The Diffusion of Health Technologies: Cultural and Biological Divergence

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Abstract

This paper proposes the hypothesis that genetic distance to the health frontier influences population health outcomes. Evidence from a world sample suggests that genetic distance–interpreted as long-term cultural and biological divergence–is an important factor in understanding health inequalities across countries. In particular, the paper documents a remarkably robust link between genetic distance and health as measured by life expectancy at birth and the adult survival rate. Also, the evidence reveals that the link has strengthened considerably over the 20th century which highlights the increasing effects of globalization on health conditions across countries through the transmission of health technologies.

Key Words: Population Health, International Diffusion of Health Technologies, Globalization, Cultural and Biological Divergence.

JEL: I12, I15, J10, N3, O11, O33

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1 Introduction

While inequalities in mortality outcomes across countries in the last century were reduced, considerable disparities persist even today.[1] For example, life expectancy at birth in Sweden at the start of the new millennium was 78 years whereas the corresponding figure in Malawi was only 51 years. What breeds this discrepancy in health across countries–the health gradient? The current paper takes the health gradient as a puzzle to be examined and seeks to contribute to a more profound understanding of the answer to this important and intriguing question.

In this paper, the focal point is on the diffusion of international health technologies in the 20th century. On this, Preston (1975, p.237) has concluded that "factor exogenous to a country's current level of income probably account for 75-90 per cent of the growth in life expectancy for the world as a whole between the 1930s and 1960s" where the spread of health technologies is thought of as exogenous–similar conclusions have been derived in other research (see Deaton, 2004; Cutler et al., 2006; Soares, 2007).[2]

This paper hypothesizes that a country genetically closer to the health frontier benefits more from new health technologies, compared to countries genetically further away, in their capability of diffusing these technologies and thereby driving down mortality. To test the hypothesis, I use a measure of genetic distance to the United States taken from Spolaore and Wacziarg (2009). This variable can be interpreted as an aggregate measure of cultural and biological long-term divergence to the US. Thus, the proposed hypothesis is based on the view that divergence–especially culturally divergence–interacts with modern health technologies in determining mortality outcomes. This observation is not new, for example, Caldwell (1990, p.51) writes that "where the greatest success over mortality have been gained, this achievement has been the product of an interaction between certain cultural and social characteristics on the one hand and easy accessibility of basic modern health services on the other" which, essentially, elaborates my hypothesis in a nutshell. A somewhat similar point is made in Deaton (2004, p.108): "today, the health of most people in the world, in rich as well as poor countries,

[1] See Becker et al. (2005) for a paper that documents convergences in life expectancy across countries.

[2] Table 6 in Appendix A, also reproduces the basic insight made in Preston (1975) for a wider group of countries, over the 1960-2000 period, by demonstrating that time fixed effects explain the bulk of variation in life expectancy at birth.

depends on their ability to locally adopt health knowledge and health technologies that have been discovered and developed and developed elsewhere". The current hypothesis builds on the presumption that this ability is in part captured by long-term divergence to elsewhere (the health frontier). Also, the fact that many health technologies (knowledge) are realizable even for poor countries today opens up a channel whereby long-term divergence may affect the health gradient around the income channel.

The novelty of the current paper is to utilize genetic distance, as proposed by Spolaore and Wacziarg (2009), to measure cultural divergence and to show that this variable is indeed a powerful and robust determinant of the health gradient at the country level. For example, the empirical analysis below demonstrates that a one-standard-deviation increase in genetic distance to the US is associated with a 55.6% of a standard deviation decrease in the adult survival rate, in the year 2000, controlling for a range of geographical, socioeconomic and historical characters . Moreover, the analysis demonstrates that there was no effect of genetic distance at the start of the 20th century. I take this as evidence for the proposed hypothesis because the globalization and efficacy of health and medical technologies were relatively limited at that period of time.

These findings contribute to the literature in two important ways. First, the findings identify the effect of technological progress on population health. Because of identification issues, such as reverse causality, this is a somewhat unexplored area (Bloom and Canning, 2007). However, my study utilizes a variable–genetic distance–where this is *not* a concern, to show that technological progress is indeed an essential determinant of the health gradient. Second, my findings also add to discussion of how countries health conditions are affected by globalization (Deaton, 2004). In fact, the empirical results provided here indirectly reveal that faster transmission of health technologies (globalization) has a significant positive effect on population health outcomes across countries.

This study relates to the research of Spolaore and Wacziarg (2009). Their focus is, however, on how genetic distance explains variation in output per capita.[3] In particular, they explain

[3]In an interesting contribution, Ashraf and Galor (2010b) study the relation between within country genetic diversity and historic economic outcomes, as well as contemporary outcomes. The analysis reveals a U-shaped relation which implies that one, in principle, can pinpoint an "optimal" level genetic diversity.

their finding of a negative effect of genetic distance on output per capita by the fact that long-term divergence acts as a barrier to the diffusion of all technologies. This research supports their finding but suggests that a central mechanisms through which genetic distance influences output negatively is the health channel as an intermediate.[4] Put more schematically, I argue that interaction between health technologies and cultural divergence $\Rightarrow$ health outcomes $\Rightarrow$ output per capita.

A complementarity hypothesis is proposed by Galor and Moav (2007). They argue persuasively that the timing of the transition from hunter-gather to agricultural society (the Neolithic Revolution) is pivotal for contemporary inequality in life expectancy across countries. They posit that the rise of agriculture launched the evolution of crowd infectious diseases through more dense populations. This, in turn, produced an evolutionary advantage for descendants of populations who made the agricultural transition early on. To support their hypothesis, they regress the timing of the Neolithic Revolution, adjusted with post-1500 migration flows, on life expectancy at birth in the year 2000 and they show that en earlier transition date is associated with higher life expectancy. The hypothesis put forward here underscores the importance of modern health technologies in symbiosis with long-term divergence. Crudely speaking, one can parallel my hypothesis to sophisticated geography hypothesis, where, because of technological drift, being genetically distant to the US has a contemporary adverse effect on health outcomes whereas the hypothesis put forward by Galor and Moav (2007) is more based on evolutionary biological line of thought.

The study by Papageorgiou et al. (2007) claim that non-health-frontier countries benefit from health knowledge embodied in medical imports in terms of lower mortality rates. Importantly, though, I demonstrate that the relation between health and genetic distance is robust to their argument which suggests that the influence of genetic distance on mortality outcomes is *not* per se operating through medical imports and, more generally, openness to trade.

