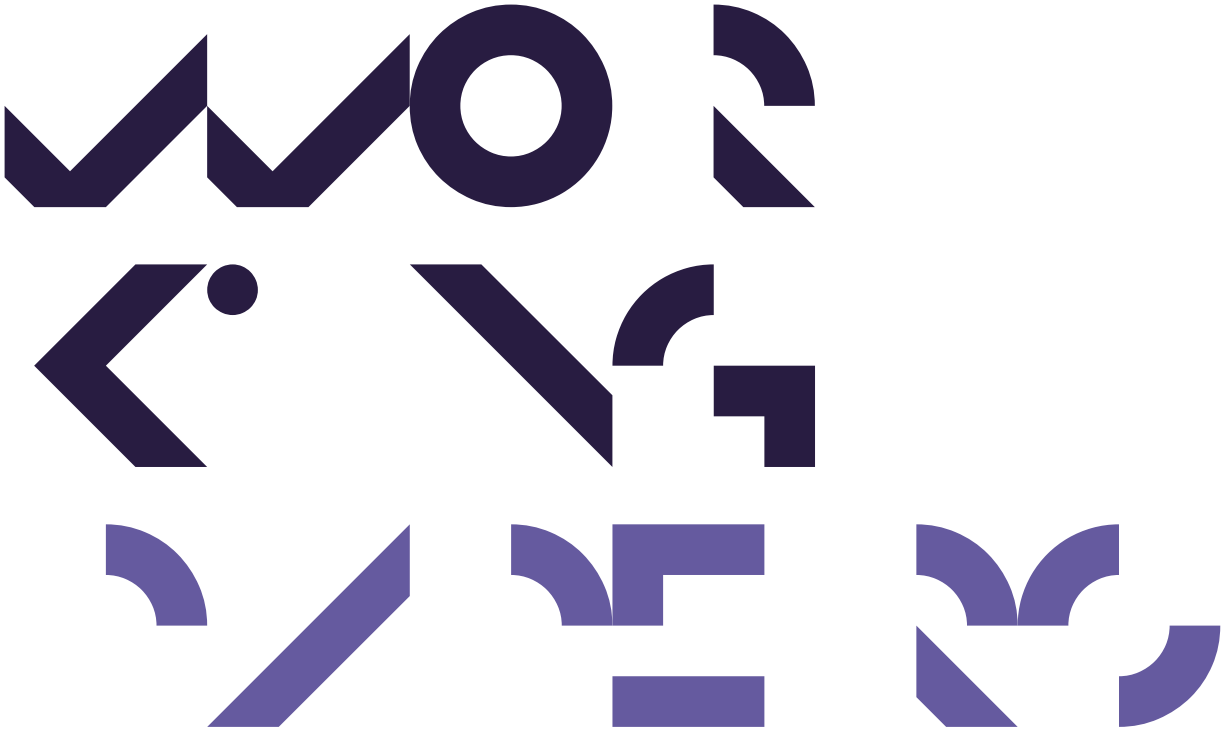

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Causes of mortality and development: Evidence from large health shocks in 20th century America

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Causes of mortality and development: Evidence from large health shocks in 20th century America

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Abstract

Exploiting pre-intervention variation in flu/pneumonia, tuberculosis and maternal mortality, together with time variation arising from medical breakthroughs starting in the late 1930s, this paper studies the aggregate impact of large health shocks across US states. The analysis demonstrates that the shocks influenced income per capita in different ways. While the shock to flu/pneumonia mortality has been conductive for development, the large reduction in the incidence of tuberculosis deaths has been a negative force in the development of US states over the second-half of 20th century. In addition, the decline in maternal mortality has a fragile, but positive relationship with income per capita. Because these specific health shocks affected mortality across the life cycle differently, the evidence here underscores the general tenet of regarding health as multifaceted.

Key Words: Economic development; Mortality; Population growth; Large health shocks; Medical innovations; US states.

JEL: I15; J24; O11; O51.

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1 Introduction

While the last century marked an unprecedented increase in population health around the world—enclosed by vast welfare improvements—the question of how this episode in itself has influenced the process of economic development is highly debated in the literature. Framed in a policy perspective, could improvements in health conditions in poor countries raise income? By studying the aggregate response to large health shocks across US states, this research seeks to contribute to this fundamental question.

This paper's main contribution is to provide estimates of the shocks to flu/pneumonia, tuberculosis, and maternal mortality, which began *circa* 1937, on subsequent aggregate economic development. I pay attention to these specific mortality causes on the following two grounds. First, they are expected to have different age profiles. Whereas flu/pneumonia mortality was concentrated on children, tuberculosis was mainly a burden on adults. Maternal mortality is, of course, related to death among women of child-bearing age. Therefore, paying attention to these particular causes of mortality possibly reveals how mortality of different ages is related to economic development. Second, since flu/pneumonia and tuberculosis are the two great killers that became treatable during this period, large reductions in them can expected to have macroeconomic implications.[1]

The paper estimates the magnitude of the effect of the health shocks on economic development exploiting two sources of variation. The first is time variation arising from medical breakthroughs around the period 1937-1950. For the considered mortality causes, the most notable drug innovations were sulfa drugs and antibiotics (see, e.g., Acemoglu and Johnson, 2007; Jayachandran et al., 2010).[2] The second is cross-state variation in pre-intervention mortality, which is intended to capture intensity of treatment. The constructed health-shock variables are the interaction between these two sources of variation, and only these shock variables are treated as exogenous in the regressions.

[1] In contrats to devleoping countries of that time, and due to prior eradication effort, malaria was *not* a big killer in the 1930's US (see, e.g., Bleakley, 2007).

[2] During this period, flu/pneumonia mortality fell from 0.12 percent (0.12 death per 100 individuals) to 0.03 percent, tuberculosis mortality from 0.05 percent to 0.02, and maternal mortality decreased from 0.49 percent (0.49 deaths per 100 births) to 0.09 percent (calculations are based state means).

The baseline results suggest, on the one hand, that a one-standard-deviation decline in flu/pneumonia and maternal mortality are associated with a 13 and 8 percent long-run increase in income per capita, respectively. And, on the other hand, that a one-standard-deviation decrease in tuberculosis leads to a long-run fall in income per capita of 9 percent.

