Eye Disease and Development*

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Abstract: This study advances the hypothesis that cross-country variation in the historical incidence of eye disease has influenced the current global distribution of per capita income. The theory is that pervasive eye disease diminished the incentive to accumulate skills, thereby delaying the fertility transition and the take-off to sustained growth in income per capita. In order to gauge the influence from eye disease incidence empirically, we draw on an important fact from the field of epidemiology: Exposure to solar ultraviolet B radiation (UVB-R) is an underlying determinant of several forms of eye disease; the most important being cataract, which is currently the leading cause of blindness worldwide. Using a satellite-based measure of UVB-R, we document - consistent with the proposed hypothesis - that societies more exposed to UVB-R are poorer and underwent the fertility transition with a significant delay compared to the forerunners. These findings are robust to the inclusion of an extensive set of climate and geography controls such as latitude, temperature and precipitation. Moreover, using a global data set on economic activity for all terrestrial grid cells from the Yale G-Econ project, we show that the link between UVB-R and economic development survives the inclusion of country fixed effect.

Keywords: Comparative development, eye disease, climate

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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.[1] The present study contributes to this debate by examining the link between eye disease and aggregate labor productivity.

Specifically, this study advances the hypothesis that historical variation in the incidence of eye disease has influenced the current global distribution of per capita income. The theory is that eye disease adversely affects the incentive to invest in human capital, thereby instigating a delayed fertility transition and take-off to persistent economic growth. By contributing to a differential timing of the growth take-off, which first occurred in Western Europe during the 18th century, the incidence of eye disease emerges as an important determinant of present-day comparative development.

A key challenge in testing this hypothesis is the lack of data on the historical incidence of eye disease around the world. The World Health Organization (WHO) has recently produced comprehensive survey data on disease incidence, including various forms of eye disease. But contemporary disease incidence may not be a reliable guide to disease incidence a century ago, say.[2]

In order to overcome this problem we therefore examine the link between a fundamental determinant of a cluster of eye diseases and economic development: solar ultraviolet B radiation (UVB-R). Epidemiologically, UVB-R has been shown to be a determinant of several forms of eye disease of which the most important is *cataract*. The proposition that stronger UVB-R leads to cataract has been established theoretically, through experimental work, and through a substantial number of epidemiological studies that relate UVB-R exposure to cataract incidence within human populations (e.g., Javitt et al., 1996; Brian and Taylor, 2001; West, 2007). The UVB-

[1] Some research suggests that health improvements may dramatically accelerate growth (e.g., Gallup and Sachs, 2001), whereas other studies raise doubts as to whether an improved health status in the population will have a growth enhancing effect at all (e.g., Acemoglu and Johnson, 2007).

[2] The UN launched the so-called "Vision 2020" campaign in 1999, which aims to eradicate preventable blindness (Foster and Resnikoff, 2005). As a result, a host of eye diseases are targeted for intervention, which might differentially impact on disease incidence in the developing world; the available survey data at hand is from 2004, five years after the campaign started. Moreover, in the richer parts of the world many (now curable) eye diseases are being treated, for which reason the disease incidence potentially becomes artificially low by historical standards.

R/cataract connection is particularly significant, as cataract is the single most important determinant of blindness; in 2002, 48% of global blindness was attributable to cataract alone (Lansingh et al., 2007). UVB-R is however also suspected of influencing the incidence of two other eye diseases: *pterygium* and *macular degeneration* (e.g. Gallagher and Lee, 2006). Like cataract, both of these diseases negatively influence visual acuity, and may thus also have had a deleterious effect on economic development.[3]

Against this background we invoke a satellite-based measure of UV damage potential, constructed by the US National Aeronautics and Space Administration (NASA), as an (exogenous) indicator of the historical incidence of the above mentioned cluster of eye diseases. Using recent survey data from WHO we document, consistent with the findings from epidemiology, that our measure of UVB-R predicts current cross-country differences in cataract incidence. This finding provides some assurance that our UVB-R variable may be an empirically meaningful indicator of historical eye disease incidence.[4]

We then proceed to document that countries more exposed to UVB-R are significantly poorer today as compared to countries less exposed. This result is robust to the inclusion of a rather demanding set of correlates with both UVB-R and economic development, including (absolute) latitude, precipitation and average temperature.

Taken at face value, the estimated effect of UVB-R (induced eye disease) on contemporary income per capita is economically significant. Our most conservative estimate, in the cross-country setting, implies that a one standard deviation increase in UVB-R lowers early 21st century GDP per capita by roughly 60%. This is a large effect; probably too large to plausibly reflect the direct impact of disease on individual-level earnings. But if UVB-R influenced the timing of the take-off to sustained growth, a much larger impact on current income per capita can be motivated via UVB-R's impact on e.g. historical human capital accumulation and technological change.

[3] Cataract is a clouding of the lens, which leads to blurred vision and ultimately to blindness. Pterygium is a (benign) growth of the conjunctiva, which influences an affected individual's vision if it reaches the cornea. When the macular degenerates, the individual's vision becomes blurred, ultimately rending it impossible to see fine details.

[4] Cataract is singled out in this check partly due to its key importance in terms of global blindness, partly because survey data on its incidence is available; WHO has not examined the incidence of e.g. pterygium.

Consistent with the take-off interpretation, we find that the strong correlation between UVB-R and prosperity emerges during the 20th century; it did not exist in the 18th and 19th century. Moreover, also consistent with the "take-off interpretation", we find that UVB-R is a robust predictor of the year of onset of the fertility transition, which is a strong marker of the onset of sustained growth (see e.g., Galor, 2005, 2010; Dalgaard and Strulik, 2010). The link between UVB-R and the delay of the fertility transition is quantitatively large enough to reasonably account for our reduced form estimate of the influence of UVB-R on current income per capita.

Naturally, there are alternative interpretations of an empirical link between UVB-R and economic development that cannot be ruled out *a priori*. First, one may worry that UVB-R captures another (seemingly obvious) epidemiological mechanism: skin cancer. If the incidence of skin cancer is higher in regions more exposed to UVB-R, our reduced form estimate might be convoluting an impact from mortality. Second, it seems plausible that UVB-R is (spuriously) correlated with other climate-related diseases. That is, perhaps our UVB-R estimate is capturing the influence from a (much) larger set of diseases that just happens to be pervasive in regions highly exposed to UVB-R. Finally, one may worry that UVB-R is (spuriously) correlated with relatively time invariant determinants of productivity of a non-climatic nature, such as institutions and/or cultural values and norms.