Other papers have studied determinants of life expectancy or mortality on potentially exogenous factors. Among them, Pritchett and Summers (1996) exploit exogenous variation in income to determine the causal effect on various measures of health-status. They find a sig-

[4]Where the health channel is the strong cross-country correlation between output per capita and health (Preston, 1975; Bloom and Canning, 2000, 2007)

nificant effect of income in reducing infant and child mortality but they find no effect on life expectancy. These findings are also to some extent recovered in the present paper.

The remainder of the paper continues as follows. Section 2 elaborates on the hypothesis and presents a theoretical model to facilitate the empirical analysis. Section 3 briefly presents the empirical framework. Section 4 outlines the assembled dataset. Section 5 and 6 give the regressions results. Finally, section 7 concludes.

2 The hypothesis

This paper hypothesizes that genetic distance to the US, as a measure of long-term divergence, behaves as barrier for the diffusion of international health and medical technologies (knowledge) which is mirrored in population health outcomes.

There are several reasons to why this should be a reasonable hypothesis to test. Firstly, and essential for the hypothesis, is what Vallin and Meslé (2004) denote as the "health transition" which, broadly, refers to the international diffusion of new health technologies (shocks) where the speed and diffusion depend on country specific characteristics. In this regard, the authors themselves emphasize culture as one important characteristic. The hypothesis here simply says that this argument can, in part, be captured by cultural divergence to the health frontier.

Secondly, along similar lines, Caldwell (1980, 1990, 1992) argues that the interaction with culture divergence to Western countries and health technologies is a strong determinant of the mortality level in developing countries. For example, Caldwell (1992, p.213) concludes that "rapid mortality decline in the Third World depends on access to both modern curative and preventive medicine and the fullest possible collaboration with these systems in both belief and action" and genetic distance may be viewed as an excellent summary of divergence in such beliefs. In Caldwell (1990), he asserts that one persistent result, from various micro-studies, is that there are major ethnic or cultural discrepancies in mortality even after controlling for income and education. Caldwell (1980; 1992) also suggests that the strong correlation between female education and child mortality, found in many studies (see e.g., Cleland and van Ginneken, 1988), is because schooling produces a change in beliefs and behavior toward a so-called "Western-system" which he denotes as a deculturating experience.

Thirdly, besides the cultural channel, there may also biological angle to the hypothesis as well. While the topic is still debated, a branch of the biomedical literature has been arguing that there exist disparities in drug responsiveness and efficacy among different ethnic and racial groups within countries. For example, with respect to beta-blockers–which is used to treat heart related conditions–African Americans respond less well compared to European Americans (Tate and Goldstein, 2004). Similarly, Drake et al. (2008) claim that "there are well-documented disparities among ethnic and racial groups with respect to asthma prevalence, mortality and drug response". Since, genetic distance, inevitably, correlates with this type of ethnic and racial classification, a similar mechanism may be operating between countries. In other words, it is hypothesized that, on average, populations genetically distant to the medical (health) frontier may respond less well to new medicine because new medications are biased toward populations living in the proximity of the health frontier–represented here by the US.

One implication of the current hypothesis is that there should be no health gradient in genetic distance before the rise of modern health technologies. Even though an exact date for this "event" is hard to pinpoint, some authors have argued that the efficacy and diffusion of medicine in the start of the 20th century were weak–see, among others, McKeown (1972) and Caldwell (1992). Accordingly, I test for a correlation between genetic distance and life expectancy in 1900 and, as Section 6 shows, there seems to be no correlation at that period of time.

Finally, the choice of the US as health frontier should be motivated. First, this is the selection of Spolaore and Wacziarg (2009) as frontier for new technologies in general. Second, Kremer (2002) reports that the US pharmaceutical market accounts for 39.9 percent of the world market in 1998. Third, Papageorgiou et al. (2007, p.411) argue that the US, with nine other Western countries, "supply the bulk of medical products and carry out the vast majority of medical R&D". Notice, if a different country in that group was considered as frontier in the analysis below, e.g. UK, then similar results are obtained as this group of countries is genetically near the US (see Figure 1).[5]

The following section places the hypothesis in a theoretical context.

[5] This holds for all countries in the group except for Japan. The ten countries are: Belgium, France, Germany, Italy, Japan, Netherlands, Sweden, Switzerland, UK, and US.

2.1 Theoretical model

This section constructs a simple theoretical overlapping generations model in order to illustrate the hypothesis in a theoretical context. The proposed model draws on the ideas from the endogenous longevity literature (see Philipson and Becker, 1998; Chakraborty, 2004) which fits the purpose of supporting the empirical counterpart well

In this model, agents in country i live for two periods, denoted by the first and second period, respectively. All agents born at time t have a probability of $X_{it+1} \in]0;1)$ of surviving to the second period. The probability of survival, X_{it+1}, depends upon health investments, h_{it}, made in the first period, the diffusion of new health technologies $\Delta_h(1-\rho d_i)$, where $\Delta_h > 0$ denotes new health technologies discovered at the frontier, d_i is genetic distance to the frontier and ρ is a positive constant ensuring that $\rho d_i \in (0;1)$. Hence, in accordance with the proposed hypothesis, I assume that health inventions are realizable (and exogenous) to country i but it is the interaction with cultural/biological divergence to the frontier that determines the effectiveness in reducing mortality.

The survival probability also depends on the former generation's level of health, indicated by X_{it}. Summarizing these arguments gives the following relation:

$$X_{it+1} = e^{\Delta_h(1-\rho d_i)} h_{it}^{\eta} X_{it}^{\delta}, \tag{1}$$

where $\eta, \delta \in (0;1)$ and I have, additionally, assumed a particular functional relationship among the health inputs. Accordingly, it is assumed that health technologies complement private health investment–where private health investments, h_{it}, can be thought of in terms of basic nutrition (calorie intake) and care. That is, new health technologies make private health investments more productive in increasing survivability. Nevertheless, the efficacy of this interaction rest on genetic distance, d_i, to the frontier.