The paper hereafter examines *how* the health shocks are related to development. This analysis starts by confirming prior beliefs. The flu/pneumonia-mortality shock is mainly related to reduction in child mortality, whereas the shock to tuberculosis is associated with a significant drop in adult mortality. Next, it is shown that the positive effect of the flu/pneumonia shock on income per capita is primarily driven by a negative force on population size through altered fertility behavior. The negative effect of the tuberculosis shock on income seems to be explained by a positive response to population, as there are *no* offsetting effects in fertility. Finally, the maternal-mortality shock is *not* linked to measures of aggregate mortality or population size, even though a negative reaction of fertility is recovered. Further, there seems to be a robust positive relationship between the shock to maternal mortality and capital worker, which cannot be attributed to capital shallowing or deepening.

The rest of the paper is organized as follows. Section 2 discusses how the paper contributes to the literature. Section 3 describes the dataset. Section 4 uncovers the main findings and presents robustness test. Section 5 goes beyond the main results and studies the channels of influence. Section 6 concludes.

2 Related literature

The paper is in spirit and objective close related to the seminal work of Acemoglu and Johnson (2007), henceforth AJ. Using international country data, their empirical strategy exploits similar medical breakthroughs, together with variation in pre-existing cross-country mortality rates of 15 diseases (mortality causes), to show that life expectancy has a statistically insignificant effect on income per capita. For the purpose of generating sufficient explanatory variation, the authors pool these 15 diseases into a single shock variable.[3] The present analysis shows

[3]The authors demonstrate identical results with a shock variable (instrument) that only aggregates tuberculosis, pneumonia, and malaria. Nonetheless, following the approach outlined here, one obtains imprecise

that this is possibly not without innocence. In fact, I obtain results very close to AJ for the US states if the flu/pneumonia and tuberculosis-shocks are combined into a single-index shock variable. The interpretation hereof is at the heart of the conclusion in this paper: health should be understood as multifaceted and various health improvements may accordingly have disparate effects on economic development.[4] One cannot, however, infer any conclusions by separating each disease in the international dataset. This can be interconnected with the issue of measurement error, which is an independent concern in the AJ study. For example, as argued in Bleakley (2010), many developing countries did not have adequate vital-registration systems in the 1940s. The data used here, on the other hand, are based on a nationwide system of death registration. Measurement error is, therefore, less likely to be an issue for current analysis. Another advantage of studying health improvements among US states is that the states are confounded by identical formal institutions. This means that the health-shock variables cannot be capturing institutional changes at the federal level.

In various ways, previous papers have attempted to clarify why AJ's results are so contradictory to the prevailing view—in the cross-country literature—arguing that health and income per capita are positively related. Cervellati and Sunde (2011a; 2011b) argue that effect of life expectancy on income per capita is non-monotonic. Following the framework of AJ, their analysis reveals that the relationship between life expectancy and income per capita is negative and statistically insignificant before the onset of the demographic transition, but positive and significant after its onset. Inspired by Unified Growth Theory (Galor, 2011), the authors provide a compelling economic explanation for this. Yet, another explanation could simply be that measurement error is more likely be an issue for ex-ante demographic-transition countries. Other papers have put forward concerns about the empirical approach in AJ (e.g., Bloom et al., 2009; Aghion et al., 2010). For example, Aghion et al. (2010) argue that AJ's estimates may be biased downward, as the regressions do *not* include initial life expectancy.

To the best of my knowledge, the present paper is the first, in this debate, to deploy the empirical framework, as suggested by AJ, in an alternative aggregate analysis. As already

estimates on them separately (based on own calculations).

[4] On the other hand, if the interests lies in the total effect of several health improvements, this paper, to some extent, supports the main result in AJ.

indicated, this exercise suggests that measurement error might blur a new understanding of the results in AJ. That is, the health shocks influenced income per capita in different ways.

Comparing cross-state cohort differences in fertility, Albanesi and Olivetti (2010) demonstrate that the 1930s decline in maternal mortality caused an inverted U-shaped pattern in fertility—the US baby boom and baby bust. This paper argues that the maternal-mortality shock affected fertility negatively. Nevertheless, this is not contradictory evidence. Because they compare fertility for a cohort of women that was giving birth before the sulfa-drug era, to two subsequent cohorts of women that enjoyed the benefits of less risky births. The empirical strategy here compares ex-ante sulfa-drugs fertility to average fertility the following 30 years.[5]

A large macro-empirical literature examines the relationship between economic growth and aggregate measures of health (e.g., life expectancy). The general interpretation is that health is conductive for development. 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), and Murphy et al. (2008). Exploiting geographical cross-country variation, the research by Lorentzen et al. (2008) find infant and adult mortality to impair economic growth. It is difficult to know why the conclusion in the present paper is rather different. One explanation could be that the quasi-natural experiment of the mid-20th century America is *not* generalizable to the their settings. Another could be the fact that their conclusion is inferred without considering the effect of child mortality.[6]

Finally, this paper is also connected to a line of research in the micro-empirical literature that exploits comparable type of variation to identity the effect of health on various outcomes variables. Examples of such studies are: Bleakley (2007), Bleakley and Lange (2009), Jayachandran and Lleras-Muney (2009), Lucas (2010), and Bhalotra and Venkataramani (2012). For instance, the latter paper finds a positive long-run effect of the introduction of sulfa-drugs on productivity and well-being for individuals living in the US.

[5] Looking at the international evidence, Albanesi (2011) recovers a similar pattern.

[6] With respect to human capital, Hazan (2011) provides evidence suggesting that childhood health is the most important determinant for the acquisition of education.

3 Data

This section briefly describes the data used in the analysis. The main outcome variable is GDP per capita, which often will be referred to as income. In the channel-of-influence analysis, the following outcome variables are used: child mortality, adult mortality, population size, capital per worker, secondary schooling enrollment rate, and fertility.

For the construction of the health-shock variables, I use state mortality rates for flu/pneumonia, tuberculosis, and maternal mortality in 1937, which is at the onset of the period with substantial declines in the mortality causes. Following the approach in AJ, the post-treatment period is 1950 onwards, this assumption is tested in section 4.3. A health-shock variable is then the interaction between an indicator variable that turns on in 1950 onwards and the pre-intervention mortality rate for a specific cause.

