Eye Disease and Development*

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Abstract: Using cross-country data, this study examines the impact of cataract on economic development. We hypothesize that an earlier onset of vision loss reduces the return to human capital investments, which delays the onset of the demographic transition and lowers long-run labor productivity. Empirically, there is a strong negative correlation between cataract prevalence and labor productivity. However, since cataract can be cured surgically given adequate resources, prevalence rates are unlikely to be exogenous. We therefore develop an epidemiologically founded identification strategy: the age of onset of cataract is strongly affected by exposure to solar ultraviolet B radiation. Our IV estimates supports a detrimental impact of cataract on aggregate labor productivity. The size of the impact can plausibly be accounted for by a differential timing of the demographic transition.

Keywords: Comparative development, eye disease, cataract, climate

JEL Codes: O11; I00; Q54

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1 Introduction

Over the last few years there has been a lively debate on the impact of health and longevity on long run economic development. Some research suggests that health improvements might dramatically accelerate growth (e.g., Gallup and Sachs, 2001), whereas other studies raise doubts as to whether an elevated health status in the population will have a growth enhancing effect at all (e.g., Acemoglu and Johnson, 2007).

The present study contributes to this debate by examining the link between eye disease and aggregate labor productivity. We hypothesize that the incidence of eye disease has had an important impact on the historical growth record. In particular, we argue that by inducing an inherently lower return to human capital investments, a higher incidence of eye disease has served to delay the timing of the demographic transition and thereby the emergence of persistent economic growth, which was first observed in Western Europe during the 18th century. By contributing to a differential timing of the growth take-off, eye disease should emerge as an important determinant of present-day comparative development.

In exploring this hypothesis we focus on a particular eye disease: cataract. A major motivation for this choice of focus is that cataract is the single largest determinant of global blindness; in 2002 48% of global blindness was attributable to cataract (Lansingh et al., 2007).[1] We document, using data for a cross section of countries, that low vision due to cataract is a strong causal determinant of differences in labor productivity. This conclusion is reached invoking 2SLS estimation, where we employ an identification strategy designed to generate climate induced variation in the pervasiveness of cataract in a population. Moreover, we argue that the size of the estimated impact from cataract can plausibly be accounted for by variations in the timing of the take-off to sustained growth.

[1] In beginning of the 21st century 37 million people were blind from cataract worldwide (Foster and Resnikoff, 2005).

In order to measure the extent of cataract in a cross-county setting we employ data constructed by the World Health Organization (WHO) on years lost to disability (YLD) from cataract. YLD is calculated as the product of new incidences per year, a "severity weight", and a measure of the duration of the condition. The latter two elements are constants implying that all cross-country variation stems from the data on new incidences. YLD is expressed as a rate per 100,000 people in the population.

With this measure in hand we begin by documenting that cataract "intensity" is strongly and negatively correlated with average labor productivity. The OLS correlation is robust to a large set of controls for institutions, geography, culture and historical characteristics of individual countries. While our OLS results are suggestive of an impact of cataract on labor productivity, they are unlikely to establish causality since the prevalence of disability due to cataract undoubtedly depends on per capita income.

In order to make further progress we develop an epidemiologically founded identification strategy designed to generate exogenous variation in cataract prevalence. Epidemiologically, the age of onset of cataract, and thus its overall prevalence in a population with a stable age structure, is affected by solar ultraviolet B radiation (UVB-R); stronger UVB-R leads to an earlier onset of cataract (e.g., Javitt et al., 1996; Brian and Taylor, 2001; West, 2007) . The link between UVB-R and cataract has been established theoretically, through experimental work, and through a substantial number of epidemiological studies that relates UVB-R exposure to cataract incidence. With this theoretical foundation we invoke a satellite-based measure of UVB damage potential, constructed by the US National Aeronautics and Space Administration (NASA), as an instrument for cataract incidence. Consistent with the epidemiological literature we find a strong positive correlation between our measure of UVB-R and cataract incidence, making the former a viable candidate instrument for the latter.[2]

Our main result is that cataract strongly affects labor productivity; an increase in UVB-R induced cataract intensity by one percent lowers GDP per worker by roughly 0.7%, conditional on an extensive list of controls for climate, geography, institutions, culture and country specific historical characteristics.

[2] The physiological link between UVB-R and skin cancer is unlikely to jeopardize identification, as explained below.

The economic size of the estimated impact from cataract suggests that eye disease must have had a substantial historical influence on the growth path of individual nations. Consistent with this interpretation we find that UVB-R, the underlying determinant of cataract, is a robust predictor of the onset of the demographic transition, which historically have served as a strong marker of the onset of persistent growth (e.g., Galor, 2005). Also consistent with the take-off hypothesis, we find that UVB-R is a robust predictor of contemporary human capital levels. Importantly, the link between UVB-R and the timing of the demographic transition is quantitatively large enough to account for our 2SLS estimates of the influence of (UVB induced) cataract on labor productivity.

Naturally, there are various threats to identification. In particular, one may worry that UVB-R is either causally affecting other diseases, skin cancer in particular, which may influence growth via mortality, or that UVB-R is correlated with other climate-related variables that influences productivity.

We address these concerns in several ways. First, we explain why, mainly on evolutionary grounds, UVB-R is unlikely to affect growth through skin cancer and mortality. Second, throughout the analysis we carefully control for an extensive list of auxiliary climate and geography variables, which previously have been suggested as growth determinants. The list includes absolute latitude, precipitation, frost, climate zones, distance to coast or river, and others. It turns out that identification is not jeopardized by their inclusion in the empirical model. Third, we provide a set of placebo tests of our instrument. Specifically, we show that UVB-R, conditional on our controls, does not predict other diseases that are particularly prevalent in the tropics, but epidemiologically should be independent of UVB-R (like malaria, trachoma and hookworm). Against this background we conclude that the critical exclusion restriction, required for 2SLS to deliver consistent results for cataract is plausible.

The present study contributes to the macro literature which examines the impact of mortality and morbidity on development (e.g., Gallup and Sachs, 2001; Young, 2005; Acemoglu and Johnson, 2007; Weil, 2007; Ashraf, Lester and Weil, 2008; Lorentzen, McMillan and Wacziarg, 2008; Cervellati and Sunde, 2009; Kalemli-Ozcan , 2009; Chakraborty, Papageorgiou and Sebastian-Perez, 2009; Aghion, Howitt and Murtin,

2010). While previous contributions have measured health by variables such as life expectancy, height and HIV infection rates, we focus on eye disease.

Overall, our empirical work suggests that morbidity holds strong explanatory power vis-à-vis contemporary income differences. At the same time our results imply that contemporaneous improvements in morbidity may not have large effects on growth going forward, since the impact we observe today is likely the accumulated outcome of past events. In this sense, our results strikes something of a middle ground between previous contributions that suggest the impact from health on productivity is modest or negative (at least, in the short to medium run; see Young, 2005; Acemolgu and Johnson, 2007; Ashraf, Lester and Weil, 2008) and contributions that uncover a strong positive impact on growth (e.g., Gallup and Sachs, 2001; Lorentzen, McMillan and Wacziarg, 2008) .

The analysis proceeds as follows. In the next section we discuss why cataract may influence long run productivity. Section 3 contains our empirical analysis, and Section 4 concludes.

2 Why should cataract matter to labor productivity?

Cataract is an opacity of the lens of the eye, which leads to impaired vision and ultimately to blindness. The condition is progressive and, after its time of onset, may proceed slowly over a time horizon of years, or rapidly in a matter of months. In terms of risks in contracting cataract age is the strongest factor; in large part because environmentally induced damage accumulates over time. In the end most people ultimately experience cataract if they live long enough. Yet the timing of its onset varies considerably across individuals and countries.

While cataract is commonly viewed as a disease that only inflicts the elderly in the Western world, the situation is different in many developing countries. Jarrvit et al. (1996) provide evidence from population surveys in India and China regarding the incidence of cataract as a function of age. Non-trivial fractions of the populations are affected (see Table A.1 in the Appendix). In the study from India nearly 15% of the

population aged 30 years or older were affected. In China the comparable number was about 20% for the population aged 40 or above.[3]

A major problem is that many individuals who suffer from cataract in present-day developing countries do not receive adequate treatment. There are several reasons for this state of affairs.

For one thing, there is often a fundamental lack of know-how. In Africa, for instance, the number of ophthalmologists is very low: numbers such 1:1,000,000 have been reported (Foster, 1991). Inevitably, this extreme supply constraint will limit the possibility of receiving cataract surgery.

A second issue is that the quality of the treatment (if available) is often low in poor countries. For example, evaluating cataract surgery in urban India, 50% of the outcomes were classified by international experts as "poor" or "very poor", reflecting only limited post-operation vision (Dandona et al., 1999).