In the working period, agents supply one unit of labor endowment and earns a wage income of w_{it} which is divided between savings, s_{it}, for second period consumption, c_{it+1}, and private health investment, h_{it}. In the economy, there exists a perfect annuity market which distributes the savings of those who die prematurely toward members of the same generation. The periodic budget constraints therefore becomes:

$$h_{it} + s_{it} = w_{it}, \tag{2}$$

$$c_{it+1} = \frac{R_{it+1}}{X_{it+1}} s_{it}. \tag{3}$$

The gross real rate of interest, earned in the domestic capital market, is denoted by R_{it+1}. The representative agent from generation t generates expected utility from:

$$U_i^t = X_{it+1} \frac{c_{it+1}^{1-\sigma}}{1-\sigma}, \tag{4}$$

$0 < \sigma < 1$ is the coefficient of constant relative risk aversion.[6] The representative agent maximizes eq. (4) subject to eqs. (1)-(3) which produces the following closed form solutions:

$$h_{it} = \frac{\eta}{1-\sigma+\eta} w_{it}, \tag{5}$$

$$s_{it} = \frac{1-\sigma}{1-\sigma+\eta} w_{it}. \tag{6}$$

Now for the supply side of the economy, suppose that output per worker is described by the following function:

$$y_{it} = A_i k_{it}^{\alpha}, \tag{7}$$

where $\alpha \in (0;1)$ is the capital share, $k_{i,t}$ is capital per worker and A_i is determined by new technologies, also discovered at the frontier, and the ability to diffuse them:

$$A_i = e^{\Delta_y (1-\lambda d_i)}, \tag{8}$$

Δ_y is new technologies other than health technologies, $\lambda > 0$ ensures that $\lambda d_i \in (0;1)$. Notice, eq. (8) is along the lines developed in Spolaore and Wacziarg (2009).

[6] The assumption $0 < \sigma < 1$ implies that the flow utility is positive which ensures a meaning full solution for health investments. As an alternative, one could add a positive constant, ensuring that the flow utility will be positive, and only assume that $0 < \sigma$, as it is normally assumed. However, this implies that I can not obtain a closed solution. For more on this issue, in general, see e.g., Hall and Jones (2007).

Assuming that factors are paid by their marginal products and capital depreciates fully within one period yields the usual conditions:

$$w_{it} = A_i(1-\alpha)k_{it}^{\alpha}, \tag{9}$$

$$R_{it+1} = A_i\alpha k_{it}^{\alpha-1}. \tag{10}$$

The final element of the model is the capital market clearing condition $k_{it+1} = s_{it}$.[7]

Using eqs. (1)-(10), the subsequent expression for the survival rate can be obtained:

$$\ln X_{it+1} = -\left(\Delta_h\rho + \Delta_y\delta\eta\right)d_i + \eta\alpha\ln k_{it} + \delta\ln X_{it} + \Delta_h + \Delta_y + \ln\frac{\eta(1-\alpha)}{1-\sigma+\eta}. \tag{11}$$

This equation shows that genetic distance lowers the survival rate by means of two channels. The first channel is the interaction with new health technologies, which, as mentioned above, is empathized by many scholars to be important. The second channel operates through income, because genetic distance captures the ability to diffuse other technologies as well, it also influences the wealth of the economy and thereby health–wealthier is healthier in this simple model. But the hypothesis under investigation is captured only by the first channel. Thus, in estimating the effect of diffusing health technologies on health, a trade-off between omitted variable and reverse causality bias emerges. Indeed, by the inclusion of income as control, the second channel can be eliminated–reducing omitted variable bias–but this strategy rises the problem of reversed causality. Although, I admittedly have no perfect solution for this dilemma, I attempt to deal with this in two way. First, I estimate the effect without income but with some exogenous geographical controls know to be important determinants of income. Second, I include income but in order to minimize the risk of reverse causality, income is include with a time lag.

Since genetic distance (d) is fairly constant over a 100-year period, a time increasing effect of genetic distance on the survival rate (X) is evidence of that Δ_h increases over time which then signifies the development of new health technologies and/or globalization of health technologies.

In the start of the empirical analysis, I assume that $\delta = 0$ and estimate the level equation. Later on the growth approach is pursued.

[7] Thus, it is assumed that international capital flows are restricted and international health knowledge is not. This is only a modelling assumption which is not crucial for my theoretical results.

Finally, while there certainly are several other factors influencing mortality outcomes, eq. (11) is merely meant to clarify the proposed hypothesis. In fact, the empirical analysis below includes a range of other controls not given in eq. (11).

3 Estimating framework

The primary estimation framework can be derived from the theoretical model. The estimation equation therefore follows from eq. (11):

$$\ln X_{ijt} = \alpha + \beta d_i + \pi' Z_{it} + \mu_k + \upsilon_{ijt}, \tag{12}$$

X_{ijt} is a measure of health status in the ith country by three indicators, $j = 1, 2, 3$: life expectancy at birth, infant survival rate and adult survival rate in period t where the initial focus is on the year 2000.

The genetic distance from country i to the US is given by d_i. For future reference, the genetic distance between country 1 and 2 relative to the US is $D_{12} \equiv \mid d_1 - d_2 \mid$.

Z_i denotes a set of other controls (see below), μ_k's denote a full set of continent dummies and, finally, υ_{ijt} is the disturbance term. Again, the hypothesis under investigation is $\beta < 0$.

Because genetic, geographic and linguistic distance to the US are likely to be correlated and all potentially influence the outcome variables, $Z_i \forall t$ always includes physical distance to the US and a dummy equal to one if the main language is English.

4 The data

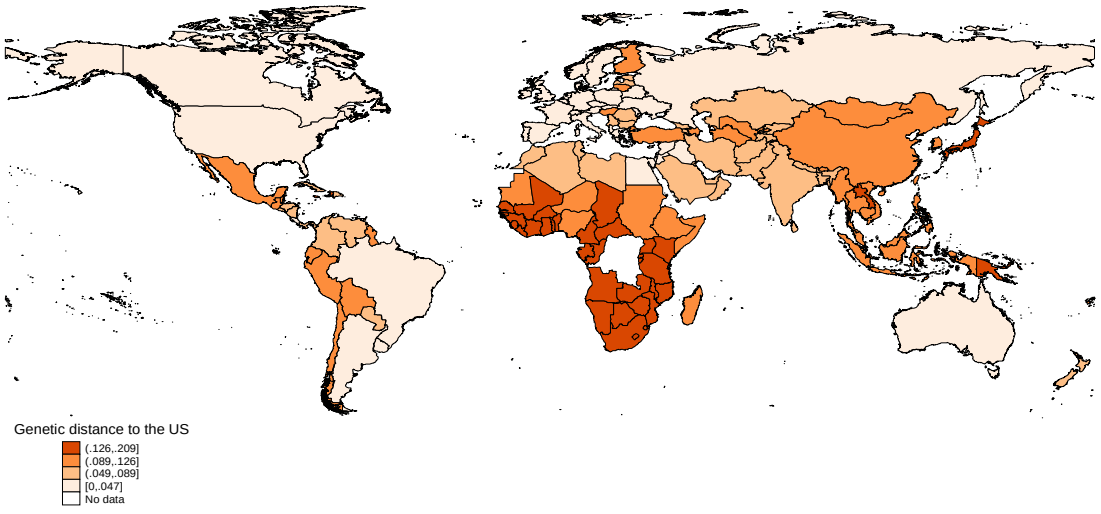
This section describes the dataset assembled to perform the empirical analysis.[8]

The main dependent variables I seek to explain are three mortality outcomes in the year 2000, as already indicated, these are: life expectancy at birth, infant and adult survival rates, in that order. The distinction is made because it reveals some interesting insights.