Because of data availability, the main sample consists of 48 states—all states, but Alaska, District of Columbia, and Hawaii—observed over the period 1940-1980. Summary statistics by census region is provided in Table 1.[7]

Table 1 about here

4 Estimation strategy and results

This section presents the empirical strategy and the main results of the paper, which are estimates of the health shocks on log GDP per capita (log income). These results are reported using 10 and 40-year panel models for the observation period 1940 to 1980. Further, section 4.3 supports the general interpretation by flexible regression estimates, which tests when and if the shocks became related to income.

4.1 Estimation strategy

By comparing the change in income in the pre-shock period relative to post-shock period between US states, the estimation strategy has the flavor of a standard differences-in-differences

[7] Precise definitions and data sources of all variables are given in Table 1a in Appendix.

approach. However, the current strategy exploits cross-state variation in pre-shock mortality rates as a measure of the intensity of the health shocks (treatment). One crucial factor here is that different states had dissimilar incidences in the mortality causes—as seen from Table 1, this is indeed the case. Of course, another key factor for the validity of the strategy is the assumption that the medical breakthroughs were a function of factors that are external to each US state.

The model is formalized by the following reduced-form estimation equation:

$$\log y_{it} = \alpha + \sum_{j=1}^{3} \beta_j \left(mortality_{ji} \times I_t^{\text{post}} \right) + \mathbf{Z}'_{it}\pi + \delta_i + \tau_t + \varepsilon_{it}, \tag{1}$$

where y_{it} is GDP per capita (or income) for state i in period t, $mortality_{ji}$ is the pre-intervention mortality rate of cause j in state i. The mortality causes are: maternal mortality, flu/pneumonia, and tuberculosis.[8] I_t^{post} is an indicator that equals one in 1950 onwards (pre-shock period). These main explanatory variables are loosely referred to as the health-shock variables. $\mathbf{Z}_{it}$ is set of time varying conditioning variables, δ_i and τ_t are state and time fixed effects, and ε_{it} is the disturbance term. Notice, $\hat{\beta}_j > 0$ corresponds to a positive effect of the health shock on income.

Section 4.4 also reports estimates allowing for mean reversion in GDP per capita. This is done in two steps. Firstly, notwithstanding of the issue of Nickell bias, the lagged dependent variable is simply included as a conditioning variable. Secondly, the $\beta_j's$ are estimated by GMM estimation, as suggested by Arellano and Bond (1991).

4.2 Basic findings

Table 2 reports the results of regressing log income on each cause-specific mortality-shock measure separately and jointed together. All regressions include state and time fixed effects. Starting with the 10-year panel model: columns (1)-(3) show that while the shocks to maternal and flu/pneumonia-mortality are positively linked to income, the effect of the shock to tuberculosis is unrelated. Next, because the shocks are likely to be interrelated, column (4) includes

[8] As mentioned, pre-invention mortality rates are measured in 1937. Similar results are obtained if 1935-36 mortality rates are used (not reported).

them simultaneously. I find that the coefficient on the flu/pneumonia-mortality shock retain its significance and becomes larger in magnitude, whereas the coefficients on the tuberculosis shock changes from positive to negative and statistically significant. This possibly indicates that the small positive—but insignificant—effect in column (3) is driven a positive correlation to the other shocks. Further, the fact that these two health shocks have opposite signs suggests that pooling them together into one health-shock variable—as is the modelling technique in AJ—materializes in an statistical insignificant effect on income. Indeed, the effect of this composite health-shock variable on income is estimated to 0.45 with standard error 0.67 (not reported). Finally, the magnitude of coefficient on maternal mortality reduces somewhat, but continues to be positive and statistically significant.

Table 2 about here

The latter four columns of Table 2 show that the 40-year-panel model delivers similar results. However, consistent with a long-run interpretation, the estimates are generally larger in magnitude. This type of specification also permits a graphical assessment of the findings. Specifically, the conditional effects of the health shocks, provided in column (8), are depicted as partial correlation plots in Figures 1 to 3, respectively. For one thing, they illustrate that the basic relations are not due to outliers.[9]

Figures 1-3 about here

Before proceeding further, the magnitude of the shocks should be evaluated. Based on the estimates in column (4) and the mean values of the pre-shock mortality rates, a reduction of 50 percent in maternal mortality leads to 14 percent increase in the level of income. The corresponding number for flu/pneumonia mortality is 30 percent, and a fall of 50 percent in tuberculosis mortality is related to a decline in income by roughly 8 percent. For comparison, the pre-shock cause-specific mortality rates in the US are comparable to mortality rates in contemporary developing countries as Bolivia, Cambodia, or Uganda.

[9] In Figures 2 and 3, Nevada could be argued to be an outlier. Yet, excluding this state, similar results are obtained.

4.3 Timing

Based on the work of AJ and Jayachandran et al. (2010), the framework in the previous subsection assumes 1950 onwards to be the post-shock period. This is now formally tested by a generalized difference-in-differences equation that takes the following form:

$$\log y_{it} = \alpha + \sum_{k=1940}^{1980} \sum_{j=1}^{3} \beta_{jk} \left(mortality_{ji} \times \lambda_t^k \right) + \mathbf{Z}_{it}'\pi + \delta_i + \tau_t + \varepsilon_{it}, \tag{2}$$

where the summation term represents the interaction between the pre-shock cause-specific mortality rates and time dummies, otherwise are the variables defined as in equation (1). The observation period is 1930 to 1980. The estimated $\beta's$ therefore give the time varying effect of the health shock on income relative to the baseline period 1930.

Table 3 presents the estimates of the $\beta's$ from regressing (2). Columns (1)-(3) report the results when the shocks are entered separately and column (4) considers them together. The following insights are made. Firstly, a general discontinuity in the pattern over time is observed around 1950. For instance, considering the flu/pneumonia shock, the estimate of β changes from negative to significantly positive in 1950 (see column 4). This finding supports the use of 1950 as cutoff date. Secondly, the correlations between the cause-specific mortality rates and income persistently increase in magnitude from 1950. Although, extending the observation period until 2000 reveals a peak in the magnitude of correlations around 1980 (not reported). Finally, it is also worth pointing out that similar conclusions are reached if dynamic effects are included in the regressions.

