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Abstract

This study documents that the growth in life expectancy over the 20th century decreased per capita GDP growth and increased population growth. By exploiting significant advances in medical technologies, starting to diffuse in the 1940s, the analysis establishes that countries with higher levels of infectious-disease mortality prior to the medical breakthrough experienced higher growth rates in life expectancy and population size, and lower growth rates in per capita GDP in the time after the medical breakthroughs. These findings are robust to the inclusion of initial life expectancy and initial GDP per capita. The evidence presented here therefore complements the conclusions inferred in the research by Acemoglu and Johnson (2007).

Key Words: Life expectancy; Health shock; Long-run economic growth.

JEL: I10; O40; J11.

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1 Introduction

How the rise of life expectancy during the second half of 20th century affected the economic development of nations is debated in the macro-empirical literature. While most studies find positive cross-country correlations between life expectancy and GDP per capita,[1] the research by Acemoglu and Johnson (2007), henceforth AJ, which exploits exogenous sources of within-country variations in life expectancy, demonstrates that the level of life expectancy increased population size but lowered the level of GDP per capita. In a recent article, Bloom et al. (2013), henceforth BCF, however, argue that the empirical model in AJ is misspecified and that including initial life expectancy and initial GDP per capita inverts their conclusions.

The concern raised by BCF is that life expectancy in 1940 (initial life expectancy) affected subsequent growth rates in per capita GDP. They argue that due to mean reversion in life expectancy, the analysis in AJ underestimates the effect of life expectancy on GDP per capita. While, in theory, one might reason that initial life expectancy is an omitted variable in AJ's baseline empirical model, it cannot be tested the way proposed by BCF. In particular, the present paper shows that their theoretical argument is not testable in panel including only two data points per country with country fixed effects. This test requires at least three data points per country (see section 2). In other words, AJ's original dataset, which is utilized in the BCF study, only have the dimensions to study how the level of life expectancy affects the level of GDP per capita if one at the same time wants to control for country fixed effects.

The conclusion of this paper is therefore derived from a panel including the data points 1900, 1940, and 1980, which we use to calculate the growth rates in GDP per capita and life expectancy over the periods: 1900–1940 and 1940–1980 for each country. Thus, the additional data point in 1900, in contrast to BCF, leaves the possibility to eliminate the country fixed effects as we end up with two data points per country *after* calculating the growth rates in life expectancy and GDP per capita.

[1] An incomplete list of some important studies includes Sala-i-Martin (1997), Sachs and Warner (1997), Bloom et al. (2004), Zhang and Zhang (2005), Tamura (2006), Weil (2007), Murphy et al. (2008), and Lorentzen et al. (2008). The study by Dalgaard and Strulik (2013) demonstrates that the reverse mechanism, i.e. the effect from income to health, might explain a large share of the positive cross-country correlations between life expectancy and GDP per capita.

While data availability on GDP per capita in 1900 reduces the sample to 35 countries, the main finding of this paper shows that the growth rate of life expectancy decreases the growth rate of GDP per capita. The estimate is robust to controlling for initial log life expectancy and initial log GDP per capita. Consistent with these results, the analysis also uncovers statistically significant positive effects of the growth rate in life expectancy on population growth for the full sample of 47 countries. In both models, the instrumental variable—which we argue follows the same logic as a *difference-in-differences* estimator with treatment measured as a continuous variable—has strong predictive power for the growth rate of life expectancy. But, as the only instrumental variable present is instrumenting for the growth rate of life expectancy, we must rely on initial life expectancy (i.e., lagged life expectancy) being exogenous conditional on country and time fixed effects. While this assumption is questionable, the OLS estimate of lagged log life expectancy on per capita GDP growth is negative in all the specifications and, in most specifications, the effect is also statistically significant. Moreover, we show that the 2SLS estimate of the growth rate of life expectancy remains negative even without initial life expectancy in the model. In sum, the evidence strongly suggests that both the level and the growth rate of life expectancy had negative effects on the growth rate of GDP per capita.