[8] Data sources and further details of all variables are given in the data Appendix and a cross correlation matrix for the most important variables is depicted in Table 7 Appendix A.

The key explanatory variable is the current genetic distance to the US (d). This variable is constructed on the basis of genetic distance between world populations from Cavalli-Sforza et al. (1994) and was matched to countries by Spolaore and Wacziarg (2009), using ethnic composition data, in the 1990s, from Alesina et al. (2003). Genetic distance can, in principle, be converted into time elapsed since the two populations shared a common ancestor population. One can, to some extent, compare genetic distance to a variable such as latitude. Geographic gradients in income or disease rates are well-known in the literature. However, it is obviously not the geographic location (e.g., latitude), per se, that is causally related to the gradients but rather a host of underlying variables like sunlight (Andersen et al., 2010), temperature, rainfall and so on. By the same token, genetic distance is based on comparison of neutral genes (think of eye-color). Nonetheless, the underlying variable, captured by genetic distance, is a measure of long-term divergence which I hypothesize to, especially, effect the ability to diffuse health technologies. Of course, opposed to latitude, genetic distance is influenced by human behavior in the very long run (migration). Nevertheless, in the short run the variable is reasonably exogenous to human-economic activities. A world map visualizing the genetic distance to the US is given in Figure 1.[9]

Figure 1: Countries and their genetic distance to the US



Data source: Spolaore and Wacziarg (2009)

[9]For a nice comprehensive description of the genetic distance variable see Spolaore and Wacziarg (2009).

Because the current ethnic composition may be endogenous to mortality–in the long run–I follow the approach by Spolaore and Wacziarg (2009) and utilizes the historic genetic distance, as of 1500 CE, to England as instrument for the current genetic distance to the US.

For exogenous controls, I use a range of geographically related variables, reflecting different aspects of geography. Additional controls include a range of other variables accounting for socioeconomic country characteristics and historical variables for early development. Overall, the control variables are introduced as the analysis progresses (all control variables are also described in the Data appendix).

5 Regression results

The first four columns of Table 1 report the estimates when the dependent variable is life expectancy in 2000. Column (1) shows that in absence of any controls,[10] there is a highly significant negative effect of genetic distance to the US. Taken at face value, the size of the coefficient implies that a one-standard-deviation increase in genetic distance to the US is associated with a decline in life expectancy of 13.6%–equal to a 76.7% of a standard deviation decrease in life expectancy. Column (2) includes continent fixed effect and the magnitude of the coefficient on genetic distance is reduced by around 39 percent which is to be expected. That is, the coefficient in the first specification is capturing that countries within a given continent are genetically more similar.[11]

To capture geographical factors simultaneous influence on genetic distance and life expectancy, column (3) includes exogenous geographical controls. First, share of land in tropics (*TROP*) is included due to the well-known gradient in disease rates (Bloom and Sachs, 1998) and since *TROP* is more prevalent in some geographical areas than others, it likely correlates with genetic distance to the US. Second, other aspects of geography may indirectly impact health through income, to circumvent this, column (3) also includes log mean distance to cost or river (*DISTCR)* and percentage of arable land (*ARAB*). Consistently, the inclusion of these geographical controls reduces the magnitude of genetic distance to the US on life expectancy a

[10] Besides the log distance to Washington D.C and a dummy equal to one if the main language is English.

[11] Continental fixed effects also soak up spatial correlation inflating the standard errors.

Table 1–Survivability and Genetic Distance to the United States

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	OLS	OLS	OLS	OLS	OLS	OLS	2SLS
Dep. variable:		*life expectancy*			*infant survival*	*adult survival*	
		$\ln X_{1,2000}$			$\ln X_{2,2000}$	$\ln X_{3,2000}$	
d	-2.587***	-1.577***	-1.087***	-0.988***	-0.0996*	-2.111***	-2.265***
	(0.172)	(0.276)	(0.241)	(0.227)	(0.0543)	(0.340)	(0.414)
TROP			-0.0879***	-0.0349	-0.00483	-0.0198	-0.0161
			(0.0227)	(0.0270)	(0.00630)	(0.0364)	(0.0336)
ARAB			-0.00194***	-0.000511	7.31e-05	-0.00156*	-0.00157**
			(0.000541)	(0.000607)	(0.000147)	(0.000815)	(0.000783)
DISTCR			-0.0364***	-0.0264***	-0.00307*	-0.0460***	-0.0450***
			(0.00820)	(0.00713)	(0.00169)	(0.0133)	(0.0127)
GDPPC				0.0576***	0.0191***	0.0202	0.0197
				(0.0153)	(0.00345)	(0.0209)	(0.0204)
Constant	4.599***	4.429***	4.903***	4.102***	6.671***	7.410***	7.407***
	(0.142)	(0.192)	(0.232)	(0.245)	(0.0643)	(0.490)	(0.522)
Observations	147	147	142	128	128	128	126
R^2	0.610	0.709	0.788	0.845	0.804	0.717	0.719
Standardized β on d	-0.767	-0.467	-0.324	-0.288	-0.128	-0.556	-0.597
Cont. fixed effects	NO	YES	YES	YES	YES	YES	YES

Notes: All regression includes log distance to Washington DC and a dummy equal to one if the main language spoken is English
Robust standard errors in parentheses.*** p<0.01, ** p<0.05, * p<0.1

little but the negative relationship remains highly significant and is still large in magnitude.[12]

To isolate the effects of the proposed channel, I now include log income per capita ($GDPPC$). But in order to lower the risk of reverse causation, I use $GDPPC$ from 1990. Column (4) takes $GDPPC$ into account, the effect of genetic distance decrease only slightly in magnitude and income per capita has the expected positive significant effect on life expectancy.[13]

The results, thus far, suggest that there exists a sizeable negative effect of genetic distance to the US on life expectancy. In particular, a one-standard-deviation increase in genetic distance is associated with a 5.3% decline in life expectancy equivalent to 28.8% of a standard deviation decrease in life expectancy.