Table 3 about here

4.4 Extensions

The results thus far show that i) the shock to maternal mortality has a positive correlation with income ii) the flu/pneumonia-mortality shock is associated with a rise in income and iii) the tuberculosis shock is related to a decline in income. The current subsection studies extensions to the basic model specification, which provided these findings.[10]

[10] The subsection only presents estimate from the 10-year panel model. Still, comparable estimates are obtained with the 40-year panel model (available upon request).

The first three columns of Table 4 consider whether regional convergence accounts for the results. The empirical strategy utilizes that some regions (states) were plagued by higher pre-shock mortality rates than other. But if high-mortality regions had differential trends of income prior to the health shocks, as documented in Baier et al. (2007), the health-shock variables may simply be capturing regional income convergence. I start by taking this concern into account in two ways. First, column (1) includes census regional-specific time trends. The estimate on the maternal-mortality variable cut in half and loses its significance, though it still remains positive. The coefficient on flu/pneumonia mortality also reduces somewhat in magnitude, but there continues to be a positive and statistically significant relationship to income. The negative effect of the tuberculosis shock on income reduces, but retains its statistically significance. Second, columns (2) and (3) demonstrate that similar conclusions are acquired when estimating dynamic models to control for convergence in the outcome (i.e., with lagged dependent variables). In summary, only the positive effect of the maternal-mortality shock on income is non-robust to income convergence.

While columns (1)-(3) just demonstrated that the coefficients on flu/pneumonia and tuberculosis-mortality shocks are unlikely to be driven by preexisting trends, column (4) presents evidence from regressing log GDP per capita 1900-1940 on the health-shock variables 1940-1980. All three coefficients are highly insignificant and the point estimates on the flu/pneumonia and tuberculosis-shocks are considerably different in magnitude compared with the baseline specification. Consistent with the hypothesis, this falsification exercise shows that the health shocks have no explanatory power over income in the pre-shock period.

The latter two columns of Table 4 show the coefficients from alternative sample periods. As seen from columns (5) and (6), similar conclusions are reached when, for example, considering the 1930-1970 period or the 1930-2000 period.

Finally, it can be noted that the present robustness exercise refrains from including a range of control variables. Instead, the results should be interpreted in reduced-form sense. The subsequent section, however, moves beyond the basic reduced-form findings to ask the questions of why and how the health shocks are correlated with development.

Table 4 about here

5 Channels of influence

What are the channels through which the reductions in maternal mortality, flu/pneumonia, and tuberculosis possibly affected US economic growth? This section examines how the shocks relate to aggregate mortality, population size, physical and human capital, and fertility. The common denominator of these channels is that the literature—at one time or another—has hypothesized health to be an important factor in them.[11]

5.1 GDP per capita, child and adult mortality

This subsection studies whether the health shocks are related to aggregate measures of mortality. Supported by previous research, there a priori reason to believe that the age profiles from the three mortality causes are different. The evidence suggests that flu/pneumonia mortality was concentrated on children, whereas tuberculosis was a burden on adult individuals (see, e.g., Acemoglu and Johnson, 2006).[12] If the empirical analysis here retrieves similar conclusions, this could paint a picture of childhood health as being the more important determinant of economic development.

Table 5 presents the results. Column (1) displays the correlates with child mortality. As expected, the coefficient on flu/pneumonia mortality is negative and significantly larger in magnitude compared to the tuberculosis-shock variable. This column also shows that maternal mortality is *not* related to child mortality. In column (2), adult mortality is the outcome variable. While the tuberculosis shock has the expected negative correlation, the reduction in flu/pneumonia mortality has a positive relationship with adult mortality, which is somewhat unexpected. Even so, the evidence in columns (1) and (2) are consistent with the prior beliefs that the reduction in flu/pneumonia (tuberculosis) mortality had a relatively large effect on childhood (adult) health. The maternal mortality-shock is also unrelated to adult mortality.

The remaining columns of Table 5 revisit the effect on income with the aggregate mortality variables as additional explanatory variables. Column (3) adds adult mortality to the baseline

[11] See, e.g., Cervellati and Sunde (2005), Soares (2005), Acemoglu and Johnson (2007), Lorentzen et al. (2008), Ashraf et al. (2008), de la Croix and Licandro (2012), Herzer et al. (2012).

[12] Per definition, maternal mortality is concentrated on women of child-bearing age

specification. This completely eliminates the effect of the tuberculosis shock on income, whereas the coefficients on the other health-shock variables are barely influenced. Moreover, adult mortality is significant positively associated with income. Next, augmenting the model with child mortality further removes the positive effect of the flu/pneumonia shock on income, but the correlation between maternal mortality and income still remains unaffected (column 4). The estimate of child mortality on income is negative and significant. As a final exercise, column (5) reports 2SLS estimates, instrumenting child and adult-mortality with the flu/pneumonia and tuberculosis-shocks. Taken at face value, the estimates imply that a one-standard-deviation decline in child (adult) mortality is associated with 27 (24) percent increases (decrease) in income per capita, pointing to childhood health having a stimulating effect on income, while adult health tends to depress it.

Table 5 about here

5.2 Population size

Because a reduction in mortality mechanically brings about an increase in population—which tends to depress income—but, on the other hand, may have a long-run offsetting effect through fertility behavior, this specific channel has been central in the discussion of how health improvements influence economic development. Column (1) of Table 6 reports the average effects of the health shocks on population size. Consistent with the results thus far, population size is significantly negatively associated with the flu/pneumonia shock and significantly positively related to improvements in tuberculosis mortality. Therefore, one plausible channel through which the flu/pneumonia mortality-shock could have influenced income positively is the population channel. The opposite conclusion can, of course, be made for the tuberculosis health-shock. Again, I find no relationship between the shock to maternal mortality and population size. Column (5) demonstrates that the long-run estimates are larger in magnitude, which is a consistent pattern for all the channel estimates presented in Table 6.