In addressing the first concern, we begin by explaining why, mainly on evolutionary grounds, UVB-R actually should *not* predict skin cancer in a cross-country setting. Consistent with the evolutionary argument, we show that UVB-R is not correlated with the incidence of skin cancer. Accordingly, it seems unlikely that the correlation between UVB-R and economic development can be attributed to a confounding influence from skin cancer.

In addressing the second concern, we submit UVB-R to a demanding set of placebo tests. That is, we ask whether UVB-R predict diseases (some of which are particularly pervasive in tropical areas) that should be *unrelated* to UVB-R on epidemiological grounds. The list includes malaria, hookworm and HIV/AIDS. In each instance we are unable to reject the null of zero correlation between UVB-R and the respective disease,

conditional on our full set of climate/geography controls (i.e., in a setting where UVB-R *does* predict cataract incidence).

In order to address the third concern we move beyond the use of the country as the unit of analysis. Instead we employ a global data set on economic activity for all terrestrial grid cells from the Yale G-Econ project (see Nordhaus et al, 2006). This data set enables us to examine the association between UVB-R and economic activity conditional on the set of controls that we employ in the cross-country regressions *as well as* country fixed effects. We expect country fixed effects to pick up the influence from political institutions and (country-wide) cultural traits. In this setting, where we solely rely on within country variation, we continue to find that UVB-R discourages economic development.

In sum, our robustness checks show that the UVB-R/income gradient can neither be attributed to skin cancer nor to other diseases that previous studies have shown to impact on growth (such as malaria and hookworm).[5] Moreover, the UVB-R/income nexus does not seem to be caused by a confounding influence from other key geographical determinants of prosperity, institutions, and culture. As a result, we are led to the conclusion that the most plausible explanation for the UVB-R/income gradient is that differential (historical) incidence of eye disease has had an important effect on the contemporary world distribution of income per capita.

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 Perez-Sebastian, 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 also imply that

[5] See Gallup and Sachs (2001) on malaria; Bleakley (2007) on hookworm.

contemporaneous improvements in (this kind of) 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; Acemoglu 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 eye disease may influence long run productivity; Section 3 discusses our empirical strategy; Section 4 contains our empirical analysis whereas Section 5 examines alternative interpretations of the link between UVB-R and income (e.g., skin cancer). Finally, Section 6 concludes.

2 Why eye disease should matter to labor productivity

As observed in the Introduction, the present study focuses on forms of eye disease which are expected to be influenced by UVB-R; of these eye diseases, cataract deserves special attention because it is the single most important cause of blindness world wide.

Cataract is an opacity of the lens of the eye, which leads to impaired vision and ultimately to blindness. The condition is progressive and may (after its time of onset) proceed slowly, over a time horizon of years, or rapidly, in a matter of months. In terms of risks of contracting cataract, age is the strongest factor 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. In the study from India nearly 15% of the population aged 30 years or older

was affected. In China the comparable number was about 20% for the population aged 40 or above.[6]

The only treatment of cataract is eye surgery, which historically was a rather precarious proposition.[7] During the 20th century the surgical techniques improved massively, but the procedure is still the work of a specialist. Unfortunately, such specialists are scarce in many developing countries. In Africa, for instance, the relative number of ophthalmologists is minuscule: fractions as low as 1:1,000,000 inhabitants have been reported (Foster, 1991). Inevitably, this extreme supply constraint limits the possibility of cataract treatment in many poor places, even today.[8] Much like cataract, surgery is needed for the treatment of pterygium; macular degeneration, by contrast, can only be prevented.

Accordingly, corrective eye surgery is unlikely to have played an important role historically, and even during the 20th century access to adequate treatment is likely to have been severely limited in many places around the world. It is therefore plausible that eye disease in general and cataract incidence in particular may have influenced comparative development. More concretely, one may envision at least two separate channels through which eye disease may influence living standards: a static and a dynamic channel.

The static channel derives from reduced labor market effort by working-age individuals inflicted by eye disease. The static channel is unlikely to be quantitatively very important however. A sense of magnitudes can be constructed by assuming that the fraction of the population suffering from cataract contributes *nothing* to prosperity; this is obviously an exaggeration designed to provide an upper bound for the impact of

[6] 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.

[7] A preferred method for dealing with cataract historically 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. (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.

[8] Another problem 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).

cataract via this participation channel. Hence if cataract was eliminated GDP per capita would rise with the share of the total population suffering from cataract. Using data deriving from the study from India mentioned above this would amount to an overall increase in income per capita by 4.3%.

The static channel is unlikely to capture the full effect of eye disease in general and cataract in particular. The potential dynamic effect of eye disease is best viewed 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 aim of this literature is to elucidate the forces that triggered the abrupt change in income per capita growth, which first occurred in Western Europe sometime late in the 18th century. A key contention of this body of work is that the fertility transition was instrumental in facilitating the remarkable growth acceleration.

The theoretical reasoning motivating a decisive link between the fertility transition and the growth acceleration is easy to grasp. Prior to the fertility transition, increases in income stimulated fertility and thus translated into greater population levels, which in turn kept income per capita levels from rising persistently due to diminishing returns. In other words, Malthusian forces lead to stagnating living standards (e.g., Ashraf and Galor, 2010). After the fertility transition, however, rising income is associated with declining fertility. The reversal of the income/fertility nexus, which is the outcome of the fertility transition, has several critically important effects on the growth process (Galor, 2011). The fertility transition serves to reduce capital dilution, and thus to increase resources per capita, which stimulates labor productivity. Moreover, it facilitates intensified child investments in the form of human capital accumulation. By stimulating productivity, higher human capital investments subsequently paves the way for a virtues circle involving rising per capita income, further reductions in fertility, and greater child investments. In addition, the fertility transition temporarily increases the relative size of the working age population, thereby stimulating growth in income per capita.

The leading theory for the onset of the fertility 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, 2011, Ch. 4). 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, say, will work to *lower* the return on human capital accumulation. As a consequence of a lower inherent return to skills, high incidence of eye disease may therefore serve to delay the onset of the fertility transition. For this reason, an income gap will emerge between countries with respectively high and low incidence of 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. A formal model, which predicts that variations in health status may have lead to a differential timing of the take-off, along the lines of the argument sketched above, is developed in Hasan and Zoabi (2006).