Pritchett and Summers (1996) find the cross country relationship between the infant survival rate and income level to be particularly strong whereas the relationship between life expectancy and income is not. Those observations hint that it might be interesting to study the effect of genetic distance on the infant and adult survival rates separately. In columns (5) and (6), the dependent variables are the infant and adult survival rate, respectively, otherwise the specifications are similar to that of column (4). Both specifications have the expected negative signs, implying that genetic distance to the US is associated with a negative effect on survivability. However, the magnitude on the infant survival rate is rather small and is only significant at the 10% level while the effect on adult survival is "large" in magnitude and highly significant (also compare the standardized beta coefficients on genetic distance reported in Table 1). For the adult survival rate, a one-standard-deviation increase in genetic distance is associated with a 55.6% of a standard deviation decrease in the adult survival rate. Figure 2 plots the partial correlation between the adult survival rate and genetic distance–the health gradient in genetic distance–and it shows that the result is not driven by a small number of unimportant countries or outliers.[14]

[12]Similar results are obtained if I, alternatively, include absolute differences to the U.S. for the geography variables (results available upon request).

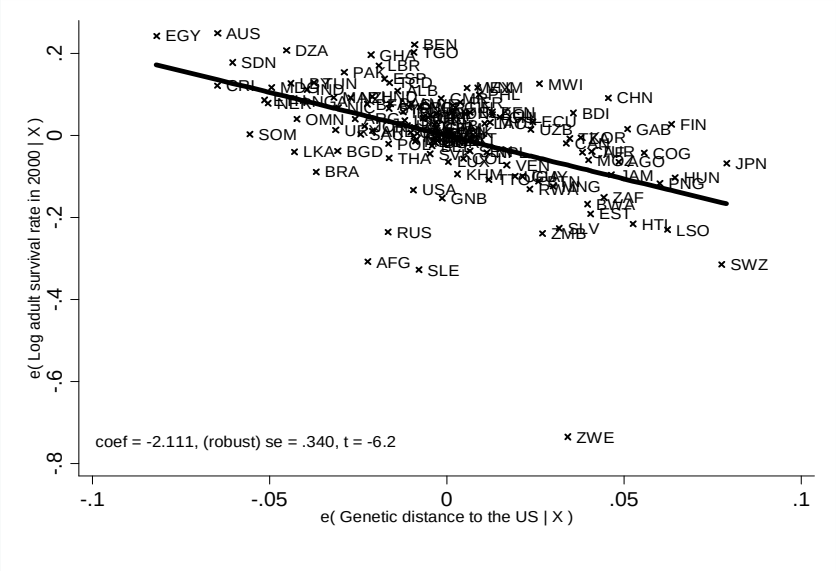
[13]I have also tried to include average year of schooling in the workforce, from Baier et al. (2006), as a measure for economic development. This does, however, not change any of the results. Irrespectively of the problems with reverse causation, I have also tried to included log income per capita in 2000 (instead of 1990), which increases the number of observations, again similar results are obtained.

[14]From Figure 2 one might infer that Zimbabwe (ZWE) is an outlier. However, dropping this observation

As a whole, the results imply that genetic distance to the US mostly influences life expectancy through the adult survival rate and *not* the infant survival which instead seems to be more sensitive to log income per capita (*GDPPC*). The fact that genetic distance has no significant impact on the infant survival rate is also in line with an argument put forward in Acemoglu and Johnson (2007, p.951). Indeed, they argue that their instrument for health (medical inventions) is not that strongly related to infant survival because the main medical discoveries in the 1940-200 period mainly affected adult survivability.

Last, I address the issue that the current ethnic composition of the US could be evidence of some omitted variable that also influences survival directly. Column (7) presents the two-stage-least square result for the adult survival rate where I use historic genetic distance in 1500 to England as instrument (*dHIST*). The estimate of the genetic distance variable remains statistically significant at the 1% level, and is larger than those obtained with OLS.[15]

Figure 2: Partial correlation plot



Data source: Column 6 of Table 1

Overall, the results in Table 1 point to an impact of genetic distance to the US on life

does not affect the result noticeable. See Figure 3 in Appendix A, for the corresponding partial plot without Zimbabwe.

[15]Which, as usual, suggests that measurement error in the ethnic composition, creating attenuation bias, is likely to be more important than omitted variables biases.

expectancy at birth which is primarily driven by its impact on adult survivability.

The rest of the paper is devoted to establish the robustness of this result.

6 Robustness check

Encouraged by the previous section, the specification most compatible with the proposed hypothesis–and most loyal to the theoretical model in section 2.1–is the one with the adult survival rate as the measure for health. For this reason, the robustness analysis revolves around this model.

In general, this section demonstrates a remarkably robustness of genetic distance on adult survivability over the period 1960-2000. Moreover, it reveals that genetic distance has no association with life expectancy in the year 1900 which supports the technological interpretation of the correlation between genetic distance and mortality outcomes.

Additional controls: The validity of my results, obtained so far, depends on the assumption that no omitted variable affects the adult survival rate and at the same time correlates with genetic distance to the US. For this reason, I now substantiate further the robustness of the result by including additional controls. Notice, because the last section established that the health gradient in genetic distance is not due to the income channel and because of reversed causation, the robustness analysis refrains form including $GDPPC$ in any of the following specifications.