Finally, it bears mentioning that the composite health-shock variable—comprising flu/pneumonia and tuberculosis-mortality—has a positive and statistically significant effect on population size (not reported). This actually means that the conclusions derived from this composite measure

are identical to those of AJ; an statistically insignificant effect on GDP per capita, but a positive effect on population size. Nevertheless, in contrast to AJ, I recover a positive effect on total GDP that is significantly different from zero, as one would expect given the positive estimate for population size.[13]

Table 6 about here

5.3 Physical and human capital

The shocks to maternal and flu/pneumonia-mortality are associated with higher levels of physical capital per worker (column 2 of Table 6). One forthright explanation is capital deepening of a shrinking population. Conditioning on log population reveals that while this is, indeed, the case for the flu/pneumonia shock, the estimate on maternal mortality is robust to this, which points to a positive correlation between the shock to maternal mortality and saving rates. The tuberculosis-shock variable bears a negative relationship with capital per worker, which seems to be completely explained by capital shallowing of a growing population.[14]

Column (3) of Table 6 shows that only the shock to flu/pneumonia-mortality is significantly correlated with secondary schooling enrollment rates. In particular, the coefficient estimate is positive and has a p-value of 7 percent. This coefficient implies that a 50 percent reduction in flu/pneumonia mortality is related to 11 percentage point increase in secondary schooling enrollment.

As a final point, it is seen that coefficients are larger in magnitude when considering the corresponding long-run models (column 5-8).

5.4 Fertility

Lastly, I look at how the health shocks are related to fertility. As suggested, the population channel appears to be central in the discussion of how aggregate health improvements relate

[13] As argued in Bleakley (2006) "...their estimates for GDP are insigificantly different from zero. This is surprising: apparently these countries cannot do anything with that extra population."

[14] These conditional results are not reported in the paper, but available upon request.

to economic development. Because the fertility-mortality correlation can be regarded as an important sub-component in this, it is of natural interest to study this specific relationship.

According to the estimated coefficients on the health shock-variables in column (4) of Table 6, a one-standard-deviation decrease in maternal mortality is associated with a fall of 0.13 children ever born to a woman age 35-44, while a shock of corresponding magnitude to flu/pneumonia mortality is related to decrease of 0.21 children. In contrast, the tuberculosis shock is completely unrelated to fertility. Importantly, these outcomes are consistent with the aforementioned population channel estimates.

6 Concluding remarks

This paper starts out by asking the question of whether adverse health conditions impair development in contemporary poor countries. Utilizing important medical inventions in 20th century America, as a quasi-natural experiment, the empirical analysis indicates that some health improvements are conductive for economic development, whereas others are *not*. On the one hand, the decline in flu/pneumonia mortality is associated with a rise in income per capita, with a fall in fertility, and with a decline in population size. On the other hand—through the population channel—the tuberculosis-mortality shock is negatively related to economic development. Further, the decline in maternal mortality is associated with a rise in income per capita, which is not statistically robust. The analysis, therefore, provides new insights on the argument that different health improvements affect behavior differently and thereby economic development differently.

Finally, coming back to the introductory question, improving health certainly increases welfare in term of longer and healthier lives, however, we cannot expect all such improvements to spur economic progress in them self.

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Table 1—Summary statistics by census region

		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
			period 1937					period 1940-1980			
Census region	# of states	Maternal mortality	Flu/pneumonia	Tuberculosis	Log GDP per cap.	Log population	Child mortality	Adult mortality	Log capital per worker	Second. enrollment	Fertility
NE	6	0.462	0.125	0.041	9.391	13.98	0.885	7.291	10.82	0.862	2.460
		(0.143)	(0.024)	(0.008)	(0.349)	(0.873)	(0.444)	(1.689)	(0.344)	(0.095)	(0.399)
MA	3	0.420	0.098	0.051	9.630	16.13	0.819	7.485	10.18	0.897	2.221
		(0.053)	(0.098)	(0.003)	(0.308)	(0.462)	(0.398)	(2.152)	(0.363)	(0.063)	(0.332)
SA	8	0.573	0.127	0.059	9.413	14.75	0.985	8.275	10.99	0.755	2.514
		(0.143)	(0.016)	(0.009)	(0.518)	(0.812)	(0.477)	(1.952)	(0.645)	(0.148)	(0.320)
ESC	4	0.605	0.146	0.072	9.163	14.92	1.115	8.523	10.67	0.685	2.734
		(0.100)	(0.002)	(0.012)	(0.626)	(0.214)	(0.615)	(1.716)	(0.697)	(0.168)	(0.219)
WSC	4	0.623	0.139	0.061	9.388	15.06	1.161	8.117	10.06	0.775	2.718
		(0.093)	(0.014)	(0.008)	(0.635)	(0.648)	(0.676)	(1.659)	(0.711)	(0.170)	(0.303)
MTN	8	0.504	0.134	0.074	9.524	13.48	1.243	9.061	11.06	0.858	2.808
		(0.186)	(0.038)	(0.067)	(0.483)	(0.706)	(0.683)	(2.663)	(0.480)	(0.113)	(0.429)
PAC	3	0.423	0.098	0.049	9.659	15.22	0.979	7.989	11.36	0.907	2.354
		(0.032)	(0.004)	(0.016)	(0.369)	(0.996)	(0.482)	(2.036)	(0.402)	(0.062)	(0.432)
WNC	7	0.426	0.107	0.032	9.361	14.37	0.943	6.881	11.29	0.870	2.745
		(0.063)	(0.018)	(0.013)	(0.470)	(0.721)	(0.438)	(1.327)	(0.457)	(0.085)	(0.426)
ENC	5	0.384	0.101	0.045	9.568	15.68	0.937	7.597	10.94	0.871	2.527
		(0.045)	(0.015)	(0.007)	(0.356)	(0.418)	(0.468)	(1.817)	(0.379)	(0.065)	(0.426)
US	48	0.494	0.120	0.054	9.443	14.64	1.019	7.949	10.90	0.829	2.598
		(0.137)	(0.026)	(0.031)	(0.483)	(1.034)	(0.541)	(2.044)	(0.550)	(0.132)	(0.407)

Notes: Table reports means by US census regions (standard deviations in parenthesis). The cause-specific mortality variables, in columns (1)-(3), are measured in 1937. The remaining variables are measured over the period 1940-1980 (columns 4-10). Maternal mortality is deaths per 100 live births. Flu/pneumonia and tuberculosis are in percent (i.e., # deaths per 100 individuals). Child mortality age 1 to 15 is measured in percent. Adult mortality is age 15 to 50 is measured in percent. Second. enrollmet is the secondary schooling enrollment rate.