The rebuttal by Acemoglu and Johnson (2013) demonstrates that the original AJ-results remain robust to controlling for initial life expectancy in a 10-year panel model framework. Specifically, they include life expectancy in 1900 interacted with time fixed effects. This should soak up any mean reversion in life expectancy, and this approach is meaningful if one wants to control for initial life expectancy. However, the estimated coefficient quantifies the level effect of life expectancy on GDP per capita conditional on country and time fixed effects. Our study is the first to quantify the growth effect of life expectancy on per capita GDP growth conditional on country and time fixed effect, and the results are consistent with the negative level effects reported in Acemoglu and Johnson (2013). This also suggests that the BCF concern is addressed by taking out the country fixed effect in the AJ study.

In the empirical framework of AJ, the first-stage estimates compare the log level of life expectancy before and after the health shock, which occurred due to the breakthrough of new medical technologies such as, e.g., antibiotics. Their reduced-form estimates make a similar comparison but with the log level of GDP per capita as the outcome variable. In our study,

the first-stage estimates compare the growth rate of life expectancy before and after the health shock, and our reduced-form estimates compare the growth rate of GDP per capita before and after the health shock.

The results of the present study, thus, confirm the overall conclusion in the AJ study: the 20th century rise in the health of nations did not narrow the vast inequalities in GDP per capita. When reflecting on this conclusion, however, it is important to note that other research has demonstrated that the same population health improvements led to increases in other indicators of economic development; for example, human-capital outcomes (Hansen, 2013a, 2013b).

Aghion et al. (2010) develop a theoretical model which, in fact, shows that the growth of per capita GDP depends upon both the level and the growth rate of life expectancy. This model provides a theoretical foundation for the BCF study. Aghion et al., however, take the theoretical predictions of the model to the data in a cross-country framework—i.e., without country fixed effects—and find that both levels and growths rates are positively related to the growth of per capita GDP. Exploiting within-country variation instead, along with the IV strategy proposed by AJ, we arrive at the opposite conclusion.

In various ways, previous studies have attempted to clarify why AJ's within-country results are contradictory to the prevailing view in the cross-country literature, arguing that the health-income relationship is positive. For example, Cervellati and Sunde (2011a; 2011b) argue that the effect of life expectancy on GDP per capita is non-monotonic. Exploiting the same empirical setup as AJ, their analysis reveals that the impact of life expectancy on GDP per capita is negative, but statistically insignificant, before the onset of the demographic transition, whereas after its onset the effect is positive and significant.

The paper is organized as follows. Section 2 shows that the estimates reported in BCF must be interpreted as being unconditional on country fixed effects. Section 3 describes the dataset. Section 4 outlines the empirical strategy and presents the main results of the paper. Section 5 concludes.

2 On the interpretation of the BCF estimates

This section explains why the estimates derived in BCF cannot be interpreted as how changes in log life expectancy (i.e., growth rate of life expectancy) and the level of initial life expectancy affect changes in log GDP per capita (i.e., growth rate of GDP per capita) with country fixed effects.

In this paper, we argue that the test proposed by BCF is possible to implement when following the empirical framework of AJ, however, it requires a panel including at least three data points per country (e.g., 1900, 1940, and 1980) and not only two data points per country (i.e., 1940 and 1980) as are the time dimensions of the original dataset in AJ, which is later utilized by BCF to test their growth model.

Let us start by substantiating the argument that the estimates reported in BCF actually are unconditionally on country fixed effects. The original structural model in AJ is given by the following equation:

$$y_{it} = \beta x_{it} + \zeta_i + \mu_t + \epsilon_{it} \tag{1}$$

where y_{it} is log GDP per capita, x_{it} is log life expectancy at birth, ζ_i and μ_t are country and time fixed effects, and ϵ is the error term. The coefficient β is as such not estimable as the country fixed effects are generally not observed. However, it is possible to eliminate the country fixed effects from the error by means of first differencing (or by including country dummies). Doing this provides the following estimation equation:

$$\Delta y_{it} = \beta \Delta x_{it} + \Delta \mu_t + \Delta \epsilon_{it}, \tag{2}$$

where $\Delta y_{it} \equiv y_{it} - y_{it-1}$, $\Delta x_{it} \equiv x_{it} - x_{it-1}$, and so on. Now it is important to note that (2) is an *estimation equation*, and the interpretation of the estimated coefficient $\hat{\beta}$ should be related to the structural model in eq. (1): $\hat{\beta}$ provides an estimate of how the log level of life expectancy affects the log level of GDP per capita conditional on country and time fixed effects.[2] The interpretation should therefore *not* be related to the estimation equation (2), that is, $\hat{\beta}$ does *not* provide an estimate of how the growth in life expectancy influences the growth in GDP per capita controlling for country fixed effects.

[2] See e.g. Wooldridge 2002, pp.267 and 279.

To test their hypothesis, BCF add initial log life expectancy, x_{it-1}, and initial log GDP per capita, y_{it-1}, to the estimation equation in (2):

$$\Delta y_{it} = \alpha_t + \beta \Delta x_{it} + \gamma x_{it-1} - \lambda y_{it-1} + \varepsilon_{it}, \tag{3}$$

where $\alpha_t \equiv \Delta \mu_t$ and $\varepsilon_{it} \equiv \Delta \epsilon_{it}$. This approach is not correct: if BCF have a theoretical reason to believe that initial life expectancy and initial GDP per capita should have been included in the empirical model, they should have included these variables in the structural model given by eq. (1).

Now suppose that the "true"specification has the following form:

$$y_{it} = \beta x_{it} + \gamma x_{it-1} - \lambda y_{it-1} + \zeta_i + \mu_t + \epsilon_{it},$$

where x_{it-1} and y_{it-1} have been added to the structural model in eq. (1) instead of the estimation in eq. (2). This equation is equivalent to:

$$\Delta y_{it} = \Lambda y_{it-1} + \beta \Delta x_{it} + \Gamma x_{it-1} + \zeta_i + \mu_t + \epsilon_{it}, \tag{4}$$

where $\Lambda \equiv -(1+\lambda)$ and $\Gamma \equiv (\gamma + \beta)$. When the country fixed effects, ζ_i, are unobserved, estimating the coefficients Λ, β and Γ necessitates a panel with at least three data points. For example, in a panel including the dates 1900, 1940, and 1980, we are able to calculate the growth rates in GDP per capita and life expectancy over the two time periods: 1900–1940 and 1940–1980. This now leaves us with two data points per country, which can be used to separate country fixed effects, ζ_i, from the error term, ϵ_{it}. This shows that it is as such *not* possible to estimate the growth model as proposed by BCF based on only two data points per country with country fixed effects.

The estimates reported in BCF can be understood in terms of the subsequent empirical model:

$$\Delta y_i = \bar{\lambda} y_{i,1940} + \bar{\beta} \Delta x_i + \bar{\gamma} x_{i,1940} + \bar{\epsilon}_i \tag{5}$$

where $\Delta z_i = \log z_{i,1980} - \log z_{i,1940}$ for $z_i = y_i, x_i$ and $\bar{\epsilon}_i$ is the error term, which includes unobserved country specific characteristics. This model corresponds to the standard cross-country growth-regression model. In the framework of eq. (4), country specific characteristics are captured by country dummies (or by first differencing). However, because eq. (5) is estimated

on the basis of only one observation per country, an additional necessary condition for the OLS estimate of $\bar{\beta}$ to be unbiased and consistent is that the growth rate of life expectancy (or log change) is orthogonal to the country specific characteristics, which is not likely to be the case. Moreover, as the instrumental variable developed by AJ is derived upon the logic of a difference-in-differences estimator, their IV approach does not resolve this issue, that is, the validity of AJ's IV strategy hinges upon the ability to control for country fixed effects.