In Table 2 additional geographical and historical controls are included. First, I check whether my particular choice of measure for geography influences the results. While proportion of land in the tropics ($TROP$) and absolute latitude ($ALAT$) are highly correlated, $ALAT$ may be more appropriate for the idea that technology normally diffuses more easily at same latitudes. Furthermore, whether countries are landlocked ($LOCK$) may be related to the ability to diffuse new health technologies, seeing that such countries, in general, have difficult access to the outside world (Soares, 2007). In column (1) and (2) these variables are included separately and in Column (3) all geographical variables, considered, are included together. My estimates of the effect of genetic distance on adult survivability remains negative a highly significant.[16]

[16] As for the geographical variables in the previous section, similar results for genetic distance are obtained if

Table 2–Robustness analysis I

	Geography			History–early development			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Dependent variable: *adult survival:* $\ln X_{3,2000}$						
d	-2.451***	-2.057***	-1.873***	-1.941***	-1.886***	-1.942***	-1.819***
	(0.357)	(0.272)	(0.272)	(0.294)	(0.307)	(0.328)	(0.315)
ALAT	-0.00170		-0.00267				
	(0.00119)		(0.00213)				
LOCK		-0.118***	-0.0814***				
		(0.0290)	(0.0307)				
TROP			-0.109*	-0.0482*	-0.0330	-0.0140	-0.0253
			(0.0629)	(0.0262)	(0.0277)	(0.0338)	(0.0277)
DISTCR			-0.0355***	-0.0449***	-0.0553***	-0.0454***	-0.0538***
			(0.0123)	(0.0112)	(0.0108)	(0.0122)	(0.0113)
ARAB			-0.00253***	-0.00304***	-0.00279***	-0.00222***	-0.00260***
			(0.000783)	(0.000697)	(0.000780)	(0.000729)	(0.000770)
LPD				0.0259***			
				(0.00734)			
STAT					0.0985***		
					(0.0374)		
FERT						-0.000326	
						(0.000750)	
NRW							0.0155*
							(0.00820)
Constant	7.368***	7.047***	7.780***	7.581***	7.794***	8.392***	7.668***
	(0.434)	(0.263)	(0.477)	(0.360)	(0.353)	(1.298)	(0.419)
Observations	142	147	142	140	136	118	142
R^2	0.607	0.645	0.703	0.700	0.687	0.717	0.682
Standardized β on *d*	-0.657	-0.546	-0.499	-0.521	-0.507	-0.506	-0.488

Notes: All regression include continental FE. log distance to Washington DC and a dummy equal to one if the main language spoken is English. All regressions estimated by OLS. Robust standard errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

Second, for the reason that genetic distance is a measure of time elapsed since two populations has been one, genetic similar countries are more likely to share the same economic history, an aspect which might directly impact adult survivability. Although the inclusion of income per capita, in the previous section, is intended to capture some of this matter, it might not suffice. For example, genetic similar countries may have made the transition to agriculture earlier than countries which are genetically distant. In previous studies, the timing of the Neolithic Revolution has been shown to be crucial for early economic development (Ashraf and Galor, 2010a). But an early Neolithic Revolution need not to be associated with higher per capita income today (Galor, 2011). Still, early development might influence contemporary health performance. For example, up to 25% of European American is, to some extent, protected against HIV infection and progression while this is not the case for other ethnic groups (Stephens et al., 1998). One may reason that this is due to the European American-population long-term history of living in more densely populated areas which, in essence, is the hypothesis put forward by, Galor and Moav (2007). However, genetic distance to the US might also pick this up because it measures ethnic and racial ancestry. Therefore, I now include controls for early development. As a measures for early development I use: log population density of year 1500 CE (LPD), an index for state history from 0 to 1500 CE ($STAT$), the onset (date) of the demographic/fertility transition ($FERT$) and the timing of the Neolithic revolution (NRW). As already mentioned, the latter variable is used in Galor and Moav (2007) to test their hypothesis. Column (4)-(7) expand upon these variables of early development but they only have a negligible effect on my estimate of genetic distance to the US.[17]

Previous studies have shown that ethnic and linguistic diversity, within a country, have an adverse effect on growth and redistribution (Easterly and Levine, 1997; Alesina et al., 1997 and Desmet et al., 2008) potentially influencing survivability through the provision of public health. These observations, together with the result in Ahelrup and Olsson (2009), that ethnic diversity is related to genetic distance, make it worthwhile to include a measure

I include the absolute difference to the US (result available upon request).

[17] Also notice, the correlation between the timing of the Neolithic Revolution and genetic distance to the US is rather high (-0.736, see Table 7). One interpretation of this correlation could be along the lines of Sokal et al. (1991). They argued that agriculture in Europe was diffused by means of population migration, explaining the correlation with the genetic makeup.

of ethnolinguistic fractionalization (*ELF*). Column (1) of Table 3 includes *ELF*, importantly, though, genetic distance is unaffected by this.

Besley and Kudamatsu (2006) point toward a link between health outcomes and democracy across countries. Specifically, the authors argue that democracies, in general, will be more concerned with public health issues. Undoubtedly, genetic distance to the US and the level of democracy is related. Column (2), therefore, includes a variable for the degree of democracy prevailing in the country in the year 1990 (*POLIT2*).[18] This does not change the coefficient on genetic distance and it confirms the results obtained in Besley and Kudamatsu (2006) that there is a positive relation between democracy and health. As an additional measure of provision of public health service, I include the share of population with access to safe water (*WATER*) in column (3). This variable has the expected positive sign but the magnitude of genetic distance remains unaffected.

Caldwell (1986) and Filmer and Pritchett (1999) find that religion is an important determinant of infant mortality. Therefore column (4) includes that share of Muslims in a country (*MUSL*) and the share of Catholics (*CATH*). Both variable have practically no impact on the adult survival rate and, again, the genetic distance variable is unaffected.

Papageorgiou et al. (2007) emphasize the importance of medical technology diffusion on health outcomes. Their study uses medical imports as a measure for the diffusion of medical technology. For 66 medical-importing countries, the authors show that diffusion is an important contributor to health performance as measured by cross country mortality rates. Column (7) of Table 3 recreates their basic insight by demonstrating that medical import (*MEDI*) has a significant positive effect on the adult survival rate. The regression in Column (8) reproduces my basic result for this smaller sub-sample: genetic distance still has a negative effect on the adult survival rate. Column (9) incorporates both variables and shows that the magnitude of the coefficient on *MEDI* is reduced substantial while the effect of genetic distance on adult survivability is barely affected. This comparison, once more, suggests that genetic distance is an important determinant of the adult survival rate.

Notice, I have also checked whether my results hinge on the inclusion of Sub-Saharan coun-

[18] As an alternative robustness check I have also tried to include an index for institutional quality (*SOCIN*), used in Hall and Jones (1999). Similar results are obtained.