Table 2—Cause-specific mortality and GDP per capita: Main results

	Dependent variable is log GDP per capita							
	10-year panel model				40-year panel model			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
maternal mortality $\times$ I^{post}	0.899***			0.576**	1.165**			0.678**
	(0.324)			(0.256)	(0.444)			(0.329)
flu/pneumonia mortality $\times$ I^{post}		4.628***		5.153***		6.424***		7.870***
		(1.479)		(1.536)		(1.957)		(2.017)
tuberculosis mortality $\times$ I^{post}			0.814	-2.757***			0.865	-4.305***
			(0.878)	(0.982)			(1.057)	(1.306)
# of observations	240	240	240	240	96	96	96	96
# of states	48	48	48	48	48	48	48	48

Notes: The table reports OLS estimates. All regressions include state and time fixed effects. The observation period is from 1940-1980. Maternal mortality is deaths per 100 live births (measured in 1937), flu/pneumonia mortality is number of deaths per 100 individuals (measured in 1937), and tuberculosis mortality is number of deaths per 100 individuals (measured in 1937). I^{post} is an indicator that equals one in 1950 onwards. Standard errors are clustered at the state level.

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

Table 3—Cause-specific mortality and GDP per capita: Dynamics of the health shocks

	Dependent variable is log GDP per capita			
	10-year panel model			
	(1)	(2)	(3)	(4)
maternal mortality × 1940	0.0660			0.148
	(0.0861)			(0.103)
maternal mortality × 1950	0.460**			0.376**
	(0.188)			(0.186)
maternal mortality × 1960	0.946***			0.742**
	(0.287)			(0.283)
maternal mortality × 1970	1.223***			0.950**
	(0.373)			(0.376)
maternal mortality × 1980	1.231***			0.826**
	(0.428)			(0.350)
flu/pneumonia mortality × 1940		-0.352		-1.147**
		(0.346)		(0.560)
flu/pneumonia mortality × 1950		1.798*		1.968*
		(1.019)		(1.106)
flu/pneumonia mortality × 1960		3.998***		3.127*
		(1.454)		(1.672)
flu/pneumonia mortality × 1970		5.233***		4.209*
		(1.694)		(2.154)
flu/pneumonia mortality × 1980		6.072***		6.723***
		(1.988)		(2.080)
tuberculosis mortality × 1940			0.0776	0.485
			(0.273)	(0.356)
tuberculosis mortality × 1950			0.00555	-1.577*
			(0.694)	(0.815)
tuberculosis mortality × 1960			1.151	-1.567
			(1.139)	(1.180)
tuberculosis mortality × 1970			1.468	-2.123
			(1.216)	(1.288)
tuberculosis mortality × 1980			0.943	-3.820***
			(1.175)	(1.350)
# of observations	288	288	288	288
# of states	48	48	48	48

Notes: The table reports OLS estimates. All regressions include state and time fixed effects. The observation period is from 1930-1980. Mortality variables, as defined in Table 2, are interacted with each time period from 1940 to 1980 (comparison year is 1930). Standard errors are clustered at the state level. *** p<0.01, ** p<0.05, * p<0.1.

Table 4—Cause-specific mortality and GDP per capita: Extensions

	Dependent variable is log GDP per capita, 10-year panel model					
	Regional convergence	Lagged dependent variable		Falsification test	Alternative time periods	
		OLS	GMM (AB)		1930-1970	1930-2000
	(1)	(2)	(3)	(4)	(5)	(6)
maternal mortality $\times$ I^{post}	0.294	0.198	0.319*	0.642	0.615**	0.681**
	(0.237)	(0.150)	(0.163)	(0.440)	(0.249)	(0.312)
flu/pneumonia mortality $\times$ I^{post}	2.799**	3.764***	4.218***	-1.680	3.675**	4.981***
	(1.237)	(0.929)	(1.025)	(2.531)	(1.483)	(1.764)
tuberculosis mortality $\times$ I^{post}	-2.163***	-1.942***	-2.847***	-0.434	-1.998**	-3.054***
	(0.724)	(0.582)	(0.761)	(1.409)	(0.966)	(1.069)
Regional time trends	Y	N	N	N	N	N
Lagged log GDP per capita	N	Y	Y	N	N	N
# of observations	240	240	240	238	240	384
# of states	48	48	48	48	48	48

Notes: Columns (1), (2) and (4)-(6) report OLS estimates. Column (3) reports GMM estimates (Arellano–Bond, rhs-variables are treated as exogenous) All regressions include state and time fixed effects. If otherwise is not indicated in the column, the observation period is 1940-1980. Maternal mortality is deaths per 100 live births (1937), flu/pneumonia mortality is number of deaths per 100 individuals (1937), and tuberculosis mortality is number of deaths per 100 individuals (1937). I^{post} is an indicator that equals one in 1950 onwards. Regional time trend is the interaction between time fixed effects and the 8 census regions of the US. Column (4) regresses log GDP per capita 1900-1940 on the RHS variables measured in the period 1940-1980. Standard errors are clustered at the state level.