Conceptually, the first-stage estimates in AJ's empirical framework compare the log level of life expectancy before and after the health shock, which is similar to a *difference-in-differences* estimation strategy. The reduced-form estimates, in this analysis, make a similar comparison but with the log level of GDP per capita as the dependent variable. In order to empirically test the general idea proposed by BCF, section 4.1 outlines a model where the first-stage estimates compare the growth rate of life expectancy before and after the health shock, and the reduced-form estimates compare the growth rate of GDP per capita before and after the health shock.

3 Data

This section describes the dataset. The analysis focuses on two outcome variables: the growth rate of GDP per capita and the population growth rate. As discussed in section 2, if we want to calculate the growth rates of the variables and still be able to eliminate country fixed effect, the panel must include at least three data points per country. To maximize the number of countries included in the sample, we consider the dates: 1900, 1940, and 1980 (or 2000). This implies that we can calculate the growth rates of GDP per capita and population size for the two periods: 1900–1940 and 1940–1980 (or 1940–2000). These two data points per country can then be used to control for country-specific factors by, for example, including a full set of country dummies. These data come from Maddison (2001), Acemoglu and Johnson (2007), and Goldewijk et al. (2010). The explanatory variable is the growth rate of life expectancy at birth, which is calculated over the same time periods, obtained from the UN Demographic Yearbook.

The empirical strategy relies on measuring the intensity of the health shock caused by the wave of medical innovations from the 1940s to the mid-1950s. In order to capture this aspect, data on preintervention mortality rates (deaths per 100s) of up to 15 infectious diseases are

collected from Acemoglu and Johnson (2007). They include: malaria, pneumonia, tuberculosis, influenza, cholera, smallpox, shigella, whooping cough, typhus, plague, yellow fever, scarlet fever, diphtheria, measles, and typhoid. The common factor of the diseases is that they became treatable as a result of the medical innovations around this period of time. Because the baseline analysis is performed using two data point per country (i.e., 1900–1940 and 1940–1980), the postintervention period is 1940–1980 (or 1940–2000).

Due to data availability on GDP per capita in 1900, the base sample consists of 35 countries. However, when considering the specifications with the population growth rate as the outcome variable, the sample is increased to 47 countries, which constituted the base samples in AJ and BCF.

4 Empirical strategy and results

4.1 Empirical strategy

The second-stage regression follows directly from eq. (4):

$$gy_{it} = \Lambda y_{it-1} + \beta g x_{it} + \Gamma x_{it-1} + \zeta_i + \mu_t + \epsilon_{it}, \tag{6}$$

where instead of log differences, we have used actual growth rates of per capita GDP and life expectancy.[3] Specifically, $gy_{it} = e^{y_{it}-y_{it-1}} - 1$ is the growth rate of GDP per capita, y_{it} is the log of GDP per capita, $gx_{it} = e^{x_{it}-x_{it-1}} - 1$ is the growth rate of life expectancy, x_{it} is the log of life expectancy at birth, ζ_i and μ_t are country and time fixed effects, and ϵ_{it} is the error term. We also report results from specifications where the dependent variable is the population growth rate and the lagged dependent variable is, therefore, log population size.

Because of problems with omitted variables and reverse causality, OLS estimates of β in eq. (6) are not likely to provide the causal effect of the growth rate of life expectancy on the per capita GDP growth rate. The empirical strategy exploits the international epidemiological transition as a source of exogenous variation in the growth rate of life expectancy. In particular,

[3] We use actual growth rates since differences of logs are imprecise approximations for the long-run growth rates (i.e., the changes in z are relatively large). However, we obtain qualitatively similar results when using log differences.

the subsequent first-stage relationship between the growth rate of life expectancy and the health shock is used:

$$gx_{it} = \alpha \sum_{d=1}^{15} M_{di40} \times I_t + \Lambda y_{it-1} + \Gamma x_{it-1} + \zeta_i + \mu_t + \tilde{\epsilon}_{it}, \tag{7}$$

where M_{di40} is the 1940 death rate of disease d in country i, I_t is an indicator variable which equals one in the period after the medical breakthroughs (i.e., the period 1940–1980).[4] Thus, the instrumental variable that we use (i.e., the interaction $\sum M_{di40} \times I_t$) is equal to zero in the first period 1900–1940 and equal to the sum of the mortality rates of the 15 infectious diseases in 1940 for the second period 1940–1980. This is a difference-in-differences estimator where treatment is measured as a continuous variable. We think of this as similar to the estimators used in e.g. Bleakley (2007), Lucas (2010) and Nunn and Qian (2011).