A third issue is the costs of surgery, including transport, which may be prohibitively high for the poor. But even if surgery is offered for free, citizens in poor countries often seem reluctant to take advantage of the offer to undergo surgery. For instance, when rural Kenyans were offered free cataract surgery only 70% took the offer (Reshef and Reshef, 1997). Similar numbers are found in a study in Nepal (Snellingen et al., 1998). The reason for this behavioral pattern is not clear, but it may involve skepticism towards modern medicine; perhaps to some extent a rational attitude, in light of previous remarks about the quality of treatment.

As a consequence of these factors there is good reason to believe that a substantial fraction of the population inflicted by cataract will not receive treatment in a developing country context, for which reason they will become disabled and ultimately blind. Similar conditions may well have prevailed in currently rich nations historically, as the treatment of cataract used to be very crude.[4]

[3] In these studies only individuals with visual acuity of 20/30 or worse were recorded as suffering from cataract. A visual acuity of 20/30 means that at a 20 feet distance to the familiar test chart for eyesight, the individual can read letters that a person with 20/20 vision (the reference standard) can read at 30 feet's distance.

[4] A preferred method for dealing with cataract involved the displacement of the lens using a needle; a method called "couching". It is noteworthy that this procedure has been practiced at least since 1000 B.C.

As a result, one may envision (at least) two separate channels through which the prevalence of cataract may influence average labor productivity: a static and a dynamic channel. The static channel derives from reduced labor market effort by working-age individuals inflicted by cataract. The size of this effect on income per capita is likely to be modest. An upper bound can be constructed by assuming the fraction of the population that suffers from cataract contributes nothing to prosperity. Hence, if cataract was eliminated, GDP per capita would rise with the share of the total population suffering from cataract. Using data deriving the study from India, mentioned above, this would amount to an overall increase by 4.3% (Jarrvit et al., 1996). This static channel is, however, unlikely to capture the full effect of cataract since the condition plausibly has dynamic ramifications as well.

The potential dynamic effect of cataract is best understood seen through the lens of the literature that models the transition to the modern growth regime (Galor and Weil, 2000; Galor and Moav, 2002; Lucas, 2002; Hansen and Prescott, 2002; see Galor, 2005 for a survey). The goal of this literature is to elucidate the forces that trigged the abrupt change in income per capita growth, which first occurred in Western Europe sometime late in the 18th century. A common element in this body of work is the contention that the demographic transition played an instrumental role in facilitating the remarkable growth acceleration.

The theoretical reasoning, motivating a tight link between the demographic transition and the growth acceleration, is easy to grasp. Prior to the demographic transition increases in income stimulated fertility and thus translated into greater population levels, which kept income per capita levels from rising. In other words, Malthusian forces lead to stagnating living standards. After the demographic transition, however, rising income is associated with declining fertility. The dramatic reversal of the income/fertility nexus, which is the outcome of the demographic transition, has several critically important effects on the growth process. The demographic transition serves to reduce capital dilution and thus increase resources per capita, thereby stimulating labor productivity. Moreover, it facilitates intensified child investments in the form of human capital accumulation. By stimulating productivity, higher human capital investments

(e.g., Corser, 2000), testifying to the fact that cataract was a well known condition requiring treatment even in antiquity, in spite of shorter life spans.

subsequently paves the way for a virtues circle involving rising per capita income, further reductions in fertility, and greater child investments.[5]

The leading theory for the onset of the demographic transition is that a gradually rising return on human capital accumulation eventually triggered a substitution of child quantity (family size) for child quality (capital investments per child) at the household level (Galor, 2005). According to this theory, the inherent return on skill accumulation is key to an understanding of comparative differences in the timing of the onset of the fertility decline, and thus the emergence of sustained growth (Galor, 2010). This is where eye disease may have played a role. By lowering the time span over which skill investments can be recuperated, an early onset of cataract will work to lower the return on human capital accumulation. As a consequence of a lower inherent return to skills, high cataract incidence may therefore serve to delay the onset of the demographic transition. As a result of this delay, an income gap will emerge between countries with high and low incidence of the eye disease. A century later this divergence, attributed to a differential timing of the take-off to sustained growth, should be detectable in the data.

To illustrate these ideas a little more formally, consider the following crude representation of the long-run growth process. For a county i at time $t > s_i$, the level of (log) GDP per worker, y_{it}, can be written

$$y_{it} = y_{i0} + (t - s_i)g,$$

where s_i is the country specific timing (year) of a take-off in growth in labor productivity, or the timing of the demographic transition as argued above.[6] The implicit assumption is that between time zero and s_i, the economy stagnates; y_{i0} can be viewed as the "level of subsistence", or the equilibrium level of income per capita prior to the take-off. But then, for all $t > s_i$, the economy grows at the rate $g > 0$. We assume that g, the long run trend growth rate, is shared by all countries. Moreover, suppose the timing of the take-off is explained by some underlying characteristic, x_i, and by other (orthogonal) factors, $\tilde{s}_i$. That is,

[5] In addition, the demographic transition temporarily increases the relative size of the working age population, thereby stimulating growth in income per capita. In the present context, however, we focus on labor productivity, for which reason this effect is immaterial.

[6] This mechanical way of capturing the impact of a differential timing of the take-off on 21st century income outcomes is inspired by Lucas (2000).

$$s_i = \tilde{s}_i + \tau x_i,$$

where τ is a parameter capturing the impact of *x* on *s*.

Now imagine we run a cross-county regression of y_{it} on x_i, and that the two equations above represent the data generating process for *y*; that is, we estimate the equation $y_{it} = a + bx_i + \varepsilon_{it}$. Assume that x_i is uncorrelated with y_{i0} as well as (by construction) $\tilde{s}_i$. Then the OLS estimate, $\hat{b}$, for the impact of *x* on *y* can be written:

$$\hat{b}_t = \frac{E(y_i x_i)}{\sigma_x^2} = \tau g \frac{\widetilde{N}_t}{N} \frac{\tilde{\sigma}_{x,t}^2}{\sigma_x^2},$$

where $\widetilde{N}_t$, a subset of *N*, is the number of countries that have managed the take-off as of time *t*, $\tilde{\sigma}_x^2$ is the variance of the characteristic *x* across the $\widetilde{N}_t$ countries, and σ_x^2 is the variance of *x* across all *N* countries.

The intuition for this result is straightforward. As we assume *x* is uncorrelated with y_0, the OLS coefficient must be zero if no counties have managed the take-off; as seen above $\widetilde{N}_t = 0$ produces $\hat{b} = 0$ However, as countries start taking off, in a systematic way related to *x*, a link between *y* and *x* emerges. In the long run, assuming all countries have had their take-off, $\hat{b} = \tau g$; a unit change in *x* instigates τ years of delayed take-off, which has *g* percent as a yearly "penalty" in terms of labor productivity.

The main point of this exercise is that even if characteristic *x* has a very limited (static) impact on the level of the growth path, measured by y_{i0} (indeed, in the example above this effect is *nil*), we might nevertheless find a (potentially substantial) impact on y_{it} due to the influence of *x* on the timing of the take-off.

More specifically, one may suppose the pure participation effect from cataract is limited. Nevertheless, if variations in cataract onset, and thus prevalence, influenced the timing of the take-off, via its influence on the return to skills, we can expect to find an impact from this eye disease on GDP per worker levels at time *t>s*.

In addition, contingent on a well identified estimate for the impact of (UVB-R induced) cataract on contemporary GDP per worker we can provide a check of the size of the point estimate by, in a first step, backing out the size of the delay that cataract should induce, given this interpretation. That is, with a reasonable guess for the steady state growth rate, g, we can calculate $\tau = \hat{b} / g$. In a second step, we can then directly examine the impact of cataract on the timing of the demographic transition so as to assess whether data supports the delay required to account for our productivity estimate.

3 Empirical analysis

The empirical analysis proceeds as follows. Section 3.1 presents our data, while Section 3.2 contains OLS regressions. In Section 3.3 we then lay out the identification strategy in detail, while Section 3.4 presents the 2SLS results. Finally, in Section 3.5 we discuss threats to identification.

3.1 Data

Our main dependent variable is GDP per worker, which is obtained from Penn World Tables 6.3. Throughout we thus focus on the impact of eye disease on labor productivity.

Our "incidence of cataract" measure for each country corresponds to the number of Years Lost to Disability (YLD) in 2004, expressed as a ratio of per 100,000 people in the population. Formally, $YLD = I \bullet w \bullet L$, where I is (new) incidences per year, w is a weight measuring the severity of the condition, and L is the average duration of the condition. The weight w is the same everywhere, and so is L. Consequently, the cross-country variation in the variable stems from I. Further details are given in the Data Appendix.