To illustrate these ideas a little more formally, with an eye to the empirical analysis to come, 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 as

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

where s_i is the country specific timing (year) of a take-off in growth in labor productivity, or the timing of the fertility transition as argued above.[9] The implicit assumption is that between time zero and s_i the economy stagnates; y_{i0} can be viewed as the subsistence level of income or, alternatively, as the equilibrium level of income per capita prior to the take-off. 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.

[9] 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).

Suppose next that the timing of the take-off is explained by some underlying characteristic, x_i, and by other (orthogonal) factors, $\tilde{s}_i$. That is,

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

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 the two equations above represent the data generating process for y; that is, we estimate the equation $y_{it} = a + bx_i + \varepsilon_{it}$, where ε_{it} is noise. 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:

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

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

The intuition for this result is straightforward. Since 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, $\hat{N}_t = 0$ 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 experienced 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.[10]

The main point of the 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 may nevertheless find a (potentially substantial) impact on y_{it} due to the influence of x on the timing of the take-off. In the context of the case at hand: even

[10] Here we are ignoring convergence for simplicity, which may be important post take-off. However, as long as income convergence is not complete the timing of the take-off will matter to observed cross-country income differences.

if the static (participation) effect from cataract is limited a substantial impact on income per capita can emerge if eye disease incidence influenced the timing of the take-off.

Observe finally that contingent on an estimate for the impact of eye disease incidence 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 eye disease should induce, given this interpretation. That is, with a 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 eye disease on the timing of the fertility transition (a theoretically meaningful proxy for the "year of take-off") so as to assess whether data supports the delay required to account for $\hat{b}$ our income estimate.

3 Empirical Strategy

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

$$\log(y_i) = \beta_0 + \beta_1 \log(E_i) + Z_i'\gamma + \varepsilon_i, \quad (1)$$

where y is labor productivity (GDP per worker) or GDP per capita, E is the historical incidence of eye disease and Z is a vector of additional controls.

As is well known, the level of income per capita is explained, at the proximate level, by availability of capital (physical, human) as well as productivity (technology and macroeconomic efficiency). Following the literature on "fundamental determinants of productivity" we do not control for these proximate sources of growth. Rather, we attempt to understand comparative development by introducing variables that *ultimately* should explain why some countries have more capital and higher productivity and therefore have attained a higher level of income per capita (e.g., Acemoglu, 2009, Ch. 4). The key hypothesis of the present study is that the historical incidence of eye disease is one such "fundamental determinant".

In measuring E we face the challenge that survey data on historical eye disease incidence is unavailable. As a result, we have to employ an indirect approach in capturing the eye disease incidence by employing data on UVB-R.[11]

The use of UVB-R is motivated by its epidemiological impact on various eye diseases. First and foremost, UVB-R is known to influence the incidence of cataract. Theoretical mechanisms connecting cataract with UVR-R have 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.[12]

UVB-R is also suspected of influencing the incidence of two other eye diseases: pterygium and macular degeneration (e.g. Gallagher and Lee, 2006). It should be noted, however, that there in an ongoing debate as to whether – or the extent to which – UVB-R influence pterygium and macular degeneration. Still, at this point in time we cannot rule out that UVB-R may be capturing a cluster of eye diseases: cataract, pterygium and macular degeneration. Accordingly, we proxy the historical incidence of eye disease, E, by employing data on UVB exposure.

With regards to Z 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).

[11] 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, with UVB wavelength being 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.

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

Our estimations are performed by OLS. As a result, the key issue is whether it can reasonably be argued that our UV variable is capturing eye disease and not other covariates with (fundamental determinant of) living standards. It will become apparent when we present our data on UVB-R that it features a very strong latitude gradient: the simple correlation between our measure of UV exposure and absolute latitude is -0.95. Since latitude may capture a host of mechanisms we include it in *Z.* Accordingly, in our full specification, identification is obtained from the variation in UV exposure which is orthogonal to absolute latitude.

Two climate/geography traits create variation in UV radiation beyond absolute latitude: cloud cover and 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 variation in UV exposure that is orthogonal to latitude. It is worth reflecting on whether these sources of variation are problematic from the point of view of isolating an effect from eye disease.

Clearly, the elevation of a country above sea level may have independent effects on productivity. For example, Diamond (1997) discusses the challenges involved in developing complex societies in mountainous regions. If high altitude regions have had a historical growth disadvantage, the ramifications may still be felt today, which would render the interpretation of any correlation between UV and current economic development unclear.

We confront this issue is several ways. First, we control for the timing of the Neolithic revolution. If Diamond (1997) is right this should capture the indirect economic ramifications of elevation. Second, moving beyond the Diamond thesis, elevation may have a contemporary direct effect on productivity as trade costs could be higher at higher altitudes; transport by water is surely more costly at high altitude compared to at the sea level. In order to control for this channel we include distance to coast and navigable river. Further, climatic conditions change with altitude, for which reason we also control for average temperature and precipitation. Finally, in an effort to fully control for the potential impact from topography, we also include a direct measure of average elevation.

Hence, when we control for this set of variables, in addition to latitude, the variation we exploit should essentially be that related to variations in cloud cover. Now, clouds obviously have other roles to play aside from shielding humans from UV radiation. In particular, clouds may influence agricultural productivity via precipitation and perhaps temperature. That is, places with low UV radiation may be characterized by e.g. more plentiful rainfall, which has a direct productivity effect via agriculture. Fortunately, we are able to control for precipitation and temperature directly, thus eliminating this particular basis for concern.

In sum, when we control for absolute latitude, we obtain identification by comparing countries with higher or lower 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 by our controls. Hence, it seems plausible that we are capturing differential UV exposure as motivated by *cloud cover*. Moreover, it would seem reasonable to assume that this sort of variation matters little beyond the motivated eye disease channel.

To check this assessment we examine, in Section 5, the correlation between the residual UV variation (conditional on controls) and a host of other diseases, which are epidemiologically independent of UVB-R. We also examine an affliction which is epidemiologically related to UV radiation: skin cancer. Anticipating our results, we are unable to reject the null of zero impact from UVB-R in every setting. However, as documented in the next section, the residual UV variation does predict cataract incidence.