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

Table 5—Channels of influence: GDP per capita, child and adult mortality

	10-year panel model				
	Dependent variable is:				
	Prob. of dying age:				
	1-15 (child mort.)	15-50 (adult mort.)	Log GDP per capita		
	OLS		OLS		2SLS
	(1)	(2)	(3)	(4)	(5)
maternal mortality $\times$ I^{post}	0.145	-1.148	0.676***	0.770***	0.792***
	(0.180)	(1.055)	(0.205)	(0.185)	(0.178)
flu/pneumonia mortality $\times$ I^{post}	-6.436***	15.29**	3.823***	0.657	
	(1.201)	(6.399)	(1.277)	(1.193)	
tuberculosis mortality $\times$ I^{post}	-2.535**	-32.25***	0.0489	-0.0851	
	(1.215)	(3.150)	(1.267)	(1.001)	
probability of dying age 15-50 (adult mortality)			0.0870***	0.116***	0.125***
			(0.0280)	(0.0197)	(0.0269)
probability of dying age 1-15 (child mortality)				-0.423***	-0.504***
				(0.0737)	(0.105)
# of observations	240	240	240	240	240
# of states	48	48	48	48	48
First-stage F-statistics:					
adult mortality					79.48
child mortality					25.86

Notes: Columns (1)-(4) report OLS estimates and column (5) reports 2SLS estimates. All regressions include state and time fixed effects. The observation period is from 1940-1980. Maternal mortality is deaths per 100 live births (1937), flu/pneumonia mortality is number of deaths per 100 individuals (1937), and tuberculosis mortality is number of deaths per 100 individuals (1937). I^{post} is an indicator that equals one in 1950 onwards. Prob. of dying age 1-15 (15-50) is the probability of dying before the age of 15 (50) conditional of surviving to the age of 1 (15). In column (5), adult and child mortality are instrumented with flu/pneumonia mortality $\times$ I^{post} and tuberculosis mortality$\times$ I^{post}. Standard errors are clustered at the state level.

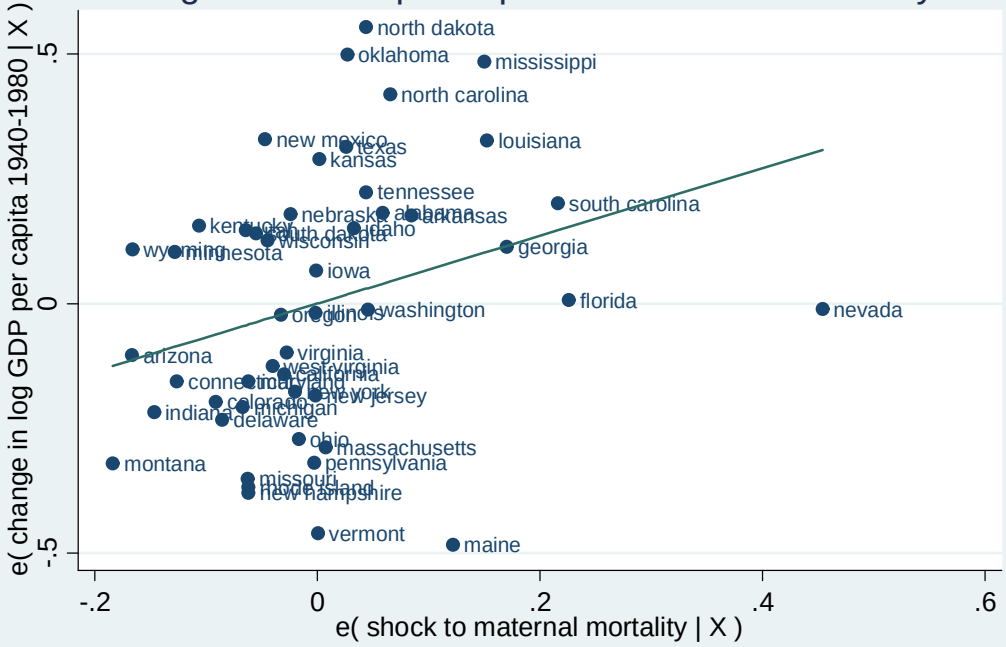
*** p<0.01, ** p<0.05, * p<0.1.

Table 6—Channels-of-influence analysis

	Dependent variable is:							
	Log population size	Log capital per worker	Second. school enrollment	Fertility	Log population size	Log capital per worker	Second. school enrollment	Fertility
	10-year panel model				40-year panel model			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
maternal mortality $\times$ I^{post}	0.469	1.102**	0.160	-0.935**	0.908	1.287*	0.268	-0.984*
	(0.357)	(0.510)	(0.161)	(0.406)	(0.603)	(0.652)	(0.207)	(0.513)
flu/pneumonia mortality $\times$ I^{post}	-6.593***	5.246*	1.842*	-6.118**	-8.758**	6.930*	2.143*	-8.557**
	(2.414)	(2.751)	(1.002)	(2.670)	(3.980)	(3.500)	(1.264)	(3.238)
tuberculosis mortality $\times$ I^{post}	7.396***	-2.512	-0.0737	1.221	10.35***	-4.192**	0.140	1.606
	(1.197)	(1.556)	(0.578)	(1.761)	(2.018)	(1.904)	(0.834)	(2.125)
# of observations	240	240	240	240	96	96	96	96
# of states	48	48	48	48	48	48	48	48

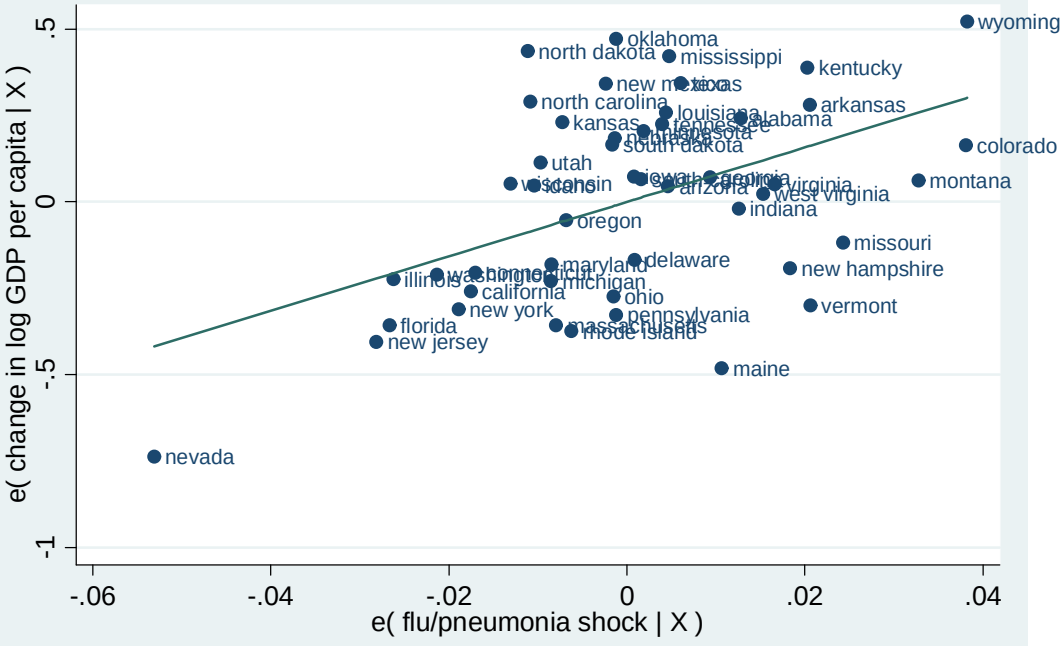
Notes: The table reports OLS estimates. All regressions include state and time fixed effects. The observation period is from 1940-1980. Maternal mortality is deaths per 100 live births (1937), flu/pneumonia mortality is number of deaths per 100 individuals (1937), and tuberculosis mortality is number of deaths per 100 individuals (1937) I^{post} is an indicator that equals one in 1950 onwards. The dependent variables are indicated in the columns (for precise variable definitions see Table 1a). Standard errors are clustered at the state level.
*** p<0.01, ** p<0.05, * p<0.1.