In selecting control variables we follow the literature on "fundamental determinants of productivity", which emphasize three major underlying causes of diverging development outcomes: Institutions, Culture, and Geography/Climate (Acemoglu, 2009, Ch. 4).

Naturally, it is impossible to fully capture the institutions of a nation, the culture of its population, and all relevant aspects of its climate and geography. Another difficulty in

controlling for the institutional setup of a nation and its cultural traits is that both are endogenously determined.

Our attempt to confront these difficulties consists of employing groups of variables from the literature that have been used as instruments for institutional and cultural developments, in addition to controls capturing the geography and climate of a country. Hence, by introducing multiple proxies for institutions and culture simultaneously, we hope to capture both more fully. In addition, by employing controls that plausibly are exogenous, we can include them in our OLS analysis as well as in the 2SLS estimations.[7]

Accordingly, in an effort to capture INSTITUTIONS we include simultaneously a full set of legal origin dummies and the country population density in 1500. Two other sets of variables, which may have influenced institutional and cultural developments in their own right, are: (i) PRE-INDUSTRIAL HISTORY, which includes the state antiquity index and time past since the Neolithic revolution, and (ii) COLONIAL HISTORY, which contain controls for the identity of the colonial power. To control for CULTURE we have selected three separate variables: fraction of the population being Catholic and Muslim, respectively, and the ethno-linguistic fractionalization index. In order to capture GEOGRAPHY we combine a full set of continent dummies, a country's distance to coast or river, absolute latitude, and percentage of a country's surface area that is covered by water (lakes and rivers). To control for CLIMATE we have selected six separate variables: malaria ecology, percentage share of land area in the tropics, average temperature (population weighted), precipitation (also population weighted), average number of frost days, and lightning density. Altogether we include 27 additional controls, assigned to five sources of influence: PRE-INDUSTRIAL HISTORY, COLONIAL HISTORY, INSTITUTIONS, GEOGRAPHY and CLIMATE.

Finally, in the context of 2SLS estimation, we employ data on UV radiation. NASA produces daily, satellite-based data for ultraviolet exposure (UVE). This measure is designed to capture the potential for biological damage due to UV radiation.

The UV index captures the strength of radiation at a particular location, and is available in the form of geographic grids and daily rasters with pixel size of 1 degree latitude x 1 degree longitude. In the analysis below we rely on data for daily local-noon irradiances

[7] Details on the sources of the data are found in the Data Appendix.

for 1990 and 2000, and produce average yearly UVE levels for each country. Following Dell, Jones and Olken (2008), we compute yearly weighted averages, where weights are given by the proportion of country population living in each corresponding 1 x 1 degree average UVE cell. In our analysis below we employ an average for the 1990 and 2000 observation. But our results are not sensitive to this particular choice; the correlation between the average and the individual UV observations for 1990 and 2000, respectively, is above 0.97. Figure 1 provides a visual illustration of the UV data, and summary statistics as well as pairwise correlations between the individual controls are found in the Appendix.[8]

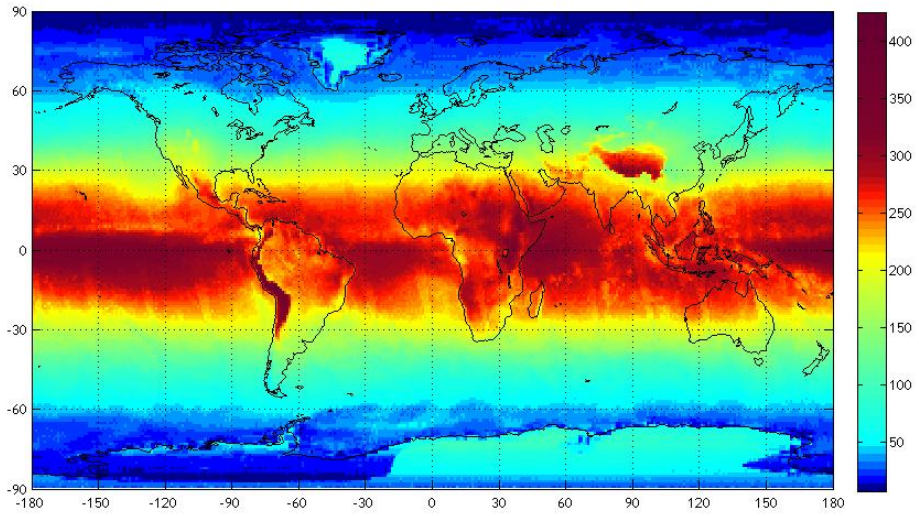

Figure 1. Daily average of biological damage potential per sq km due to solar irradiance (average 1990 and 2000).
Notes: See text or Data appendix for details on the index.

3.2 OLS results

The basic specification we take to the data has the following form

$$\log(y_i) = \beta_0 + \beta_1 \log(c_i) + Z_i'\gamma + \varepsilon_i,$$

where y is labor productivity (GDP per worker), c is cataract prevalence per capita and Z is a vector of (sets of) additional controls: PRE-INDUSTRIAL HISTORY, COLONIAL

[8] Further details on the UV data are given in the Data Appendix.

HISTORY, INSTITUTIONS, CULTURE, GEOGRAPHY, and CLIMATE. The parameter of interest is β_1.

The results from estimating this equation by OLS are reported in Table 1. The first column is the result of a regression of GDP per worker on the cataract variable. Since both variables are in logs, the coefficient is an elasticity. We therefore have that an increase in cataract of one percent is associated with roughly one half percentage decrease in labor productivity. This simple regression explains about one third of the variation in labor productivity.

[Table 1 about here]

In columns 2 to 7 we run regressions with all sets of controls, adding them one set at a time. Two things are worth notice. First, the partial association between cataract and labor productivity is significant at one percent in all columns, and the point estimate is always in the interval [-0.556, -0.293]. Second, with the exception of pre-industrial history controls, all controls are (at least marginally) significant.

In column 8 we include all control sets, amounting to a total of 27 predetermined control variables. In this case, where the number of observations drop to 89, cataract turns insignificant. However, since OLS is likely to be plagued by all variants of endogeneity bias, the results in Table 1 can obviously not form a basis for credible causal inference.

3.3 Identification

Ultraviolet (UV) radiation is a form of electromagnetic radiation which is found in sunlight. There are three types of UV radiation: A, B and C. These three varieties of UV radiation are distinguishable by their wavelength; UVA radiation has the longest wavelength (yet shorter than visible light), UVC the shortest, and UVB is in between.

Of the three forms of UV radiation, UVC is considered the most harmful to humans. Fortunately, this form of electromagnetic radiation is filtered out by the atmosphere, leaving only UVA and UVB with the potential to affect life forms on Earth.

In the present context we focus on UVB radiation (UVB-R) as it causes cataract; a proposition which, by now, has a very solid scientific foundation.[9] First, theoretical mechanisms, connecting cataract with UVR-R, has been established (see e.g. Dong et al., 2003 and references cited therein). Second, controlled animal experiments have confirmed the impact of UVB-R on the formation of cataract (e.g., Ayala et al. 2000). Third, epidemiological studies have demonstrated that greater exposure to UVB-R produces an earlier onset of cataract in human populations (e.g., Hollows and Moran, 1981; Taylor et al., 1989; West et al., 1998). It seems fair to say that a consensus has been reached on the issue.

As a result, the use of UVB-R as an instrument for cataract prevalence has a strong theoretical anchor. We therefore propose an identification strategy which schematically can be written as

$$\text{UVB-R} \rightarrow \text{Cataract} \rightarrow \text{Labor productivity.}$$

That is, we invoke our measure of UVB-R damage potential (described above) as an instrument for YLD due to cataract.

One may perhaps worry about the fact that our instrument only captures *potential* damage from UVB-R. Obviously, counter measures can be taken, and factors like whether individuals are working and spending leisure time outside matters as well. It is therefore an imperfect measure of *actual* exposure. However, it is well worth to bear in mind that we are not attempting to prove a link between UVB-R and cataract; the natural science literature has already been successful in this respect. Instead, by employing our instrument we are merely examining whether there is sufficient aggregate information in the variable in order to serve as an epidemiologically founded instrument for cataract prevalence, in spite of the obvious "noise". To this one may add that researchers in epidemiology already have employed the same UVB variable as a determinant of cataract in population studies; in an effort to capture the ambient environment (e.g. West et al., 1998). As a result, we believe our choice of instrument is well motivated theoretically as well as in terms of measurement.

[9] Surveys of the literature are found in Javitt et al. (1996) and West (2007).