Still, one may worry that the variation left in the UV variable, after controlling for latitude, distance to coast, distance to river, temperature, precipitation, elevation and timing of Neolithic revolution could be picking up omitted influence from institutions and culture. In the cross-country exercise we can try to capture some of this potential influence by also including the size of the country (see Olsson and Hansson, 2010 for a theory linking institutional development to country size), and continent dummies

alongside the other controls that in complex ways also may have influenced the formation of cultural values and institutions.[13] Despite this, doubts may legitimately linger.

Hence, instead of trying to capture institutions and culture by way of additional controls in the cross-country context, we re-examine the link between UVB-R and income in Section 5, employing a global data set on economic activity for all terrestrial grid cells from the Yale G-Econ project. We control for the same set of climate/geography variables discussed above, except for timing of Neolithic revolution (for which no data exist at this level of aggregation). Crucially, in this setting we can control for country fixed effects, which should partial out the potentially confounding influence from institutions and culture. Even so, our analysis reveals that UVB-R remains a significant detriment to economic development.

4 Empirical Analysis

The empirical analysis falls in three parts: Section 4.1 presents our data, while Section 4.2 contains our main results. Finally, Section 4.3 examines the viability of the "take-off hypothesis" as an interpretation of our results from Section 4.2.

4.1 Data

Our dependent variables in this section are: GDP per worker and per capita (PPP$) in 2004; current (2004) cataract incidence; and the timing (year) of the fertility decline. Most of this data is commonly used in the literature and therefore requires little further presentation; sources and brief descriptions are found in the data appendix. Still, a few remarks on cataract incidence are warranted.

Our "incidence of cataract" measure for each country is the number of Years Lost to Disability (YLD) in 2004, expressed as a ratio of per 100,000 people in the population (WHO, 2008). Formally, $YLD = I \cdot w \cdot L$, 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

[13] Of course, country area is also known to influence the intensity of trade and travel, which forms a separate motivation for its inclusion in Z (Frankel and Romer, 1999; Andersen and Dalgaard, 2011).

condition. The weight *w* is the same everywhere, and so is *L*. Consequently, the cross-country variation in the variable stems from *I*. Note that when we examine the impact of UV on a host of other diseases in Section 5, the data derives from the same source.

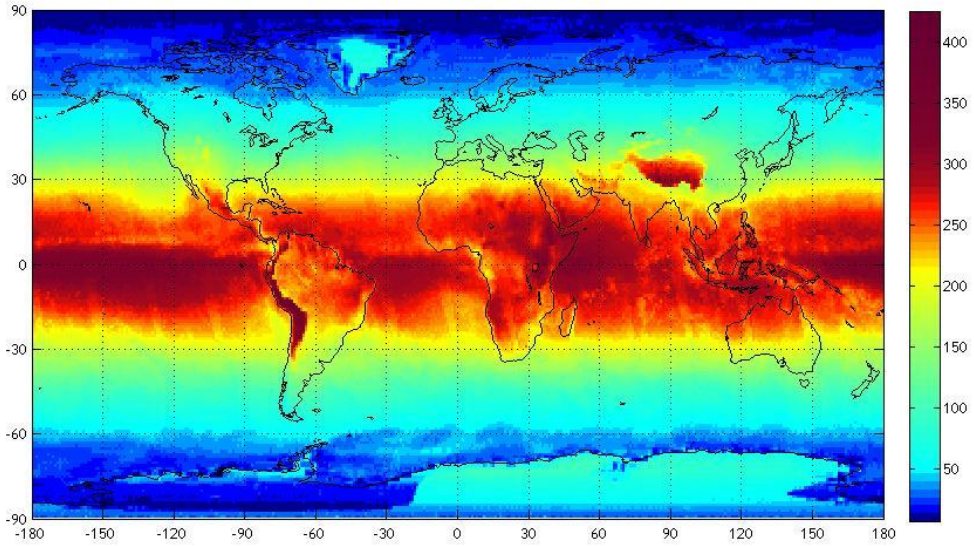

Figure 1. Daily average of biological damage potential per sq km due to solar irradiance (average 1990 and 2000).
Notes: See Data appendix for details on the index.

Our key independent variable is UV radiation. NASA produces daily, satellite-based data for ultraviolet exposure. 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 it is available in the form of geographic grids and daily rasters with pixel size of 1 degree latitude x 1 degree longitude. We rely on data for daily local-noon irradiances for 1990 and 2000, and produce average yearly UV levels for each country. That is, in our analysis below we employ an average for the 1990 and 2000 observation. 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; the correlation with latitude mentioned in Section 3 is visually obvious.

Further details on the data (including the controls discussed in the last section), summary statistics, as well as correlations between the controls and UV exposure are found in the Appendix.

4.2 Main results

The results from estimating equation (1) by OLS are reported in Tables 1 and 2, where the dependent variable is GDP per worker and GDP per capita, respectively. The first column is the result of a regression of GDP per worker on UV alone. Since both variables are in logs, the coefficient is an elasticity. We therefore have that an increase in UV by one percent is associated with a decrease in labor productivity of roughly 1.1 percent (Table 1), and 1.2 percent in the context of GDP per capita (Table 2).

[Tables 1 & 2: UV and income]

In columns 2 to 6 in the two tables we add controls sequentially, and finally include all of them at once. The partial association between cataract and labor productivity is significant at five percent (or less) in all columns.

The additional controls clearly influence the partial correlation between UV and living standards; cf. Column 6 in both tables. When all controls are added simultaneously, the UV elasticity is down to -0.8 and -0.98 for GDP per worker and GDP per capita, respectively. This suggests that some of the variation in GDP captured by UV in Column 1 is attributable to various other mechanisms, which we then manage to account for by adding controls. As demonstrated in Appendix Table A.2, the included controls account for a substantial amount of variation in the UV variable; when all are included simultaneously they account for 93% of the variation in UV. Much of the reduction in the size of the UV estimate is thus plausibly attributable to the fact that UV is strongly correlated with e.g. latitude, which influences economic prosperity in various independent ways. On physical grounds, the remaining UV variation plausibly reflects variation in cloud cover, as discussed in Section 3.