It is important to note that the sample variance in the shock variable, $\sum M_{di40} \times I_t$, is identical to the sample variance in the predicted-mortality instrument developed by Acemoglu and Johnson (2007). The only difference is the sign in the first-stage regressions. Thus, if the health shock increased the growth rate of life expectancy then the estimate on the health shock will be positive (i.e., $\alpha > 0$).

As has been argued in section 2, the first-stage estimates in our empirical analysis compare the growth rate of life expectancy before and after the health shock, whereas the first-stage estimates in AJ compare the level of life expectancy before and after the health shock. In other words, the instrumental variable that we utilize is essentially identical to the predicted-mortality variable developed by AJ, but they use it on first differences in life expectancy, while we use it on second differences in life expectancy.

4.2 Results

Tables 1 and 2 report OLS and IV estimates of eqs. (6) and (7) with the growth rate of GDP per capita as the outcome variable. The first three columns focus on the periods 1900–1940 and 1940–1980, whereas the latter three columns present results from a model where the post-intervention period is changed to 1940–2000.

[4] Because the date of the actual diffusion of the medical technologies could be driven by endogenous aspects such as public health institutions, the strategy uses the same intervention dates for all countries in the sample.

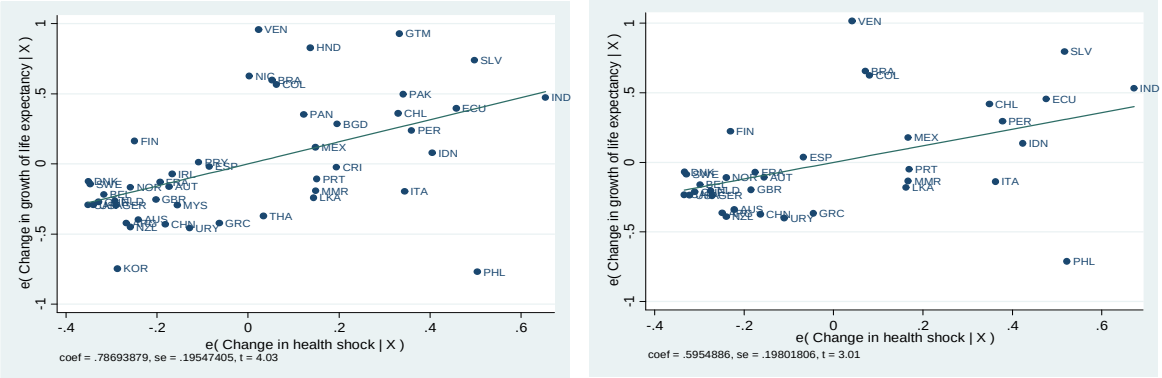
Conditioning on country and time fixed effects, column 1 of table 1 shows that the correlation between the growth rate of life expectancy and the per capita GDP growth rate is negative. Taken at face value, the estimate suggests that a 10-percentage-point increase in the growth rate of life expectancy lowers the per capita GDP growth rate by 6.7 percentage points. This relationship increases in magnitude and becomes statistically significant when adding the lagged log level of life expectancy (initial life expectancy) in column 2. Moreover, the coefficient estimate on initial life expectancy is negative and significant at the 10 percent level. This shows that both the level and the growth rate of life expectancy are negative related to GDP per capita growth when conditioning on country and time fixed effects. Column 3 demonstrates that this conclusion is robust to the inclusion of the lagged log level of GDP per capita. Finally, the remaining three columns of table 1 report similar estimates when using 1940–2000 as the post-intervention period.