A more important issue is that UV radiation clearly features a strong latitude gradient, as is plain to see from Figure 1 above. In fact, the simple correlation between our measure of UV exposure and absolute latitude is -0.95. In the 2SLS analysis to follow, we control for absolute latitude. Accordingly, when we employ UV radiation as an instrument, identification is obtained from the variation in UV exposure which is orthogonal to absolute latitude.[10]

Two climate/geography traits create variation in UV radiation which is orthogonal to absolute latitude: cloud cover and, to a lesser extent, elevation. In places with more cloud cover UV radiation is lower, and at higher altitudes UV exposure is higher. Since cloud cover and nation specific topography do not follow latitude fully, these features provide the said variation in UV exposure. It is worth reflecting on whether these sources of variation are problematic from the point of view of identification.

Clouds obviously have other roles to play aside from shielding humans from harmful UV radiation. In particular, clouds bring precipitation, which influences agricultural productivity. As a result, one worry is that places with low UV radiation are characterized by more plentiful rainfall, which has a direct productivity effect via agricultural productivity. Fortunately, we are able to control for precipitation directly, eliminating this particular basis for concern.[11]

The elevation of a country above sea level may also have independent effects on productivity. Diamond (1997) discusses the challenges involved in developing complex societies in such regions. If high altitude regions have had a historical growth disadvantage, the ramifications may still be felt today, which would jeopardize identification. However, by controlling for PRE-INDUSTRIAL HISTORY we believe the long-run development impact from elevation, as described by Diamond (1997), should be accounted for.

There may nevertheless be a few additional channels whereby the elevation of a country could matter. Trade costs may be higher at higher altitudes, as transport by water is impossible. We believe, however, that this channel is accounted for by our

[10] In the specifications where latitude enters, that is.
[11] Another weather phenomenon which emanates from clouds is lightning activity, which may influence IT diffusion by instigating power surges and sags (Andersen et al., 2008). In our regressions we also control for lightning density (strikes per square kilometer per year).

GEOGRAPHY controls: distance to coast or navigable river in particular. Finally, average temperature and precipitation are systematically different at high altitude, for which reason elevation could be correlated with agricultural productivity or disease vectors. However, temperature and rainfall are also on our list of controls.

In sum, when we control for absolute latitude, we obtain identification by comparing countries with greater or smaller UV radiation than what would be predicted by countries' latitude. In practice, these deviations have to do with cloud cover and topography. We believe that the potential blessings (unrelated to eye disease) of cloud cover and elevation above sea level are accounted for in our controls. Hence, it seems plausible to expect that our UV instrument captures cataract. This expectation is confirmed by our first stage regressions below.

We have saved for last what might seem to be the greatest challenge to identification: Theoretically, UVB-R could be a potential determinant of longevity, as excessive UVB-R leads to skin cancer, and since UVB-R simultaneously is the key source of Vitamin D, which in turn affects the immune system. We return to this issue in detail in Section 3.5.1.

3.4 Two-stage least squares and the reduced form

Table 2, which is symmetrical to Table 1, provides 2SLS results when we use UV radiation as an instrument for cataract. Note first that the instrument is strong in columns 1-5 and 7; it is weaker in columns 6 and 8. In the latter two columns a standard concern is that the usual asymptotics will not guarantee a good approximation to the actual sampling distribution. The seriousness of this concern is sometimes exaggerated for the following reason: when the first-stage F statistic allows rejection the null of no conditional relationship between UV and cataract, just identified estimation remains median unbiased with almost perfect coverage rates (Angrist and Pischke, 2009a, b).[12] Nevertheless, we do address it below.

Turning to the slope estimates, we see that cataract is always significant at five percent. Moreover, the 2SLS point estimate is bounded between [-1.387, -0.615], which is higher

[12] The coverage rate is the probability that a confidence interval includes the true parameter; a near perfect coverage rate remain close to 0.95 for a confidence level of 95%.

(in absolute terms) than the OLS point estimate. This suggests that simultaneity bias dominates any measurement error bias.[13]

[Table 2 about here]

Median unbiasedness notwithstanding, one may check the existence of a causal impact in ways that are robust to instrument strength via the reduced form. Firstly, under the null that cataract has no impact on labor productivity, we should not be able to reject that UV radiation is zero in the reduced form. This procedure is of course fully robust to weak instruments since no information on the (partial) correlation between UV radiation and cataract is used to test that there is no relationship between labor productivity and UV radiation (see Chernozhukov and Hansen, 2008). At the same time, if a causal impact exists, we know that it must be proportional to the reduced form. Secondly, one may construct weak identification robust confidence intervals using this insight (for details, see Chernozhukov and Hansen, 2008).

Table 2 reports weak identification robust confidence intervals employing the Chernozhukov-Hansen methods. In all columns these intervals have a sharp upper bound strictly below zero; for columns 6 and 8, however, the confidence intervals are fairly wide.

Table 3 reports the results from reduced-form estimation. Focusing again on columns 6 and 8, inspection of the table reveals that UV radiation is highly significant. Conditional on a valid exclusion restriction, which we consider in more detail in the next section, we may therefore conclude that a causal relationship between labor productivity and cataract exists.

[Table 3 about here]

Judging from Table 2, and invoking median unbiasedness of just identified IV, the elasticity should be somewhere between -1.387 and -0.615. To get a sense of the economic significance we may use the results from column 1 of Table 2. A one standard deviation reduction in (log) UV damage (=0.52), implies about 0.59 log points (= [-

[13] Omitted variables bias is probably less of a problem, at least not in column 8 with 27 additional control variables.

0.52*1.9*(-0.6)]) of an increase in GDP per worker, which translates into an increase in the level of GDP per worker by roughly 80%.[14]

Is this a large effect? The study by Ashraf et al. (2008) may serve as a benchmark for comparison. Using an augmented Solow model the authors calibrate the long-run impact on aggregate labor productivity from a large health improvement, corresponding to an increase in life expectancy from 40 to 60 years. The imposed individual level productivity impact from health improvements is anchored in micro estimations. According to the Ashraf et al. simulations aggregate long-run labor productivity may rise by around 15%. In this light the estimate obtained above seems very large indeed. Theoretically, however, the calibration approach of Ashraf et al. involves an economy which has already "taken off". If morbidity has served to delay the onset of growth itself, the accumulated impact on labor productivity could well be much larger than what a calibrated Solow model suggests. But how much of a delay would be required to account for the estimate above? Assuming countries, post transition, grows at two percent per year on average, the "required" delay would be $\Delta s = \log(1.80)/0.02$, or roughly 29 years. Hence, our 2SLS estimate implies that an increase in cataract, prompted by 0.52 log units of higher UV exposure, delays the growth take-off (/the onset of the demographic transition) by roughly one generation.

In an effort to gauge whether a delay of this magnitude is plausible we next examine the link between eye disease and the timing of the fertility decline.[15] Ideally, we would like to estimate the impact of cataract on the date of the fertility decline, instrumenting it by UVB-R. Unfortunately, this is not feasible since survey data on cataract prevalence only is available for 2004. But what we can do instead is to estimate the reduced form involving UVB-R and the timing of the fertility decline.

According to the hypothesis advanced above, UVB-R induced cataract prevalence has served to delay the onset of the demographic transition, thus influencing contemporary income variation. Hence, the two questions we now turn to are: Does UVB-R predict the timing of the demographic transition? And, if so, is the estimated delay in the timing of

[14] A very similar result is obtained if we invoke the reduced form results, Table 3 column 1.
[15] The cross-country data on the timing of the fertility decline is taken from Rehrer (2004). See the Data Appendix for details.

the demographic transition sufficiently large to account for the productivity effect of UVB-R induced cataract?

To limit the risk that omitted variable bias influences our estimates, we introduce the same extensive set of control variables that were employed above. Tables 4 and 5 report the results from estimating the link between UVB-R and the date of the fertility decline; the difference between the two tables consists in whether UV radiation is weighted by population shares or not.

[Table 4 & 5 about here]

The general message from the two tables is that areas exposed to more UVB-R have experienced the fertility decline at a later date. In column 1 of the two tables we note that UVB-R can account for around 60% of the variation in the date of fertility decline; when all our controls are added simultaneously we can account for about 85% of the global variation in the timing of the fertility decline.

UVB-R is significant throughout, with one exception. When the UV radiation is weighted by population, and in the context of a full set of controls, it looses significance. This is not the case when the variable is unweighted (cf. column 8 in Tables 4 and 5). It is not clear what explains this phenomenon. But it is possible that by weighting the UV variable by population, it becomes endogenous.