In the last column in Tables 1 and 2 we replace UV by cataract, which is arguably the most important eye disease in the cluster that should be epidemiologically related to UV. Consistent with the hypothesis in focus, we also find a strong correlation between cataract incidence and prosperity. It is worth noting that the R^2 in Columns 6 and 7 in either table are very similar. This suggests that UV and cataract are contributing in roughly equal proportion to the overall fit of the model, consistent with UV chiefly

affecting living standards via cataract; though not necessarily exclusively via cataract, as pterygium and macular degeneration may also be captured by UV.

Following up on the link between UV and eye disease, Table 3 provides the results from regressing cataract incidence on UV damage potential.

[Table 3: UV vs. Cataract]

If one were to assume that UV *solely* capture cataract, and not pterygium and macular degeneration (nor institutions or culture), Table 3 would reflect meaningful first stage regressions in a 2SLS set-up, with UV as an instrument for cataract. But since we cannot a priori exclude the possibility that UV is capturing other eye diseases, we have chosen to refrain from implementing a 2SLS solution on theoretical grounds. Nevertheless, the results are illuminating, as they provide an indication of whether UV plausibly is capturing eye disease or not, and they will be a useful benchmark when we run placebo regressions in Section 5.

Turning to the results we observe that UV indeed is significantly correlated with cataract incidence in all specifications; typically at the 1% level of confidence, though when we add all of our auxiliary controls (collectively spanning 93% of the variation in UV) the significance level widens to 10%. Nevertheless, the results do provide some assurance that the findings from Table 1 and 2 reflect the stifling effect on development from the historical incidence of eye disease.

Suppose then that the point estimate for UV indeed is capturing the causal impact of eye disease incidence on economic development: Is the impact economically significant? Judging from Table 2, column 6, we find an elasticity of UV radiation with respect to GDP per capita of -0.98. To get a sense of the economic significance, observe that a one standard deviation reduction in (log) UV damage (about 0.5) implies about 0.49 log points increase in GDP per capita, which translates into an increase in the level of GDP per capita by roughly a factor of 1.63 (= exp(0.5*0.98)), or 63%; the comparable number for GDP per worker is 49%.

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 estimates. According to the Ashraf et al.'s 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 sustained growth, the accumulated impact on labor productivity could well be much larger than what a calibrated Solow model suggests. But how viable is the "take-off interpretation" of the link between UV and current prosperity?

5.3 Exploring the take-off interpretation

As a first step, note that the results from Tables 1 and 2 themselves admit a simple check. As explained in Section 2, the fertility transition has three substantive effects on growth: (i) it increases resources per capita; (ii) it stimulates human capital accumulation, and thus indirectly productivity growth via technological change; and (iii) it leads to a temporary demographic dividend, whereby the size of the labor force relative to population increases. Importantly, the third effect *only* influences GDP per capita; it has no impact on GDP per worker. Consequently, the impact from UV on GDP per worker, if the estimates truly reflect the take-off mechanism, must be strictly smaller than the impact from UV on GDP per capita. Comparing columns 1-6 in the two tables shows that this pattern is present in the data: The point estimates for UV are consistently larger (in absolute value) in Table 2 compared to Table 1.

As a second check we examine the historical evolution of the UV/income gradient. If the take-off hypothesis is viable (and if the direct impact of eye disease on productivity is minimal) we would *not* expect to see a link between UV and income *prior* to the take-off;

only once countries start to take off would we expect to see a clear link.[14] Accordingly, using data on GDP per capita from Maddison (2003) we re-estimate the specifications in Tables 1 and 2, column 6, for the years 1700, 1820, 1900 and 1950. The results are found in Table 4.

[Table 4: The time varying link between UV and prosperity]

A consistent pattern emerges: starting from 1700 the size the partial correlation rises (in absolute value) until it turns significant in 1950. By 1950 the estimate is very similar in order of magnitude to those in Table 2, which also involves GDP per capita. From column 5 in Table 4, we see that the significance and the size of the estimate remain fairly unchanged when we restrict the "1950 sample" to countries for which GDP per capita data were also available in 1900. Put differently, the significance of UV in 1950 is not simply a matter of more data being available. These results support the hypothesis that UV's impact on current prosperity is mediated through the differential timing of the take-off across the world.[15]

As a third check of magnitudes, we ask: How much of a delay would be required in order to account for the GDP per worker estimate in Table 1? Assuming that countries, post transition, grows at between two and three percent per year on average, the required delay would be $\Delta s = \log(1.49)/g$ (see Section 2), or between about 13 and 20 years.

In order to determine whether a delay of this magnitude is plausible we next examine the link between eye disease and the timing of the fertility decline. According to the hypothesis advanced above, UVB-R induced eye disease has served to delay the onset of

[14] See Section 2: if $\tilde{N} \approx 0$ (i.e., no countries have taken-off), $\hat{b} \approx 0$.

[15] Some may speculate whether this table is not showing "too much". According to Galor and Weil (2000) for instance, the "take-off" was in full operation by 1900. From this perspective, it may seem puzzling that we do not detect a significant influence from UV in 1900 (perhaps already in 1820) if UV influences the timing of the take-off. This is not really a puzzle, however, for two reasons. First, the "industrial revolution" was initially confined to Europe. As a result, the continental fixed effects will pick up most of the information as long as the take-off is highly geographically concentrated. Secondly, the size of the estimate for UV is affected by the number of countries taking off and by the variation in UV across the countries that have taken off (see Section 2). Since the forerunners in the industrial revolution were a relatively small group of countries, and because Europe is a very small place climatically speaking, the variation in UV is relatively modest. Consequently, a modest estimate is expected prior to the 1900s. But as the industrial revolution diffuses, selectively, to other continents and more countries one would expect to see that (a) the point estimate for UV rises and (b) that statistical significance eventually emerges.

the fertility transition, thus influencing contemporary income variation. Hence, the two questions we now turn to are: Does UVB-R predict the timing of the fertility transition? Is the estimated *delay* in the timing of the fertility transition sufficiently large to account for the prosperity effect of UVB-R?

To limit the risk that omitted variable bias influences our estimates, we introduce the same control variables that were employed above. Table 5 (column 1-6) reports the result of estimating the link between UVB-R and the date of the fertility decline.

[Table 5: UV and fertility decline]

The general message from the table is that areas exposed to more UVB-R have experienced the fertility decline at a later date. In column 1 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 80% of the global variation in the timing of the fertility decline.

