.154]
$\Delta Ins_{i,t} \times PostTrans_{i,t-1}$	-0.116	-0.208	-0.327	-0.211
	[0.116]	[0.125]	[0.186]	[0.125]
$\Delta Ins_{i,t} \times lgdpc_{i,t-1}$	0.014	0.139	0.123	0.142
	[0.181]	[0.175]	[0.302]	[0.174]
$\Delta Ins_{i,t} \times lpop_{i,t-1}$	0.001	-0.004	0.005	-0.004
	[0.021]	[0.020]	[0.046]	[0.020]
$\Delta Ins_{i,t} \times YearTrend$	-0.002	-0.002		-0.002
	[0.002]	[0.002]		[0.002]
$\Delta Ins_{i,t} \times D_{(1920 \leq Year)}$			0.018	
			[0.180]	
$\Delta Ins_{i,t} \times D_{(1920 \leq Year < 1935)}$			0.070	
			[0.159]	
$lgdpc_{i,t-1}$	0.105***	0.210***	0.009	0.217***
	[0.027]	[0.064]	[0.017]	[0.065]
$lpop_{i,t-1}$	0.010	0.092	0.003	0.106
	[0.012]	[0.132]	[0.011]	[0.146]
$PostTrans_{i,t-1}$	-0.058	-0.041	0.051	-0.039
	[0.061]	[0.091]	[0.061]	[0.090]
Year Trend	Yes	Yes	No	
Period Dummies	No	No	Yes	
Country Dummies	No	Yes	Yes	
Joint F-Test ($\Delta Ins\_i, t-1$)	2.864	4.150	3.872	4.062
p-value	0.0680	0.0230	0.0290	0.0250
R-squared	0.725	0.766	0.746	0.767
Observations	84	84	84	84

Results from 2SLS regressions (first stage), the dependent variables are infant mortality growth ($\Delta imr_{i,t}$) and crude death rate growth ($\Delta cdr_{i,t}$), respectively. The instruments are a dummy for the placebo introduction of an universal public health care system (one period too early) and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 20: Second stage placebo treatment test: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth and other controls. The instrument is set equal to one always one period too early.

	GDP per capita Growth (Δgdpc$_{i,t}$)			
	(1a)	(2a)	(3a)	(4a)
$\widehat{\Delta imr}_{i,t}$	1.380	0.973	-0.196	0.914
	[0.936]	[0.617]	[0.846]	[0.621]
Hansen J Statistic	5.752	4.283	2.974	4.294
p-value	0.218	0.369	0.562	0.368
	Population Growth (Δpop$_{i,t}$)			
	(1b)	(2b)	(3b)	(4b)
$\widehat{\Delta imr}_{i,t}$	0.024	0.149	-0.133	0.153
	[0.283]	[0.189]	[0.255]	[0.189]
Hansen J Statistic	2.921	2.590	2.492	2.725
p-value	0.571	0.629	0.646	0.605
	GDP Growth (Δgdp$_{i,t}$)			
	(1c)	(2c)	(3c)	(4c)
$\widehat{\Delta imr}_{i,t}$	1.640	1.307	-0.308	1.244
	[1.083]	[0.798]	[0.933]	[0.803]
Hansen J Statistic	5.944	4.937	3.169	4.974
p-value	0.203	0.294	0.530	0.290
	Specification (all panels)			
	20-Year-Frequency			
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
	First Stage Statistics (all panels)			
Shea's R-Squared	0.053	0.074	0.085	0.075
F-Statistic	2.864	4.15	3.872	4.062
p-value	0.068	0.023	0029	0.025
Observations	84	84	84	84

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the the placebo introduction of an universal public health care system (one period too early) and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 21: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth and other controls (15-years data frequency).

	GDP per capita Growth (Δgdpc$_{i,t}$)			
	(1a)	(2a)	(3a)	(4a)
$\widehat{\Delta imr}_{i,t}$	-0.988***	-0.888**	-0.544	-1.009***
	[0.342]	[0.361]	[0.370]	[0.363]
Hansen J Statistic	5.367	5.532	5.240	5.438
p-value	0.252	0.237	0.513	0.245
	Population Growth (Δpop$_{i,t}$)			
	(1b)	(2b)	(3b)	(4b)
$\widehat{\Delta imr}_{i,t}$	-0.079	-0.108	-0.050	-0.066
	[0.075]	[0.089]	[0.090]	[0.094]
Hansen J Statistic	5.374	5.467	8.413	3.624
p-value	0.251	0.243	0.209	0.459
	GDP Growth (Δgdp$_{i,t}$)			
	(1c)	(2c)	(3c)	(4c)
$\widehat{\Delta imr}_{i,t}$	-1.177***	-1.111***	-0.590	-1.169***
	[0.350]	[0.356]	[0.444]	[0.379]
Hansen J Statistic	3.430	4.196	6.276	3.527
p-value	0.489	0.380	0.393	0.474
	Specification (all panels)			
	15-Year-Frequency			
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
	First Stage Statistics (all panels)			
Shea's R-Squared	0.141	0.147	0.211	0.157
F-Statistic	4.559	3.383	10.677	4.277
p-value	0.017	0.043	0.000	0.021
Observations	109	109	109	109