Panel A in table 2 reports the corresponding 2SLS estimates for the growth of life expectancy, while panel B shows the first-stage results as provided by eq. (7). Remembering that the IV used here ("Health shock") is defined inversely of the predicted-mortality instrument in AJ, the coefficient estimates in all the specifications, which are statistically significant at the 1 percent level, indicate the medical breakthroughs substantially increased the growth rate of life expectancy. Thus, the strong F-statistics from the first-stage regressions, presented in the bottom of table 2, provide verification for the significance of the IV strategy proposed by AJ. This finding stands in contrast to the claim made in BCF that the IV estimator, proposed by AJ, is no longer identified. The partial correlation plot, illustrating the first-stage relationship between the growth rate of life expectancy and the health shock, is shown in figure 1—the left hand side shows the relationship for the full sample of 47 countries, whereas the right hand side shows it for the 35 countries, which constitute the sample iwhen the outcome variable is the growth rate of GDP per capita. As we see from panel A, the estimated causal effect of life expectancy growth on per capita GDP growth is substantially stronger in comparison to the OLS correlations reported in table 1. Specifically, increasing the growth rate of life expectancy by 10 percentage points decreases the growth rate of GDP per capita by about 20 percentage points over a 40-year horizon, corresponding to 0.46 percentage point per year on average. Figure 2 shows the reduced-form partial correlation plot between the growth rate of GDP per

capita and the health shock.

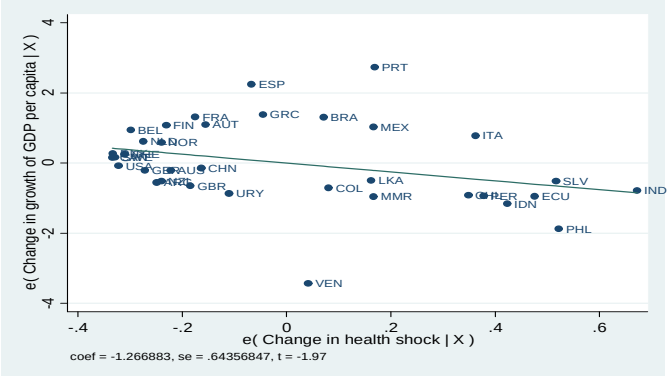
Tables 3 and 4 also report OLS and IV estimates but with the population growth rate as the outcome variable. The structure of the tables follows that of the preceding tables. Because of data availability, the sample increased to 47 countries, which constituted the base samples in AJ and BCF. Consistent with the estimates presented thus far, we observe that both the level and the growth rate of life expectancy have positive and statistically significant effects on the population growth rate. For example, according to the 2SLS estimate reported in column 1 of table 4 a 10-percentage-point increase in the growth rate of life expectancy increases population growth by 17 percentage points over a 40-year period. Finally, figure 3 demonstrates the reduced-form partial correlation plot between the population growth rate and the health shock.

Figure 1: Growth rate of Life Expectancy and the Health Shock



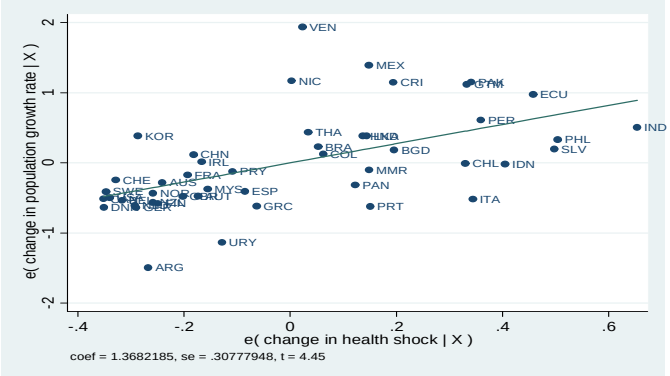
Notes: Partial correlation plot between the Δ in the growth rate Life Expectancy and the Δ Health Shock$_{15}$. LHS: sample with 47 countries. RHS: sample with 35 countries.

Figure 2: Growth rate of GDP per capita and the Health Shock



Notes: Partial correlation plot between the Δ in the growth rate of GDP per capita and the Δ in Health Shock$_{15}$. Sample with 35 countries.