Table 3–Robustness analysis II

	Institutions and Religion				Medical Imports		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Dependent variable: *adult survival:* $\ln X_{3,2000}$						
d	-2.100***	-2.138***	-2.103***	-2.240***		-2.152***	-2.011***
	(0.327)	(0.335)	(0.318)	(0.344)		(0.566)	(0.573)
ELF	0.0334						
	(0.0423)						
POLIT		0.00418*					
		(0.00225)					
WATER			0.00209*				
			(0.00116)				
MUSL				-2.45e-05			
				(0.000376)			
CATH				0.000690*			
				(0.000410)			
MEDI					0.0380**		0.0254*
					(0.0145)		(0.0138)
TROP	-0.0629**	-0.0299	-0.00203	-0.0347	-0.201***	-0.114***	-0.0936**
	(0.0308)	(0.0308)	(0.0343)	(0.0297)	(0.0407)	(0.0386)	(0.0429)
DISTCR	-0.0483***	-0.0420***	-0.0410***	-0.0449***	-0.0290*	-0.0237*	-0.0189
	(0.0118)	(0.0130)	(0.0118)	(0.0112)	(0.0156)	(0.0126)	(0.0127)
Constant	7.519***	7.467***	7.322***	7.590***	7.351***	7.323***	7.119***
	(0.345)	(0.353)	(0.506)	(0.502)	(0.467)	(0.346)	(0.356)
Observations	132	121	139	138	66	66	66
R^2	0.682	0.700	0.668	0.655	0.688	0.746	0.756
Standardized β on *d*	-0.563	-0.575	-0.565	-0.603		-0.496	-0.494

Notes: All regressions include continental FE, log distance to Washington DC and a dummy equal to 1 if the main language spoken is English. All regressions are estimated by OLS. Rob. std errors in parentheses *** p<0.01, ** p<0.05, * p<0.1

tries. Reassuringly, though, excluding those countries from the sample does not change my results significantly.

A growth approach: Up to this point, I have studied the effect of genetic distance on the level of the adult survival rate. As outlined, however, genetic distance might also influence the growth rate of the survival rate. Table 4 pursues the growth approach by incorporating the log of the adult survival rate in the year 1960 (lnX_{60}). The estimated coefficients are consistent with some conditional convergence, that is, a high initial survival rate subsequent reduces the growth rate in this variable. More interestingly for the current analysis, genetic distance has a significant negative impact on the growth of the adult survival rate in all specifications. For example, in column (3), one-standard-deviation increase in the genetic distance relative to the US is associated with 43.6% of a standard-deviation decrease in the adult survival rate, controlling for geographical, historical and economical characteristics.

Table 4–Robustness analysis III

	A Growth Approach	
	(1)	(2)
	Dependent variable: $\ln X_{3,2000}$	
d	-1.901***	-1.619***
	(0.385)	(0.366)
$\ln X_{3,1960}$	0.242**	0.180
	(0.104)	(0.115)
TROP		0.00662
		(0.0373)
DISTCR		-0.0516***
		(0.0118)
ARAB		-0.00250***
		(0.000837)
Constant	5.494***	6.367***
	(0.795)	(0.935)
Observations	133	128
R^2	0.608	0.687
Standardized β on d	-0.508	-0.436
Cont. fixed effects	YES	YES

Notes: All regressions are estimated by OLS. Robust standard errors in parentheses*** p<0.01, ** p<0.05, * p<0.1

Alternative years: Now, I investigate the time varying effect of genetic distance on the adult survival rate. Table 5 presents the results from this study where the same variation in explanatory variables is exploited by restricting the samples. The important lesson from column (1)-(5) is that the effect of being genetic distant from the US on the adult survival rate is increasing over time. As argued, this is possibly evidence of an acceleration of new medicine, new treatments and new health technologies and globalization which has made the health gradient, in genetic distance, more steep.

Because of lack of data, column (6) and (7) utilize life expectancy at birth as dependent variable, to compare the effect of genetic distance on health in start of the 20th century to the end of the century. In column (6), the effect of genetic distance to the US in the year 1900 has the wrong sign and is insignificant. Whereas in 2000, column (7), the effect of genetic distance has the correct hypothesized negative sign and is significant (using the same sample). Again, I view this as support for the proposed hypothesis because the diffusion of international medical knowledge is a precondition for genetic distance to influence mortality and this condition was, to wide extent, not meet in start of 20th century.

7 Concluding remarks

This paper put forward empirical evidence for the hypothesis of a cross-country health gradient in cultural and biological divergence to the technological health frontier. The idea behind this type of health gradient is that long-term divergence interacts with the diffusion of modern health technologies. The paper empirical documents that this health gradient is not primarily operating through geographical, historical and other social economic factors.

As whole, the results support the conclusions made in Cutler et al. (2006, p.117). They conclude that "...an acceleration in the production of new knowledge and new treatments is likely to make the health gradient steeper, with increasing gaps across educational and social class (occupational) groups, and possibly race as well. Gaps between countries may also widen". Indeed, the empirical evidence, presented here, suggests that the health gradient in cultural divergence has become more steep and that there was no gradient at all in start of the 20th century. I view this as indirect evidence for the increasing importance of the international

Table 5–Robustness analysis IV

	Alternative Dates						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Dep. var:	Adult survival rate					Life expectancy	
Year	1970	1980	1990	2000	2006	1900	2000
d	-0.757**	-0.808**	-0.923***	-2.077***	-2.278***	0.207	-0.992***
	(0.375)	(0.310)	(0.297)	(0.353)	(0.470)	(0.461)	(0.249)
TROP	-0.196***	-0.197***	-0.139***	-0.0490	0.000687	-0.0672	-0.115***
	(0.0428)	(0.0368)	(0.0357)	(0.0345)	(0.0582)	(0.0430)	(0.0234)
ARAB	-0.00130	-0.00179**	-0.00233***	-0.00270***	-0.00212**	-0.00289**	-0.00224***
	(0.000921)	(0.000722)	(0.000652)	(0.000844)	(0.000885)	(0.00129)	(0.000564)
DISTCR	-0.0246*	-0.0286**	-0.0286***	-0.0533***	-0.0422***	-0.0173	-0.0350***
	(0.0124)	(0.0110)	(0.0109)	(0.0119)	(0.0143)	(0.0149)	(0.00763)
Constant	6.700***	6.380***	6.689***	7.606***	7.530***	4.631***	4.854***
	(0.397)	(0.318)	(0.316)	(0.357)	(0.382)	(0.525)	(0.225)
Observations	121	121	121	121	121	125	125
R^2	0.746	0.744	0.705	0.641	0.602	0.802	0.844
Standardized β on d	-0.175	-0.211	-0.262	-0.561	-0.606	0.042	-0.296

Notes: All regressions include continental FE, log distance to Washington DC and a dummy equal to one if the main language spoken is English. All regressions are estimated by OLS. Robust standard errors in parentheses*** p<0.01, ** p<0.05, * p<0.1

diffusion health technologies–which one can interpret as globalization of health technologies–in determining cross-country health outcomes.