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

Table 22: Second stage: 2SLS regression of GDP per capita growth, population growth and GDP growth on infant mortality growth and other controls (30-years data frequency).

	GDP per capita Growth (Δgdpc$_{i,t}$)			
	(1a)	(2a)	(3a)	(4a)
$\widehat{\Delta imr}_{i,t}$	-1.929	-2.339**	-1.008	-2.052
	[1.304]	[1.051]	[0.697]	[1.733]
Hansen J Statistic	5.226	4.171	4.000	6.046
p-value	0.265	0.383	0.677	0.196
	Population Growth (Δpop$_{i,t}$)			
	(1b)	(2b)	(3b)	(4b)
$\widehat{\Delta imr}_{i,t}$	-0.168	-0.202	-0.022	-0.186
	[0.206]	[0.216]	[0.320]	[0.207]
Hansen J Statistic	5.546	5.985	6.717	1.805
p-value	0.236	0.200	0.348	0.772
	GDP Growth (Δgdp$_{i,t}$)			
	(1c)	(2c)	(3c)	(4c)
$\widehat{\Delta imr}_{i,t}$	-2.767*	-3.393**	-1.254**	-2.879
	[1.593]	[1.408]	[0.604]	[2.318]
Hansen J Statistic	4.307	3.883	4.234	5.257
p-value	0.366	0.422	0.645	0.262
	Specification (all panels)			
	40-Year-Frequency			
$lgdpc_{i,t-1}$	Yes	Yes	Yes	Yes
$lpop_{i,t-1}$	Yes	Yes	Yes	Yes
$PostTrans_{i,t-1}$	Yes	Yes	Yes	Yes
Year Trend	Yes	No	No	Yes
Country Specific Year Trend	No	Yes	No	No
Period Dummies	No	No	Yes	No
Country Specific Constants	No	No	No	Yes
	First Stage Statistics (all panels)			
Shea's R-Squared	0.237	0.275	0.166	0.224
F-Statistic	4.718	5.511	72.359	3.109
p-value	0.015	0.009	0.000	0.054
Observations	49	49	49	49

Results from 2SLS regressions, the dependent variables are GDP per capita growth ($\Delta gdpc_{i,t}$), population growth ($\Delta pop_{i,t}$) and GDP growth ($\Delta gdp_{i,t}$), respectively. The instruments are a dummy for the introduction of an universal public health care system and interactions (see text). Robust standard errors are in brackets; ***, **,* indicate significance at 1-, 5-, and 10-percent level, respectively.

A History of Public Health Insurance

Table 23: Introduction dates of a universal public health care system.

Country	Introduction	Source
Austria	1887	(Hofmarcher and Rack, 2001, p. 6)
Belgium	1894	(Corens, 2007, p. 15)
Denmark	1892	(Strandberg-Larsen et al., 2007, p. 19)
Finland	1944	(Vuorenkoski, 2008, p. 21)
France	1930	(Sandier, Paris, and Polton, 2004, p. 7)
Italy	1923	(Lo Scalzo et al., 2009, p. 17)
Netherlands	1941	(Schäfer et al., 2010, p. 13)
Norway	1912	(Johnsen, 2006, p. 13)
Spain	1942	(Durán, Lara, and Van Waveren, 2006, p. 15)
Sweden	1955	(Glenngård et al., 2005, p. 15)
Switzerland	1911	(Minder, Schoenholzer, and Amiet, 2000, p. 6)
United Kingdom	1948	(Robinson and Dixon, 1999, p. 5)

Austria: The industrial accident and health insurance scheme for workers was introduced in **1887**. It follows the model of Bismarck's social policy program in Germany. It is the foundation of today's social security system (comp. Hofmarcher and Rack, 2001, p. 6).