Figure 3: Population growth rate and the Health Shock



Notes: Partial correlation plot between the Δ in the population growth rate and the Δ in Health Shock$_{15}$. Sample with 47 countries.

5 Conclusion

We argued for, and showed the necessity of, at least three data points per country to test the effect of both the growth rate of life expectancy and the initial level of life expectancy on subsequent growth rates in GDP per capita. Using the proposed setup, we find that higher growth rates in life expectancy decreases the subsequent growth rate in GDP per capita. It is important to note that this finding does not preclude positive effects from health to income at the individual level. Indeed, if the channels that causes GDP per capita growth rates to decrease from higher growth rate in life expectancy operates at the macro level, the finding of the present paper does not contradict micro studies finding positive effect of health on income.

Why should findings at the micro and macro level differ? As in Acemoglu and Johnson (2007) the result can be interpreted as the consequence of Malthusian forces: if countries who benefitted most of the introduction of modern medicine relied more on fixed factors of production, then higher growth rates in life expectancy, which we show entail higher population growth rates, tend to lower workers' productivity. If this is the case, the effect of the growth rate in life expectancy on the growth rate of GDP per capita depends both on how it affects population growth and the extent to which the economy relies on fixed factors of production.

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Table 1—Per Capita GDP Growth: OLS Estimates

	Dependent variable is the growth rate of GDP per capita					
	40-year periods			60-year periods		
	(1)	(2)	(3)	(4)	(5)	(6)
Life expectancy growth rate	-0.674	-1.762**	-1.750***	-1.484*	-1.562**	-1.196**
	(0.708)	(0.868)	(0.587)	(0.797)	(0.735)	(0.546)
Log life expectancy initial		-2.950*	-4.908***		-1.791	-3.278
		(1.594)	(1.364)		(2.015)	(2.246)
Log GDP/capita initial			-2.392***			-1.283*
			(0.535)			(0.719)
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
# of observations	70	70	70	70	70	70
# of countries	35	35	35	35	35	35

Notes: The table reports OLS estimates. The dependent variable is the growth rate in GDP per capita. Life expectancy growth is the growth rate in life expectancy at birth. The preintervention periods measure growth over 1900–1940, whereas columns 1–3 measure growth over 1940–1980 and columns 4–6 measure growth over 1940–2000. Initial log life expectancy and initial log GDP per capita are levels measured in the beginning of the periods (i.e., 1900 and 1940). Standard errors are clustered at the country level.
*** p<0.01, ** p<0.05, * p<0.1.

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Table 2—Per Capita GDP Growth: 2SLS Estimates

	Dependent variable is the growth rate of GDP per capita					
	40-year periods			60-year periods		
	(1)	(2)	(3)	(4)	(5)	(6)
	A. Second-stage results					
Life expectancy growth rate	-2.127*	-2.398***	-2.101***	-2.620**	-2.085**	-2.034**
	(1.186)	(0.927)	(0.736)	(1.318)	(0.915)	(0.850)
Log life expectancy initial		-4.114	-5.550***		-1.952	-3.211
		(2.518)	(1.474)		(2.144)	(2.300)
Log GDP/capita initial			-2.391***			-1.022*
			(0.509)			(0.591)
	B. First-stage results					
	Dependent variable is life expectancy growth rate					
Health shock	0.595***	0.526***	0.529***	0.821***	1.075***	1.060***
	(0.214)	(0.081)	(0.074)	(0.237)	(0.174)	(0.204)
Log life expectancy initial		-1.768***	-1.802***		-1.127***	-0.757
		(0.233)	(0.216)		(0.332)	(0.489)
Log GDP/capita initial			-0.041			0.287
			(0.198)			(0.191)
Kleibergen-Paap F-statistic	7.73	42.34	50.70	11.97	37.78	26.81
Anderson-Rubin test [p-value]	[0.0012]	[0.0019]	[0.0084]	[0.0041]	[0.0088]	[0.0067]
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
# of observations	70	70	70	70	70	70
# of countries	35	35	35	35	35	35