UVB-R is significant throughout, consistent with the hypothesis under scrutiny. Moreover, as revealed by Column 7 and 8, the fertility decline is strongly and negatively correlated with current GDP per worker and GDP per capita; the point estimates suggest that each additional year of delayed fertility transition is associated with an (forgone) income cost of about 2%.[16]

One could envision a 2SLS approach, whereby UVB-R serves as an instrument for the fertility transition; in this case Column 6 would be the first stage, and column 6 of Table 2 would be the reduced form. The identifying assumption would be that UV has zero impact on productivity beyond that working via the take-off. That is, the assumption would be that the static effect (see Section 2) is exactly zero. While we doubt the static effect is very important (and Table 4 supports this view), it is likely too strong an

[16] Dalgaard and Strulik (2010) obtains a roughly similar estimate; their controls follow the structure of the Solow model, however, and is thus not motivated by the literature on fundamental determinants as is the case in the present analysis. But the fact that this result is robust to different empirical strategies is worth noting.

assumption to make that it completely vanishes, for which reason we do not implement a 2SLS procedure in the present context.

Returning to the link between UV and the timing of the fertility transition, UVB-R does seem to have a substantial economic impact. Consider column 6 of the table: Taken at face value the estimate implies that an increase in UVB-R by one percent delays the fertility decline by roughly 24 years. Alternatively, a one standard deviation increase in (log) UV damage (ca. 0.5 log points) delays the transition by roughly 12 years, which is broadly consistent with (though somewhat on the low side of) the delay "needed" to account for our results in Tables 1 and 2 (i.e., 13-20 years).

In sum, UV appears to have a strong impact on current prosperity, and it seems plausible that the impact is caused by a delayed onset of the fertility transition as this mechanism can, to a first approximation, account for the size of the reduced form.

5 Threats to Identification

This section falls in two parts. In 5.1 we discuss the potential problem that UVB-R epidemiologically affects skin cancer. UV is therefore causally related to another disease, which raises questions about the interpretation of our estimates. Subsequently, we discuss the potential concern that UVB-R, by exhibiting a strong climate gradient (cf. Figure 1), may be spuriously correlated with other diseases. Finally, in Section 5.3, we address the problem that UV could be spuriously correlated with other fundamental determinants of productivity: institutions and culture.

5.1 Skin Cancer

As is well known, skin cancer is caused by sun exposure: overexposure to UVB-R more specifically. At the same time UVB-R plays a more benign by also being the human body's main source of vitamin D; a key vitamin which influences the immune system, and thus ultimately longevity. Accordingly, through either mechanism, UVB-R potentially influences mortality and thereby potentially labor productivity. As it turns out, however, UVB-R is unlikely to be a *cross-country* determinant of longevity through these mechanisms 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 malignant melanoma while others do not.[18] But what is does mean 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.

As a check of this argument we re-estimated the regression performed in Table 3 (column 6), exchanging cataract incidence for incidence of skin cancer. The results are found in Table 6, column 8: UV is not significantly correlated with skin cancer, consistent with the evolutionary argument. The identification of UV with eye disease is therefore unlikely to be jeopardized by skin cancer and vitamin D supply.

5.2 Other Diseases

In spite of our attempts to carefully control for other links between climate and productivity, one may worry whether UV 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 where UV radiation is most intense; it could be the case that UV is *spuriously* correlated with other diseases that in turn exerts an 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

[17] See Diamond (2005) for a clear exposition of these points and references to the relevant literature.
[18] Malignant melanoma is by far the most dangerous type of skin cancer, but it is also least common. There are two other types of skin cancer: basal cell cancer and squamous cell cancer. Basal cell cancer, the most common type of skin cancer, almost never spreads; squamous cell cancer is more dangerous, but not nearly as dangerous as a melanoma.

exogenous controls, is correlated with diseases that epidemiologically are *independent* of UV radiation but at the same time are more pervasive in tropical regions.

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 6 reports the regression results.

[Table 6 about here]

The first column reproduces the results from Table 3, column 6 (conditional on 13 additional controls) that UV radiation is significantly correlated with cataract. The next four columns examine the correlation between UVB-R and non-UV induced eye diseases. Of particular note is the result 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, UV is not significantly correlated with this ailment.

In the remaining columns we examine the correlation between UVB-R and a list of additional eye diseases, and other diseases which have been emphasized in the literature: HIV/AIDS, Hookworm, and Malaria. Despite the fact that these diseases also are much more pervasive in tropical areas near the equator, UVB-R is not significantly correlated with any of them.

Naturally, it is impossible to rule out that UVB-R is picking up some alternative disease which is not surveyed by WHO. Still, we view these checks as a good indication that our regressions in Section 4 are plausibly isolating UV's impact on productivity via eye disease.

5.3 Institutions and Culture

So far the analysis has not explicitly dealt with two sets of fundamental determinants which might influence the association between UV and prosperity: institutions and cultural values. The purpose of this section is to address this deficiency.

Naturally, institutions and cultural values are not exogenous, but represent the outcome of historical processes. As a result, we cannot rule out that the analysis above have accounted for their influence inadvertently; that is, if institutions and culture are determined by underlying climatic or geographic characteristics, the latter controls may be capturing (in part) the influence from the former on prosperity in Tables 1 and 2.[19] Still, in an effort to push the matter a little further we now move away from the individual country as unit of analysis, and instead use a global data set on economic activity for all terrestrial grid cells from the Yale G-Econ project. This will allow us to control for country fixed effects, thereby pruning GDP per capita from the influence of institutions and culture.

Figure 2: Geographic distribution of GDP per capita 2005

Figure 2 depicts the geographic distribution of GDP per capita as of 2005, using the G-Econ data. The well known pattern that income rises as one moves away from the equator is visually obvious. As it seems doubtful that the latitude gradient is solely due to UV, we continue to follow the practice of including latitude in our regressions. Indeed, the content of Z is identical to that of Tables 1 and 2, with two exceptions: (i) we are unable to control for the timing of the Neolithic revolution; (ii) we include country fixed effects rather than regional indicators. Table 7 reports the regression results, where the dependent variable is (log) GDP per capita for 2005.[20]

Table 7: UV and prosperity, G-Econ

As is evident from the R^2 in column 4, the controls and UV explains the lion's share of the global variation in living standards. Importantly, UV remains significant conditional on country fixed effect as well as the climate and geography controls motivated in Section 3. It is worth observing that the geographic/climate controls collectively captures most of the variation in UV; 95% to be precise (See Table A.3). Despite this

[19] See e.g. Durante (2009) and Michalopolous (2008) for evidence of climate's impact on culture, and e.g. Olson and Hansson (2010) on the impact of geography on institutions.
[20] The G-Econ data base also contains data on GDP per capita for 1990, 1995 and 2000. Tables A4-A6 report the results for these years.

fact, the "regional analysis" corroborates the results from the pure cross country analysis in suggesting a detrimental impact from UV on prosperity.