These findings add to debate of what determines health improvements at the national level. They provide evidence for that scientific breakthroughs matters to a great extent for adult survivability while income per capita seems to matter lesser extent.

Appendix A

Table 6–Life expectancy and income

	Dependent variable: Log life expectancy at birth		
	(1)	(2)	(3)
Log *GDPPC*	0.134***		0.002
	(0.0155)		(0.0133)
Obs.	694	694	694
R^2	0.264	0.678	0.678
Country FE	YES	YES	YES
Time FE	NO	YES	YES

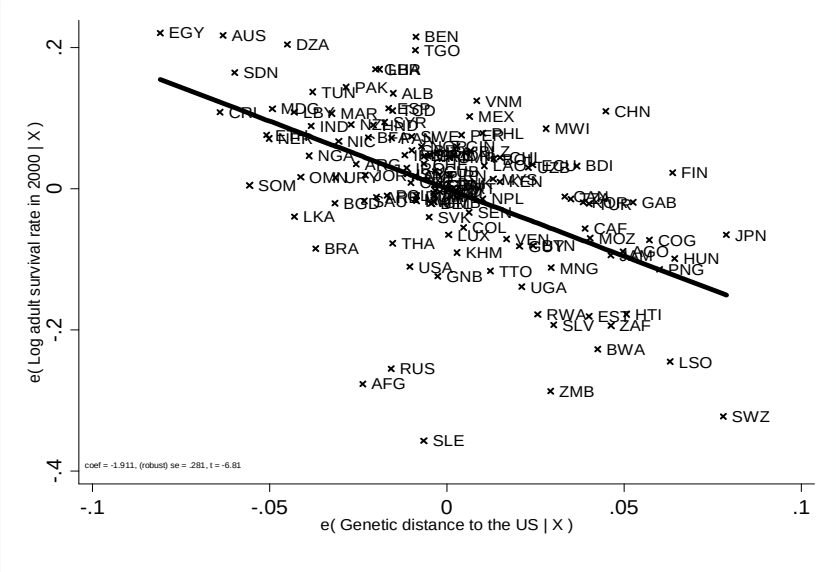
Notes: countries are the level of observation with decennial time span. The sample includes 193 countries and size of the constant is not reported. SD errors are clustered at the country level: *** p<0.01, ** p<0.05, * p<0.1

Table 7–Cross-correlations

Variables	d	X3	X1	X2	GDPPC	NRW
d	1.000					
X3	-0.754	1.000				
X1	-0.773	0.874	1.000			
X2	-0.676	0.700	0.940	1.000		
GDPPC	-0.613	0.594	0.785	0.820	1.000	
NRW	-0.734	0.614	0.630	0.541	0.434	1.000

Notes: X1, X2 and X3 are measured in 2000 and GDPPC in 1990

Figure 3: Partial correlation plot



Data source: Column 6 of Table 1 but without the observation Zimbabwe

Data appendix

Health:

$X_{3,1960-2000}$= The male adult survival rate. The probability of surviving to the age 60 conditioned on surviving to the age of 15 for the period 1960-2000. Source: World Bank's World Development Indicators.

$X_{2,1960-2000}$= The probability of an infant surviving to the age of one for the period 1960-2000. Source: World Bank's World Development Indicators.

$X_{1,1960-2000}$ = Expected length of life at birth for the period 1960-2000. Source: World Bank's World Development Indicators. Life expectancy in the year 1900 is taken from Acemoglu and Johnson (2007).

Genetic:

d=Current genetic distance to the United States which may be interpreted as the time since two populations have shared common ancestors. A higher d is associated with a larger difference in genetic distribution. For a detailed description see Spolaore and Wacziarg (2009). Source: Spolaore and Wacziarg (2009).

$dHIST$= Genetic distance to England as of 1500. Source: Spolaore and Wacziarg (2009).

Geography:

ALAT = Absolute average latitude from Equator. Source: CIA World Factbook.

ARAB = Percentage of arable land. Source: World Bank's World development indicators.

DISTCR = Nearest distance to coast line or river. Source: Gallup et al. (2001)

FROST = Proportion of land with more than five days of frost per year. Source: Master and McMillan (2001).

Geodesic distance=distance between the major cities of the countries (in measure of the great circle). Source: Centre d'Etudes Prospectives et d'Informations Internationales (CEPII).

TROP = Percentage of tropical land area. Source: Gallup et al. (2001)

LOCK = A dummy which takes on the values one if the country is landlocked and otherwise zero. Source: Gallup et al. (2001)

Early development:

NRW = Weighted average of the time elapsed since the ancestors of the population of each country in year 2000 went through the Neolithic Revolution in 1000 of years. For a more detailed description see Galor and Moav (2007). Source: Putterman (2008).

NRU = Unweighted time elapsed since Neolithic Revolution in 1000 of years. Source: Putterman (2008).

STAT = State Antiquity Index. The score reflects the existence of a government, the proportion of the territory covered, and whether it was indigenous or externally imposed. Source: Putterman (2008)

LPD = Log population densities in 1500 CE. Source: McEvedy and Jones (1978)

FERT = The year of the beginning of the fertility transition which is arguably related to the economic take off. Source Rehr (2004).

Socioeconomic:

GDPPC = log of real GDP per capita in constant prices in the year 1990. Source: Penn World Tables version 6.3.

SOCIN = An index taking on the value 0 to 1 on the social infrastructure in a given country. Source: Hall and Jones (1998).

POLIT2= a variable in the range -10-10 where a positive value indicated a democracy. Source: The Polity IV Data Base

ELF= ethnolinguistic fractionalization index. Source: Fearon (2003)

WATER= Access to an improved water source refers to the percentage of the population with reasonable access to an adequate amount of water from an improved source, such as a household connection, public standpipe, borehole, protected well or spring, and rainwater collection. Source: World Bank's World Development Indicators.

MEDI= Medical imports is the sum of pharmaceutical, medical, and other health-related imports. Source: Papageorgiou et al. (2007)

HIV= Prevalence of HIV refers to the percentage of people ages 15-49 who are infected with HIV. Source: World Bank's World Development Indicators

MUSL= Share of Muslims in a given country in 1980. Source: Acemoglu et al. (2001)

CATH= Share of Catholics in a given country in 1980. Source: Acemoglu et al. (2001)

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