Belgium: Workers created mutual benefit societies in the late 19th century, in order to protect affiliated members against the risk of disease, unemployment and incapacity to work. These early voluntary sickness funds were of small scale, organized according to employment type, and run as private initiatives, without state subsidies. The legislation for a sickness funds', which served as the legal foundation for about a century, was passed in **1894**. This legislation extended the official scope of the sickness funds' activities and introduced state subsidies (comp. Corens, 2007, p. 15).

Denmark: During the second half of the 19th century, health insurance in Denmark developed. Health insurance organizations were established by a combination of artisans and other groups. The artisan groups created their own help funds as an extension of the guilds funds, which were established by members to provide mutual help. Since **1892**, state subsidies are given to insurance schemes (comp. Strandberg-Larsen, Nielsen, Vallgårda, Krasnik, and Vrangbæk, 2007, p. 19).

Finland: Since the 1870s, municipalities have been responsible for providing basic medical services. After the War, a new act was introduced to organize municipal health care services. The right to maternal and child health care, irrespective of residence and financial situation, was established in **1944** (comp. Vuorenkoski, 2008, p. 21).

France: An act on social insurance was passed in **1930**, signalling the emergence of a universal insurance system. A system of compulsory protection for employees in industry and business, whose earnings fell below a certain level, was created by this legislation. Insurance was provided in five areas: illness, maternity, disability, old age and death (comp. Sandier, Paris, and Polton, 2004, p. 7).

Italy: In 1898, occupational accident insurance was introduced and in 1904 it became compulsory for workers in industry and in 1917 for agriculture. The fascist regime (1922-1943), pushed several changes in the health care system forward. In **1923**, the right to hospital care for the needy and indigent population was guaranteed for the first time (comp. Lo Scalzo, Donatini, Orzella, Cicchetti, Profili, and Maresso, 2009, p. 17).

Netherlands: The liberal Dutch traditions of the 19th century prevented the government from taking initiatives related to health care and health insurance. The 1913 sickness act marked the the start of government interference in the health insurance sector. However, it took decades before a system of health insurance came into operation. After many political conflicts, implementation of the 1913 sickness act was not achieved until 1930. Eventually, the act only covered sickness benefits, excluding medical expenses. Until the Second World War all attempts to introduce a compulsory insurance system failed. This was caused by resistance from health care providers. The main causes of conflict were provider participation in the sickness fund boards, the level of the income threshold for people to be accepted to the funds and the conditions of being accepted as a health care provider. A change was forced by the German occupying forces in **1941** with the constraint of the sickness fund decree. It introduced compulsory insurance for employees earning less than a certain income threshold (comp. Schäfer, Kroneman, Boerma, Van den Berg, Westert, Devillé, and van Ginneken, 2010, p. 13).

Norway: An increase in public responsibility for health matters, at the state and the municipal levels, took place the beginning of the 20th century. At first, health care insurance schemes developed, based on individual applications. The practitioners act of **1912** provided equal access to health services for everyone, regardless of their income and settlement (comp. Johnsen, 2006, p. 13).

Spain: During the last quarter of the 19th century, the development of Spanish social protection began. The National Institute of Social Insurance (INP) was created to coordinate implementation of the first social insurance policies. The first attempt to develop social health insurance for low-salaried workers was launched by the INP during the era of the Second Republic (1931-1936). In 1936, the coup by General Franco started a civil war (1936-1939) that led to the establishment of an authoritarian regime. After the civil war, many of the previous policy proposals were in some way recovered by the Francoist Government. Social security-related health care was run through the INP from **1942** by the Ministry of Labour and Social Security, until 1977 (comp. Durán, Lara, and Van Waveren, 2006, p. 15).

Sweden: The first important step towards universal coverage for physician consultations, prescription drugs and sickness compensation was taken, when a National Health Insurance Act was voted in by the Swedish Parliament in **1946**

(comp. Glenngård, Hjalte, Svensson, Anell, and Bankauskaite, 2005, p. 15).

Switzerland: The cantons and municipalities were almost exclusively responsible for health services at the inception of Switzerland in 1848. An attempt to introduce a universal system of health insurance was made in 1899 with the tabling of a health and accident insurance law. The first proposal was rejected by referendum so the proposals were changed and resubmitted. Finally, the legislation was passed by a second referendum in **1911**. The health insurance allowed insurance funds to take advantage of federal subsidies (comp. Minder, Schoenholzer, and Amiet, 2000, p. 6).

United Kingdom: The National Health Service (NHS) came into operation in **1948**. This Act was important in creating the pattern of post-Second World War health service provision in the UK. It established the principle of collective responsibility for health services, which was available to the entire population, independent of individual income, without costs at the point of use (comp. Robinson and Dixon, 1999, p. 5).