Consider the following line of reasoning. The growth process involves increasing urbanization, which implies that the distribution of the population across locations change. If urban centers are located in places with less UV radiation (in more temperate areas, say) we would expect that an earlier onset of the fertility transition *produces* less (measured) UV radiation because of the changing distribution of the population across locations associated with urbanization. That is, in places that experienced the fertility decline earlier, the population has shifted to low UVB-R areas, which mechanically reduces the population weighted UV measure. This reverse causality problem may induce a negative bias on the coefficient for UVB-R weighted by population, which could explain why the point estimate for UVB-R is smaller in column 8 of Table 4 than in column 8, Table 5.

Hence, the unweighted UVB-R variable may therefore be the better choice for the analysis at hand. As a result, we will focus on the estimates reported in Table 5 to assess the quantitative implications of UVB-R.[16]

In terms of economic significance one may observe that UVB-R seems to have a strong influence on the timing of the fertility decline. To minimize the risk of omitted variable bias tainting the UVB-R estimate, consider Table 5, column 8. Taken at face value the estimate implies that an increase in UVB-R by one percent delays the fertility decline by roughly 50 years. Alternatively, a one standard deviation reduction in (log) UV damage (=0.52) delays the transition by roughly 25 years, which is just about the delay needed to motivate our 2SLS estimate above (in the context of the identical experiment: 29 years). Hence, in this light our 2SLS estimate appears to be meaningful.

One of the major reasons why the timing of the fertility decline matters to growth is its effect on human capital accumulation. Hence, as a final check Tables 6 and 7 report the results from examining the link between UVB-R and average years of schooling in 2000.

[Table 6 & 7 about here]

Once again UVB-R proves to be strongly linked to the outcome variable in question; UVB-R can, by itself, account for 45-50% of the variation in average years of schooling in 2000. As before the UVB-R variable which is adjusted for the distribution of the population is slightly less robustly correlated with the dependent variable than the unweighted version; quite possibly for the same reason. Hence, once again we focus on the results involving unweighted UVB-R.

Turning to column 8 of Table 7 we find that an increase in UVB-R of one percent increases average years of schooling by 5.6 years. To put it differently, a one standard deviation reduction in log UVB-R is associated with 2.9 more years of schooling in 2000. If we assume, as is commonly done in the literature on development accounting (see Caselli, 2005 for a survey), that each year of schooling increases productivity by 10%, this increase in schooling can motivate a productivity effect of roughly 33% (=exp[0.52*5.6*0.1]).

[16] Similar concerns may be relevant in the context of our IV regressions above. However, our results are very similar if we use unweighted UVB-R as an instrument for cataract, rather than the weighted version.

Hence, 40 percent of the impact of cataract on GDP per worker in 2004 (=0.33/0.80) can be directly attributed to human capital accumulation. The remaining part may then be due to the indirect effects of human capital on productivity and per capita resources (i.e., land per worker and capital per worker), which occurred in the aftermath of the demographic transition.

In sum, cataract appears to have a strong impact on productivity, and it seems plausible that the impact is caused by a delayed onset of the demographic transition as this mechanism can, plausibly, account for the size of the 2SLS estimate. At a more proximate level, human capital appears to be a major transmission mechanism linking cataract to labor productivity.

3.5 Threats to Identification

This section falls in two parts. In 3.5.1 we discuss the potential problem that UVB-R epidemiologically affects skin cancer; our instrument is therefore causally related to another disease, which raises questions about the validity of the exclusion restriction. Subsequently, we discuss the potential concern that UVB-R, by exhibiting a strong climate gradient, may be spuriously correlated with other diseases; if so, this to would jeopardize identification.

3.5.1 Other potential causal effects of UVB-R

As is well known, skin cancer is caused by sun exposure: overexposure to UVB-R more specifically. At the same time UVB-R has a more benign role to play in also being the human body's main source of vitamin D; a key vitamin, which influences the immune system and thus longevity. Accordingly, through these mechanisms, UVB-R potentially influences labor productivity. If so, the exclusion restriction is suspect.

However, UVB-R is unlikely to be a cross-country determinant of longevity for evolutionary reasons. Over millennia evolutionary pressures have changed human skin pigmentation so that a balance has been struck between the beneficial and harmful effects of UVB-R on longevity. That is, a balance has been found between the need to lower the risk of skin cancer, while at the same time enabling enough vitamin D to be

absorbed through the skin. Consequently, in "high UV regions" skin complexion turned darker, while human skin color became lighter in "low UV regions".[17] Obviously, this does not mean that sun exposure is inconsequential to skin cancer; on the contrary, excessive UVB exposure is indisputably a major explanation why some individuals develop melanoma while others do not. What is does mean, however, is that UVB-R is unlikely to causally determine longevity in a cross-country setting, via its effects on vitamin D supply and skin cancer, since evolution has traded these two factors off against each other during the selection process involving local skin color.

But what if an "optimal" balance between the positive and negative impacts from UVB-R on longevity has not been achieved by way of natural selection? In that case, one may still argue the UVB-R/skin cancer mechanism could account for some of our results above. For instance, a negative correlation between UVB-R and labor productivity (the reduced form discussed above) could be attributable to higher mortality rather than eye disease (if larger life span is good for aggregate productivity). By implication, the IV analysis will be flawed as the exclusion restriction is violated.

Even winking away the evolutionary argument above, we do not believe this line of reasoning is viable for two reasons. First, empirically the contribution of skin cancer to annual deaths around the world is minuscule. WHO has produced data decomposing the causes of death for a large cross-section of countries in 2002 from which it is clear that skin cancer on average accounts for only about 0.18% of annual deaths (with a standard deviation of 0.18). Hence, the UVB-R/skin cancer/longevity channel cannot be of much practical importance at the aggregate level.[18] Second, and perhaps more importantly, the fraction of deaths due to skin cancer is not correlated with UVB-R, as shown in Figure 2, conditional on our exogenous controls from the analysis above. As seen from the figure, the (partial) correlation is actually negative, and insignificant.

[17] See Diamond (2005) for a clear exposition of these points and references to the relevant literature.

[18] The fact that skin cancer, quantitatively, is such a relatively unimportant source of annual deaths is perhaps also in some measure a result of the evolutionary mechanism mentioned above. The only two countries in the world where skin cancer accounts for roughly 1% of annual deaths (i.e., almost four standard deviations above the world average) are Australia and New Zealand, where the bulk of the population, evolutionarily speaking, are not adapted to their local environment. While the fact that Australia and New Zealand are "skin cancer outliers" does suggest that evolution has served to limit death from skin cancer, it is worth noting that, even in these two extreme cases, skin cancer remains a very modest source of annual deaths.

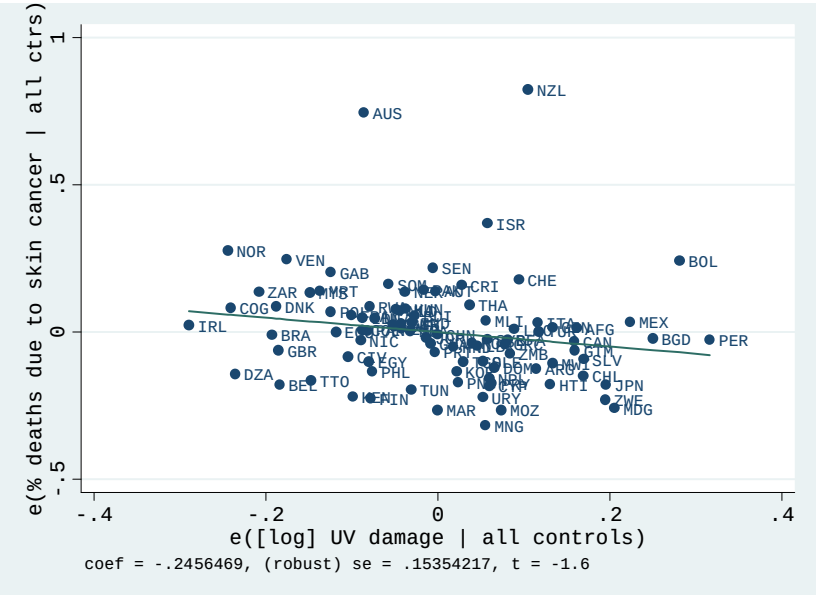

Figure 2. UV radiation and frequency of death due to Skin Cancer in 2004.
Notes: The figure shows the partial correlation between the skin cancer death rate (:=deaths due to skin cancer relative to total deaths) across the World, conditional on the full set of exogenous controls from Table 2, column 8. *Data sources*: Data on deaths is from WHO. On UV radiation see Section 3.1.