Notes: The coefficients on the life expectancy growth rate are estimated by 2SLS, whereas the remaining estimates are OLS. The dependent variables is the growth rate in GDP per capita. Life expectancy growth is the growth rate in life expectancy at birth. The preintervention periods measure growth over 1900–1940, whereas columns 1–3 measure growth over 1940–1980 and columns 4–6 measure growth over 1940–2000. Initial log life expectancy and initial log GDP per capita are levels measured in the beginning of the periods (i.e., 1900 and 1940). Health Shock is coded on the basis of 15 infectious diseases

Standard errors are clustered at the country level.

*** p<0.01, ** p<0.05, * p<0.1

Table 3—Population Growth: OLS Estimates

	Dependent variable is the growth rate of population size					
	40-year periods			60-year periods		
	(1)	(2)	(3)	(4)	(5)	(6)
Life expectancy growth rate	0.966***	1.975***	2.143***	1.925***	2.177***	1.830***
	(0.228)	(0.254)	(0.277)	(0.341)	(0.298)	(0.319)
Log life expectancy initial		2.539***	2.901***		2.441***	1.398**
		(0.542)	(0.561)		(0.685)	(0.567)
Log Population initial			-0.459*			0.984***
			(0.245)			(0.345)
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
# of observations	94	94	94	94	94	94
# of countries	47	47	47	47	47	47

Notes: The table reports OLS estimates. The dependent variable is the growth rate in population size. Life expectancy growth is the growth rate in life expectancy at birth. The preintervention periods measure growth over 1900–1940, whereas columns 1–3 measure growth over 1940–1980 and columns 4–6 measure growth over 1940–2000. Initial log life expectancy and initial log population are levels measured in the beginning of the periods (i.e., 1900 and 1940). Standard errors are clustered at the country level.

*** p<0.01, ** p<0.05, * p<0.1.

Table 4—Population Growth: 2SLS Estimates

	Dependent variable is the growth rate of population size					
	40-year periods			60-year periods		
	(1)	(2)	(3)	(4)	(5)	(6)
	A. Second-stage results					
Life expectancy growth rate	1.739***	2.265***	2.414***	2.787***	2.398***	2.154***
	(0.461)	(0.443)	(0.527)	(0.694)	(0.477)	(0.455)
Log life expectancy initial		3.076***	3.411***		2.618***	1.767**
		(0.936)	(1.125)		(0.875)	(0.776)
Log population initial			-0.516**			0.845**
			(0.252)			(0.330)
	B. First-stage results					
	Dependent variable is life expectancy growth rate					
Health shock	0.786***	0.557***	0.536***	1.031***	1.228***	1.134***
	(0.207)	(0.092)	(0.096)	(0.227)	(0.175)	(0.176)
Log life expectancy initial		-1.704***	-1.725***		-1.283***	-1.453***
		(0.164)	(0.165)		(0.271)	(0.270)
Log population initial			0.132**			0.265***
			(0.060)			(0.093)
Kleibergen-Paap F-statistic	14.32	36.51	31.01	20.55	49.29	41.25
Anderson-Rubin test [p-value]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]	[0.0000]
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
Time fixed effects	Yes	Yes	Yes	Yes	Yes	Yes
# of observations	94	94	94	94	94	94
# of countries	47	47	47	47	47	47

Notes: The coefficients on the life expectancy growth rate are estimated by 2SLS, whereas the remaining estimates are OLS. The dependent variable is the growth rate in population size. Life expectancy growth is the growth rate in life expectancy at birth. The preintervention periods measure growth over 1900–1940, whereas columns 1–3 measure growth over 1940–1980 and columns 4–6 measure growth over 1940–2000. Initial log life expectancy and initial log population size are levels measured in the beginning of the periods (i.e., 1900 and 1940). Health Shock is coded on the basis of 15 infectious diseases

Standard errors are clustered at the country level.

*** p<0.01, ** p<0.05, * p<0.1