Figure 1: GDP per capita and maternal mortality



coef = .678, (robust) se = .33, t = 2.04

Figure 2: GDP per capita and flu & pneumonia mort.



coef = 7.869, (robust) se = 2.04, t = 3.86

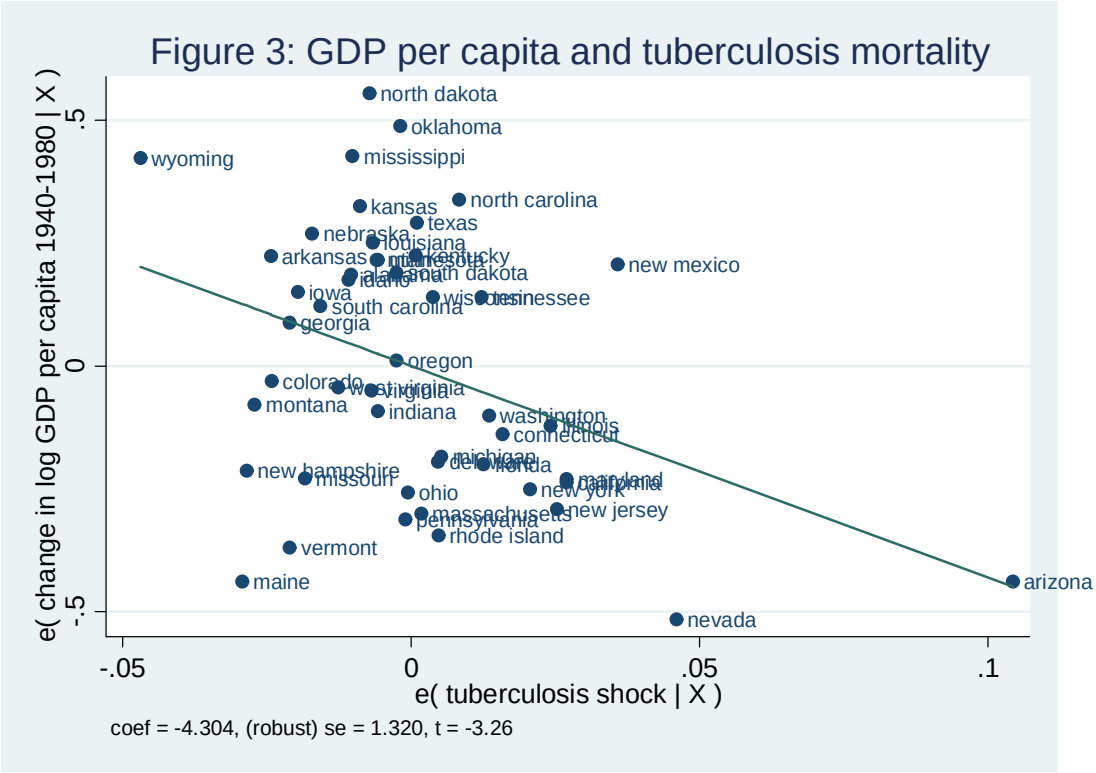


Figure 3: GDP per capita and tuberculosis mortality
e(change in log GDP per capita 1940-1980 | X)
-.5
0
.5
-.05
0
.05
.1
e(tuberculosis shock | X)
north dakota
oklahoma
wyoming
mississippi
kansas
north carolina
texas
nebraska
louisiana
arkansas
new mexico
south dakota
idaho
iowa
south carolina
georgia
oregon
colorado
montana
indiana
washington
connecticut
new hampshire
missouri
ohio
new york
new jersey
massachusetts
pennsylvania
rhode island
vermont
maine
arizona
nevada
coef = -4.304, (robust) se = 1.320, t = -3.26

Appendix:

Table A1: Data description

Variable:	**Description:**	**Source:**
GDP per capita	Gross domestic product per capita in real 2000 $	Turner et al. (2007)
Flu/pneumonia mortality	# of deaths of influenza and pneumonia per 100 individuals	Jayachandran et al. (2010)
Tuberculosis mortality	# of deaths tuberculosis per 100 individuals	Jayachandran et al. (2010)
Maternal mortality	# maternal deaths per 100 live births	Jayachandran et al. (2010)
Medical interventions dates	Used to code the health-shock variables	Acemoglu and Johnson (2007); Jayachandran et al. (2010)
Population	Population size in number of individuals	Turner et al. (2007)
Child mortality	Probability of dying between age 1 to 15 (in percent)	Murphy et al. (2008)
Adult mortality	Probability of dying between age 15 to 50 (in percent)	Murphy et al. (2008)
Capital per worker	Capital per worker in in real 2000 $	Turner et al. (2007)
Sec. school. enrollment rates	Secondary schooling enrollment rates	Turner et al. (2007)
Fertility	Number of children ever born to a woman age 35-44 in period t	Murphy et al. (2008)

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