As a result, in practice UVB-R does not explain comparative differences in the frequency of deaths due to skin cancer, and thus cannot motivate cross-country differences in longevity. The natural interpretation is that evolution, and perhaps cultural adaptation as well, is the cause of the absence of a cross-country link between UVB-R and skin cancer.[19] Overall, identification is therefore unlikely to be jeopardized by UVB-R's physiological impact on skin cancer and vitamin D supply.

3.5.2 Other potential spurious correlates with UVB-R

In spite of our attempts to carefully control for other links between climate and productivity, one may worry whether our instrument could be picking up some alternative avenue of influence. Of particular concern is a potential mapping between our UV variable and other diseases with higher incidence in tropical climate zones. That

[19] Once again, this observation is in no way inconsistent with the fact that individuals diagnosed with skin cancer with high probability have spend too much time exposed to UVB-R.

is, it might be the case that our instrument is *spuriously* correlated with other diseases that in turn exerts a direct impact on productivity.

To examine whether this issue is likely to jeopardize identification we perform a set of placebo regressions. That is, we examine whether UVB-R, conditional on our full set of exogenous controls, is correlated with diseases that epidemiologically are independent of UV radiation but at the same time are more pervasive in tropical regions near the equator. Hence, these exercises directly speak to the plausibility of the exclusion restriction in the regression reported in Table 2, column 8.

The data for the alternative diseases also derive from the WHO, and represents YLD, just as our cataract data (see Data appendix for a description of the individual diseases). Table 8 reports the regression results

[Table 8 about here]

The first column reproduces the first stage results from Table 2, column 8; conditional on 27 additional controls, UV radiation is significantly correlated with cataract. The next five columns examine the correlation between UVB-R and alternative eye diseases. Of particular note is perhaps the results for Trachoma, an infectious eye disease with a particularly high incidence rate in tropical regions in general and Africa in particular. Yet, as can be seen from column 2, our instrument is not significantly correlated with this ailment.

In columns 7-11, we examine the correlation between UVB-R and a list of additional diseases. Of particular note are the results with regard to three diseases that have been extensively studied in the literature: HIV/AIDS, Hookworm, and Malaria. In spite of the fact that these diseases also are much more pervasive in tropical areas near the equator, UVB-R is insignificantly correlated with each of them.

Naturally, it is impossible to rule out that UVB-R is picking up some alternative factor, other than cataract. Still, it is encouraging that our instrument appears to be (partially) uncorrelated with key tropical diseases, which past research have emphasized. Overall, we view this check as a good indication that the required exclusion restriction is met in our 2SLS regressions above.

4 Conclusion

In the present study we have examined the impact of eye disease on long-run development; cataract specifically.

Our key result is that cataract appears to hold strong explanatory power vis-à-vis contemporary labor productivity. This conclusion is reached invoking an epidemiologically founded identification strategy, involving the use of UVB-R as an instrument for cataract incidence.

The sizeable point estimate that we recover from our 2SLS analysis is unlikely to reflect a static participation based impact from disability due to low vision. Instead, we hypothesize that eye disease has affected the timing of the demographic transition and thus the take-off to sustained growth, by influencing the return to skill accumulation. Hence, we argue the 2SLS estimate of UVB-R induced cataract reflects the ramifications of a differential timing of the take-off.

We find support for this interpretation when we examine the link between UVB-R and the timing of the demographic transition, as well as the UVB-R/human capital nexus. Societies more exposed to UV radiation have undergone the demographic transition more recently, and have lower levels of human capital, conditional on a large set of controls for institutions, culture, geography, climate and historical country specific characteristics. Our point estimate for the impact of UVB-R on the timing of the demographic transition is of a magnitude such that it can plausibly account for the 2SLS impact of UVB-R induced cataract on contemporary labor productivity.

We are admittedly surprised by the magnitude of impact from UVB-R. For instance, our regressions involving the timing of the demographic transition suggests that increasing UVB-R by one standard deviation (conditional on a host of controls for institutions,

culture and climate/geography) instigates a delay of about one generation. While this delay is large enough to plausibly account for our 2SLS estimates on GDP per worker, one may be skeptical that we have fully identified the source of the impact. The only proximate mechanism we have been able to uncover is eye disease; cataract in particular. However, it would be a worthwhile topic for future research to investigate further if other channels could be at work. While the analysis above would seem to rule out likely alternative explanations (other climatic or geographic factors, other diseases discussed in the literature etc), additional mechanisms might exist.

DATA APPENDIX

Main variables

A. UV radiation

NASA produces a daily, satellite-based index for erythemal ultraviolet exposure (EUVE), which is an estimate of the integrated ultraviolet (UV) irradiance, calculated using a model for the susceptibility of Caucasian skin to sunburn (erythema), and controlling for the influence of column ozone amount and cloud conditions on each day. That is, the integrated amount of energy from exposure to UV radiation over a day, within a certain area, is normalized to units that relate the biological response to this radiation. The index is thus expressed in units of biological damage per sq km, which basically relates the biological response (erythema) to the incident energy, and it can be interpreted in general as an index of the *potential for biological damage due to solar irradiation*.[20]

We rely on data for EUVE daily local-noon irradiances for 1990 and 2000, and produce average yearly EUVE levels for each country. Similar to Dell, Jones and Olken (2008), we also compute yearly population-weighted averages, where weights are given by the proportion of country population living in each corresponding 1 x 1 degree average EUVE cell.[21]

The variable *UV radiation* reported in our tables corresponds to the population-weighted EUVE average for both years. The main reason for focusing in the population-weighted average is making our data-scaling comparable with Dell, Jones and Olken's (2008) temperature and precipitation data. (In the context of our paper, temperature and precipitation levels, along with absolute latitude, are among the most important potential confounders of the effects of

[20] Data and units are described at http://jwocky.gsfc.nasa.gov/datainfo/1README.UV, and available in the form of geographic grids and daily rasters with pixel size of 1 degree latitude x 1 degree longitude, at the Total Ozone Mapping Spectrometer website at NASA, http://toms.gsfc.nasa.gov/ery_uv/euv_v8.html. Countries' geographic area definitions are taken from the U.S. Board on Geographic Names' database of foreign geographic names and features, http://geonames.usgs.gov/domestic/download_data.htm.

[21] Population data for this scheme are taken from the Gridded Population of the World dataset, version 3, produced by the Center for International Earth Science Information Network of the Earth Institute at Columbia University, http://sedac.ciesin.columbia.edu/gpw/index.jsp.

exposure to UV radiation.) The results remain basically unchanged if we use un-weighted EUVE average instead, and are available upon request.[22]

B. Cataract incidence

In general, the World Health Organization (WHO) quantifies the burden of a disease, as the equivalent number of years lost of "healthy" life due to the incidence (mortality and morbidity) of the corresponding disease. This measure, called Disability-Adjusted Life Years (DALY), can be interpreted as a measurement of the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability (see http://www.who.int/healthinfo/global_burden_disease/metrics_daly/en/index.html).

Our measure for the incidence of cataract in each country corresponds to the number of DALYs due to the incidence of this disease in 2004, expressed as a ratio of 100,000 people in the population. Data from WHO (2008), available at http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html. This measure is, however, equivalent to YLD (as stated in the text). Formally, DALY = YLL + YLD, where YLL is years of life lost. YLL happens to be zero in the case of cataract, for which reason DALY and YLD coincides.

C. Labor productivity

Real GDP per worker (constant prices: Chain series). Source: Penn World Tables 6.3.

Control variables

1. Pre-industrial history:

- Years since the organized state appeared (Source: Brockstette, Chanda and Putterman., 2002), and
- time passed since the Neolithic revolution (Source: Putterman, 2006).

2. Colonial history:

- dummy variables for former British, French, Spanish, or Other colonies (Source: Treisman, 2007).

3. Institutions:

- dummy variables for British, French, German, or Socialist origin of the legal system (Source: Treisman, 2007); and
- population density in 1500. Source: McEvedy and Jones, 1978 (population data), and World Development Indicators (for Land area)

4. Culture:

- Catholics and Muslims as % of the population in 1980, and

[22] The correlation between our weighted and un-weighted EUVE averages is 97.75% in 1990 and 97.67% in 2000.

- ethno-linguistic fractionalization, 1985, defined as the probability that two randomly selected individuals from a given country will not be from same ethno-linguistic group (Source: Treisman, 2007).

5. Geography:

- Continent dummies (Africa, Asia, North America, South America, Europe, Ocenania),
- mean distance to coast or rivers (Source: Gallup, Sachs and Mellinger, 1999),
- absolute value of latitude (Source: Treisman, 2007), and
- % of inland water bodies, defined as the part of total surface area that is not land, i.e., a measure of the amount of rivers and lakes as % of the country's area (Source: WDI, 2008).