The results differ, however, in one important respect: the economic size of the impact from UV. As apparent from column 4, when UV is increased by one percent GDP per capita drops by 0.16 %, a considerably smaller effect than the 0.98% obtained in the cross country analysis (cf. Table 2). Another way to see the difference is by noticing that a one standard deviation reduction in UV (roughly 0.85 log points) implies an increase in GDP per capita of about 15% (= exp(0.85*0.16)); down from about 60% in the pure cross-country analysis.

What should we make of this change in results? An obvious interpretation is that the cross-country analysis might be tainted by omitted variable bias; apparently these omitted variables works to elevate the economic significance of UV. If this interpretation is correct, the results from Table 7 are more likely to convey accurate information about the causal influence from eye disease on long-term development than the results from Tables 1 and 2.

Another interpretation, however, would suggest that the results from Table 7 are underestimating the impact from eye disease. Migration may be a bigger issue in the context of the present analysis, compared to the cross-country exercise. If individuals tend to migrate to regions with higher productivity, which could be caused by less UVR in the first place, this will reduce interregional income variation thereby tempering the impact from UV. In practice of course, both omitted variables and migration may be contributing to the reduction in the estimate for UV.

The conservative conclusion from the analysis would be to assume the former interpretation is more important, which implies that an elasticity around 0.2 (rather than around one) is a more plausible estimate for the impact of UV on prosperity. This remains a very substantial impact however. As noted above, the simulation study by Ashraf et al. (2008) find that an increase in life expectancy by about 20 years eventually leads to an increase in GDP per capita which is quite similar to what a reduction in one standard deviation in UV produces, judged from the results in Table 7. In this respect

the within country estimates reinforces the overall conclusion that historical eye disease incidence has had a powerful impact on contemporary cross-country income differences.

6 Conclusion

The present study examines the hypothesis that eye disease has had an important effect on the long-run development process. Drawing on research from the field of epidemiology we have proposed to capture the historical incidence of eye disease, cataract in particular, by UV radiation.

Our key result is that UV radiation hold strong explanatory power vis-à-vis contemporary income per capita differences. The link between UV radiation and living standards is robust to a rigorous set of controls. We also show that while UV radiation does predict cataract, it seems unrelated to other diseases which flourishes in tropical areas like malaria or hookworm.

The sizeable point estimate we recover 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 fertility transition and thus the take-off to sustained growth, by influencing the return to skill accumulation. Hence, we argue the UV estimate reflects the ramifications of a differential timing of the take-off related to the historical incidence of eye disease.

We find support for this interpretation by showing that the impact of UV rises over time in a cross-country setting, ultimately emerging as a strong determinant of contemporary income differences during the 20th century. In addition, we also find a strong link between UVB-R and the timing of the fertility transition, a theoretically founded marker for the take-off to sustained growth. Interestingly, our point estimate for the impact of UVB-R on the timing of the fertility transition goes a long way in accounting for our estimated impact of UVB-R on contemporary labor productivity. The bottom line seems to be that the historical incidence of eye disease was an important

determinant of the diffusion of the industrial revolution and therefore of contemporary income differences.

DATA APPENDIX

Main variables

A. Biological damage due to exposure to UV radiation

NASA produces a daily, satellite-based index for erythemal ultraviolet exposure (EUVE), which is an estimate of the biological damage that ultraviolet irradiance causes to people. The index is a measure of the integrated amount of energy from exposure to UV radiation over a day, within a certain area, normalized to units that relate the biological response to this radiation.[21] The index is expressed in units of biological damage per sq km, which relates the biological response (erythema) to the incident energy, and which can be interpreted as an index of the *potential for biological damage due to solar irradiation*.

In this paper, we rely on data for EUVE daily local-noon irradiances for 1990 and 2000, and produce average yearly EUVE levels for each country. The variable *UV radiation* reported in our tables corresponds to the EUVE average for both years.

The raw UV data and units are described at http://jwocky.gsfc.nasa.gov/datainfo/1README.UV. The data are 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.

B. Cataract incidence

The World Health Organization (WHO) quantifies the burden of a specific 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 an estimate 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. 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.

Data from WHO (2008), available at

http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html.

[21] Specifically, the index is an estimate of the integrated ultraviolet (UV) irradiance (which controls for the influence of column ozone amount and cloud conditions on each day), calculated using a model for the susceptibility of Caucasian skin to sunburn (erythema).

C. Labor productivity and income per capita.
Real GDP per worker (constant prices: Chain series). Source: Penn World Tables 6.3.

Real GDP per capita (PPP). Source: World Bank.

D. Year of 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*".

E. Human capital.
Years of schooling in 2010. Source: Barro and Lee (2010), "A New Data Set of Educational Attainment in the World, 1950-2010."

Control variables

1. **Geography:**
   - Continent dummies (Africa, Asia, North America, South America, Europe, Oceania).
   - Latitude. Source: Nunn and Puga, 2010.
   - Elevation mean (average of elevation extremes). Source: CIA Factbook. Data available at http://www.nationmaster.com.
   - Mean distance to coast or rivers. Source: Gallup, Sachs and Mellinger, 1999.

2. **Climate:**
   - Area-weighted, mean air temperature and total precipitation. Constructed from the GECON 3.4 dataset. Data available at http://gecon.yale.edu/.

3. **Pre-industrial history:**
   - Time passed since the Neolithic revolution. Source: Putterman, 2006.

Other variables
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 skin cancer (melanoma and other skin carcinomas).
DALY rates for infectious, parasitic, tropical-clustered diseases that have been studied before:

- HIV/AIDS
- Malaria
- Hookworm disease.