6. Climate:

- Malaria ecology (Source: Sachs, 2003),
- % of tropical area (Source: Gallup, Sachs and Mellinger, 1999),
- mean air temperature and total precipitation, population-weighted averages of annual 1972-2001 levels, in C degrees and cm/year, respectively (Source: Dell, Jones and Olken, 2008),
- lightning density, defined as the average number of flashes per year per sq km, 1995-1999, (Source: Andersen et al., 2009), and
- av. number of frost days per unit of land area (area-weighted frost-days, Source: Masters and McMillan, 2001).

Other variables

A. Incidence of other diseases

DALY rates for the incidence of visual diseases other than cataract for which WHO (2008) reports data:

- Trachoma
- Onchocerciasis
- Glaucoma
- Macular degeneration
- Refractive errors

DALY rates for other sense organ diseases:

- Hear loss
- (All) sense organ diseases (all visual diseases, and hearing loss)

DALY rates for infectious, parasitic, tropical-clustered diseases that have been studied before:

- HIV/AIDS
- Malaria
- Hookworm disease.

B. Mortality rate for skin cancer

Number of deaths due to melanoma and other skin carcinomas in 2004, expressed as the total number of deaths in the country (WHO, 2008).

C. Fertility Decline

The date of the fertility transition for countries around the world are taken from Rehrer (2004). Rehrer (p. 21) explains the criteria for pinpointing the date of the transition:" *It has been set at the beginning of the first quinquennium after a peak, where fertility declines by at least 8% over two quinquennia and never increases again to levels approximating the original take-off point*".

D. Schooling

Average years of school of population aged 15 and over, as of 2000 (Source: Barro and Lee, 2000).

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Table 1: Correlations

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:	(log) Real GDP per worker, 2004							
(log) Cataract incidence, 2004	-0.489*** [0.0424]	-0.556*** [0.0426]	-0.494*** [0.0568]	-0.516*** [0.0487]	-0.442*** [0.0569]	-0.293*** [0.0649]	-0.408*** [0.0665]	-0,188 [0.148]
Observations (number of countries)	170	136	166	157	165	153	100	89
R-squared	0,33	0,51	0,37	0,38	0,38	0,59	0,65	0,83
Number of control variables	-	2	4	5	3	8	6	28
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,598						0,144
COLONIAL HISTORY			0,020					0,099
INSTITUTIONS				0,038				0,074
CULTURE					0,059			0,853
GEOGRAPHY						0,000		0,000
CLIMATE							0,000	0,211
ALL CONTROLS								0,000

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Cataract incidence is measured as the number of years lost due to disability, for incident cases of this disease (expressed as a rate per 100,000 people), estimated by WHO (2004).

Table 2: IV estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:	(log) Real GDP per worker, 2004							
Instrumented variable:	(log) Cataract incidence, 2004							
Instrument:	(log) w. average UV exposure, 1990-2000							
First Stage: effect of UV radiation on the incidence of cataracts								
(log) w. average UV exposure, 1990-2000	1.904***	1.859***	1.956***	2.115***	1.748***	1.069**	1.859***	1.437**
	(0.140)	(0.191)	(0.164)	(0.216)	(0.159)	(0.431)	(0.362)	(0.560)
(First-stage) F statistic	184,33	94,38	142,40	95,60	120,21	6,14	26,30	6,58
Second stage: effect of cataract incidence on real GDP per worker								
(log) Cataract incidence, 2004	-0.617***	-0.702***	-0.787***	-0.844***	-0.615***	-1.042**	-0.707***	-1.387**
	(0.0497)	(0.0553)	(0.0915)	(0.0821)	(0.0748)	(0.450)	(0.163)	(0.594)
95% confidence interval	[-0.714, -0.520]	[-0.810, -0.593]	[-0.966, -0.608]	[-1.005, -0.683]	[-0.762, -0.468]	[-1.924, -0.160]	[-1.025, -0.388]	[-2.550, -0.223]
Chernozhukov and Hansen (2008) weak-id. robust 95% confidence interval	[-0.727, -0.524]	[-0.827, -0.625]	[-0.977, -0.625]	[-1.028, -0.725]	[-0.777, -0.474]	[-5.884, -0.174]	[-1.178, -0.374]	[-9.388, -0.324]
Observations (number of countries)	170	136	166	157	165	153	100	89
Number of control variables	-	2	4	5	3	8	6	28
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,799						0,381
COLONIAL HISTORY			0,009					0,101
INSTITUTIONS				0,011				0,337
CULTURE					0,140			0,642
GEOGRAPHY						0,000		0,002
CLIMATE							0,000	0,050
ALL CONTROLS								0,000

Notes: 2SLS regressions. Robust standard errors in parentheses. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Cataract incidence is measured as the number of years lost due to disability, for incident cases of this disease (expressed as a rate per 100,000 people), estimated by WHO (2004). Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema), and the size of the population in the exposed areas. It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA. Weak-identification-robust confidence intervals constructed using Chernozhukov and Hansen's (2008, robust to heteroskedasticty) method.

Table 3: Reduced-form regressions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:	(log) Real GDP per worker, 2004							
(log) w. average UV exposure, 1990-2000	-1.175*** [0.114]	-1.304*** [0.127]	-1.540*** [0.170]	-1.785*** [0.148]	-1.075*** [0.145]	-1.114** [0.455]	-1.314*** [0.333]	-1.993*** [0.689]
Observations (number of countries)	170	136	166	157	165	153	100	89
R-squared	0,29	0,41	0,42	0,42	0,36	0,57	0,61	0,85
Number of control variables	-	2	4	5	3	8	6	28
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,719						0,246
COLONIAL HISTORY			0,000					0,028
INSTITUTIONS				0,000				0,018
CULTURE					0,001			0,416
GEOGRAPHY						0,000		0,000
CLIMATE							0,000	0,409
ALL CONTROLS								0,000

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema), and the size of the population in the exposed areas. It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA.

Table 4: Fertility decline and UV damage (weighted by population)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:					Year of the fertility decline			
(log) w. average UV exposure, 1990-2000	51.78*** [3.795]	51.47*** [4.663]	55.04*** [4.276]	56.87*** [5.488]	50.33*** [4.000]	34.09*** [11.45]	47.41*** [12.44]	18,06 [27.00]
Observations	117	100	114	114	116	112	80	71
R-squared	0,62	0,63	0,68	0,65	0,68	0,75	0,74	0,85
Number of control variables	-	2	4	5	3	7	6	27
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and Years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,977						0,823
COLONIAL HISTORY			0,000					0,844
INSTITUTIONS				0,366				0,738
CULTURE					0,000			0,454
GEOGRAPHY						0,000		0,063
CLIMATE							0,000	0,237
ALL CONTROLS								0,000

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema), and the size of the population in the exposed areas. It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA. The year of fertility decline, from Reher (2004), is an approximation to the timing of the demographic transition in each country.

Table 5: Fertility decline and UV damage (not weighted by population)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:					Year of the fertility decline			
(log) unw. avg. UV damage, 1990-2000	49.33*** [3.811]	47.29*** [4.210]	50.68*** [4.604]	53.63*** [6.547]	46.56*** [4.092]	34.18*** [10.48]	50.21*** [10.88]	50.19** [19.74]
Observations	117	100	114	114	116	112	80	71
R-squared	0,63	0,64	0,68	0,65	0,68	0,76	0,77	0,87
Number of control variables	-	2	4	5	3	7	6	27
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,695						0,639
COLONIAL HISTORY			0,000					0,710
INSTITUTIONS				0,292				0,616
CULTURE					0,000			0,337
GEOGRAPHY						0,000		0,032
CLIMATE							0,000	0,077
ALL CONTROLS								0,000

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema). It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA. The year of fertility decline, from Reher (2004), is an approximation to the timing of the demographic transition in each country.

Table 6: Years of schooling and UV damage (weighted by population)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:	Average years of schooling, 2000							
(log) w. average UV exposure, 1990-2000	-3.62*** [0.34]	-4.09*** [0.41]	-4.24*** [0.47]	-4.20*** [0.43]	-2.84*** [0.45]	-3.70** [1.44]	-1,23 [0.83]	-4.16* [2.10]
Observations	107	100	104	102	107	102	83	77
R-squared	0,47	0,51	0,57	0,57	0,56	0,69	0,68	0,85
Number of control variables	-	2	4	5	3	8	6	28
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,535						0,923
COLONIAL HISTORY			0,000					0,107
INSTITUTIONS				0,000				0,045
CULTURE					0,000			0,001
GEOGRAPHY						0,000		0,008
CLIMATE							0,000	0,022
ALL CONTROLS								0,000

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema), and the size of the population in the exposed areas. It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA. Average years of schooling for perople 15 years and older, from Barro and Lee (2000).

Table 7: Years of schooling and UV damage (not weighted by population)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Dependent variable:				Average years of schooling, 2000				
(log) unw. avg. UV damage, 1990-2000	-3.51*** [0.30]	-3.82*** [0.32]	-3.93*** [0.38]	-3.99*** [0.47]	-2.71*** [0.39]	-3.45*** [1.06]	-1.70* [0.88]	-5.61*** [1.59]
Observations	107	100	104	102	107	102	83	77
R-squared	0,49	0,52	0,57	0,58	0,57	0,7	0,69	0,87
Number of control variables	-	2	4	5	3	8	6	28
List of control groups and variables:	NONE	PRE-INDUSTRIAL HISTORY:	COLONIAL HISTORY:	INSTITUTIONS:	CULTURE:	GEOGRAPHY:	CLIMATE:	ALL CONTROLS
		Years since the organized state appeared, and years since the Neolithic revolution.	Former British, French, Spanish, or Other colony.	British, French, German, or Socialist origin of the legal system; and (log) population density in 1500.	Catholics and Muslims as % of the population in 1980, and ethnolinguistic fractionalization in 1985.	Continent dummies, mean distance to coast or rivers, latitude, and % of inland water.	Malaria ecology, % of tropical area, temperature, precipitation, lightning density, av. number of frost days.	
H0: β control variables = 0 (p-values):								
PRE-INDUSTRIAL HISTORY		0,794						0,706
COLONIAL HISTORY			0,000					0,150
INSTITUTIONS				0,000				0,007
CULTURE					0,000			0,000
GEOGRAPHY						0,000		0,001
CLIMATE							0,000	0,033
ALL CONTROLS								0,000

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema). It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA. Average years of schooling for perople 15 years and older, from Barro and Lee (2000).

Table 8: Placebo regressions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
		Other eye diseases					**Sense organ diseases**		**Infectious, parasitic, tropical-clustered diseases**		
Dependent variable: (2004, log) incidence of:	Cataracts	Trachoma	Onchocerciasis	Glaucoma	Macular degeneration	Refractive errors	Hear loss	(All) sense organ diseases	HIV/AIDS	Malaria	Hookworm disease
(log) w. average UV exposure, 1990-2000	1.437** [0.560]	0,733 [1.492]	-0,93 [0.879]	0,155 [0.366]	0,0189 [0.239]	0,0796 [0.289]	0,0102 [0.224]	0,151 [0.195]	0,455 [1.362]	1,992 [1.236]	1,105 [0.775]
Observations	89	89	89	89	89	89	89	89	89	89	89
R-squared	0,87	0,65	0,66	0,83	0,57	0,64	0,76	0,72	0,87	0,94	0,90
List of control groups:	PRE-INDUSTRIAL HISTORY, COLONIAL HISTORY, INSTITUTIONS, CULTURE, GEOGRAPHY, CLIMATE										
Number of control variables	28	28	28	28	28	28	28	28	28	28	28

Notes: OLS regressions. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively. All regressions include a constant term. The incidence of each disease is measured as the number of years lost due to disability, for incident cases of the respective disease. Incidence expressed as a rate per 100,000 people for all the listed diseases. Estimates of WHO (2004). The dependent variable in columns 2, 3, 9, 11 and 12 is log(1 + incidence), due to a large number of countries with 0 incidence of the respective disease. Otherwise, the dependent variable is log(incidence). Weighted average of UV exposure is an index of Erythemal exposure, constructed as the daily averge of integrated ultraviolet irradiance in 1990 and 2000, weighted by the susceptibility of caucasian skin to sunburn (erythema), and the size of the population in the exposed areas. It can be interpreted as an index of the potential for biological damage due to solar irradiation, given the column ozone amount and cloud conditions on each day. Raw daily data produced by NASA.

Table A1: Prevalence of Cataract (by age cohort)

Age cohort	Punjab, India	Shunyi, China
30 - 39	0,2%	-
40 - 49	2,2%	0,4%
50 - 59	14,7%	6,8%
60 - 69	42,0%	25,8%
70 - 79	57,7%	60,0%
80 +	87,8%	83,0%
Total	**15,3%**	**18,6%**

Source: Jarrvit et al. (1996)

Table A2: Correlation between UV radiation and controls for Pre-industrial history, Colonial history, Institutions, and Culture

	UV radiation (pop weighted)	Years since the organized state appeared	Time passed since the Neolithic revolution	Population density in 1500	Catholics, % pop 1980	Muslims, % pop 1980
Years since the organized state appeared	**-0,35**					
Time passed since the Neolithic revolution	**-0,47**	0,62				
Population density in 1500	**-0,36**	0,52	0,42			
Catholics, % pop 1980	**0,09**	-0,17	-0,23	0,08		
Muslims, % pop 1980	**0,16**	0,23	0,28	-0,11	-0,49	
Ethno-linguistic fractionalization, 1985	**0,47**	-0,39	-0,33	-0,34	-0,05	0,06

Table A3: Correlation between UV radiation and Geographical and Climate characteristics

	UV radiation (pop weighted)	Absolute latitude	Distance to coast/rivers	% inland water	Malaria ecology	% tropical area	Average n. frost days	Temperature (pop weighed)	Precipitaion (pop weighed)
Abs. latitude	**-0,95**								
Distance to coast/rivers	**0,19**	-0,17							
% inland water	**-0,09**	0,04	0,14						
Malaria ecology	**0,42**	-0,46	0,34	0,02					
% tropical area	**0,88**	-0,89	0,16	0,01	0,51				
Average n. frost days	**-0,87**	0,87	0,01	0,11	-0,40	-0,79			
Temperature (pop weighed)	**0,86**	-0,86	0,01	-0,10	0,50	0,78	-0,91		
Precipitaion (pop weighed)	**0,37**	-0,51	-0,20	0,06	0,14	0,53	-0,37	0,36	
Flash density	**0,57**	-0,68	0,19	0,06	0,47	0,64	-0,56	0,53	0,55

Table A4: Summary statistics

	Obs	Mean	Std. Dev.	Min	Max
Main variables:					
Real GDP per worker, 2004 (real PPP 2000 $ per worker)	170	24.726	24.423	934	118.730
Average UV damage (biological damage per sq km)	170	201,0	77,1	31,8	298,5
Pop.weighted average UV damage (biological damage per sq km)	170	199,3	77,0	32,3	328,5
Incidence of cataracts (DALYs per 100,000 population)	170	281,1	213,7	8,7	843,0
Control variables:					
Pre-industrial history:					
Years since the organized state appeared (index)	141	0,44	0,24	0,02	0,96
Time passed since the Neolithic revolution (000s years)	153	4,8	2,4	0,4	10,5
Institutions:					
Population density in 1500	166	6,3	9,6	0,02	62,5
Culture:					
Catholics, % pop 1980	170	30,5	35,2	0	97,3
Muslims, % pop 1980	170	24,2	36,0	0	99,9
Ethno-linguistic fractionalization, 1985	165	46,1	27,4	0	98,0
Geography:					
Absolute latitude	170	25,6	17,1	0	64,8
Mean distance to coast/rivers	153	0,33	0,45	0,01	2,39
% inland water	170	2,9	4,3	0	27,9
Climate:					
Malaria ecology	162	3,82	6,59	0	31,55
% tropical area	153	49,0	47,9	0	100
Average n. frost days	158	8,8	10,3	0	29,8
Temperature (pop weighed), C degrees	103	19,5	7,2	-1,3	28,6
Precipitaion (pop weighed) cm/year	103	11,5	7,0	0,48	39,8
Flash density (flahses per year per sq km)	170	9,5	8,9	0,018	44,4
DALY rates for the incidence of other diseases:					
Other visual diseases:					
Trachoma	170	19,7	55,2	0	355,6
Onchocerciasis (river blindness)	170	8,2	31,6	0	210,2
Glaucoma	170	85,5	48,1	12,4	241,2
Macular degeneration	170	127,3	33,7	50,0	282,2
Refractive errors and low vision	170	258,1	97,4	94,5	739,3
Other sense organ diseases:					
Hear loss	170	368,5	103,7	171,9	708,1
(All) sense organ diseases (all visual diseases, and hearing loss)	170	1.120,5	290,2	580,2	2.113,0
Some infectious, parasitic, tropical-clustered diseases:					
HIV/AIDS	170	1.989,8	5.147,5	1,38	38.460,5
Malaria	170	1.045,6	2.085,6	0	8.211,8
Hookworm disease	170	19,6	22,9	0	58,4
Other variables:					
Melanoma and other skin cancer (% of total deaths)	170	0,19	0,22	0	1,55
Average years of schooling, 2000	107	6,34	2,80	0,84	12,05