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Table 1
Real GDP per worker, cataract incidence, and biological damage due to exposure to UV radiation

	1	2	3	4	5	6	7
Dependent variable:				**(log) Real GDP per worker, 2004**			
(log) UV damage	-1.11***	-0.72***	-0.88***	-1.19***	-1.24***	-0.80**	
	[0.11]	[0.19]	[0.15]	[0.20]	[0.11]	[0.37]	
(log) Cataract prevalence							-0.28***
							[0.064]
1[Continent = Africa]		-1.03***				-0.88*	-0.66
		[0.31]				[0.45]	[0.42]
1[Continent = Asia]		-0.1				0.12	0.24
		[0.26]				[0.30]	[0.25]
1[Continent = Oceania]		-0.17				-0.46	-1.26
		[0.40]				[1.03]	[0.83]
1[Continent = North America]		0.15				0.25	0.092
		[0.29]				[0.44]	[0.38]
1[Continent = South America]		-0.078				0.38	0.33
		[0.29]				[0.37]	[0.31]
(log) Latitude			0.16			0.012	-0.0024
			[0.11]			[0.13]	[0.13]
Mean elevation ('000 m)			-0.12			-0.093	-0.19**
			[0.073]			[0.099]	[0.077]
(area weitghted average 1990-2008) Temperature				0.000061		-0.0062	-0.025
				[0.016]		[0.024]	[0.016]
(area weitghted average 1990-2008) Precipitation				0.0027		-0.31	-0.33*
				[0.14]		[0.20]	[0.19]
(log) Country area					0.07	0.075	0.099*
					[0.054]	[0.061]	[0.053]
Distance to coast (km)					-0.93***	-0.85***	-0.88***
					[0.15]	[0.19]	[0.19]
Distance to rivers (km)					0.17**	0.14	0.17
					[0.074]	[0.12]	[0.12]
Year of Neolithic Transition ('000 years)					0.090***	-0.0055	-0.022
					[0.030]	[0.059]	[0.055]
Observations (countries)	170	170	168	157	148	146	146
R-squared	0.28	0.42	0.3	0.32	0.48	0.58	0.6
Number of controls	0	5	2	2	4	13	13
Joint significance of control variable (p-values for the H0: all regressors (except UV damage) are jointly insignificant):							
Continent dummies		0.00				0.00	0.00
Latitude and elevation			0.09			0.61	0.04
Temperature and precipitation				1.00		0.28	0.06
Distance to coast, rivers; timing of Neolithic transition					0.00	0.00	0.00
All controls						0.00	0.00

Notes: OLS regressions. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. 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 between 15 and 59), estimated by WHO (2004). All regressions include a constant term. Europe excluded from the set of continent dummies. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table 2
Real GDP per capita, cataract incidence, and biological damage due to exposure to UV radiation

	1	2	3	4	5	6	7
Dependent variable:			**(log) Real GDP per capita, 2004**				
(log) UV damage	-1.23***	-0.83***	-1.04***	-1.31***	-1.39***	-0.96**	
	[0.11]	[0.20]	[0.16]	[0.21]	[0.12]	[0.39]	
(log) Cataract prevalence							-0.32***
							[0.071]
1[Continent = Africa]		-1.10***				-1.07**	-0.83*
		[0.32]				[0.48]	[0.45]
1[Continent = Asia]		-0.11				0.13	0.25
		[0.27]				[0.31]	[0.25]
1[Continent = Oceania]		-0.16				-0.55	-1.50*
		[0.42]				[1.07]	[0.86]
1[Continent = North America]		0.18				0.076	-0.12
		[0.30]				[0.47]	[0.41]
1[Continent = South America]		-0.024				0.35	0.28
		[0.29]				[0.39]	[0.32]
(log) Latitude			0.14			-0.017	-0.033
			[0.11]			[0.13]	[0.13]
Mean elevation ('000 m)			-0.13*			-0.084	-0.20**
			[0.078]			[0.10]	[0.081]
(area weitghted average 1990-2008) Temperature				-0.0027		-0.0045	-0.028
				[0.018]		[0.026]	[0.017]
(area weitghted average 1990-2008) Precipitation				0.067		-0.31	-0.33
				[0.15]		[0.21]	[0.20]
(log) Country area					0.06	0.06	0.089
					[0.057]	[0.066]	[0.056]
Distance to coast (km)					-0.95***	-0.86***	-0.89***
					[0.16]	[0.19]	[0.20]
Distance to rivers (km)					0.18**	0.15	0.18
					[0.079]	[0.13]	[0.13]
Year of Neolithic Transition ('000 years)					0.078**	-0.041	-0.06
					[0.033]	[0.062]	[0.056]
Constant	15.1***	13.4***	13.9***	15.4***	14.9***	14.1***	11.1***
	[0.56]	[0.85]	[1.05]	[0.85]	[0.86]	[2.08]	[0.99]
Observations (countries)	170	170	168	157	148	146	146
R-squared	0.31	0.45	0.32	0.36	0.5	0.6	0.62
Number of controls	0	5	2	2	4	13	13
Joint significance of control variable (p-values for the H0: all regressors (except UV damage) are jointly insignificant):							
Continent dummies		0.00				0.00	0.00
Latitude and elevation			0.13			0.72	0.04
Temperature and precipitation				0.90		0.32	0.05
Distance to coast, rivers; timing of Neolithic transition					0.00	0.00	0.00
All controls						0.00	0.00

Notes: OLS regressions. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. 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 between 15 and 59), estimated by WHO (2004). All regressions include a constant term. Europe excluded from the set of continent dummies. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table 3
Cataract incidence and biological damage due to UV exposure

	1	2	3	4	5	6
Dependent variable:			**(log) Cataract incidence**			
(log) UV damage	2.14***	1.21***	1.94***	1.73***	2.28***	0.80*
	[0.13]	[0.23]	[0.17]	[0.23]	[0.15]	[0.43]
1[Continent = Africa]		2.09***				1.81***
		[0.35]				[0.47]
1[Continent = Asia]		1.18***				1.25***
		[0.35]				[0.41]
1[Continent = Oceania]		-0.29				-1.39
		[0.48]				[1.45]
1[Continent = North America]		0.41				0.22
		[0.37]				[0.51]
1[Continent = South America]		0.77**				0.70
		[0.34]				[0.45]
(log) Latitude			-0.17**			-0.094
			[0.078]			[0.087]
Mean elevation ('000 m)			-0.092			-0.015
			[0.071]			[0.13]
(area weitghted average 1980-2008) Temperature				0.047***		0.029
				[0.015]		[0.029]
(area weitghted average 1980-2008) Precipitation				-0.40***		-0.14
				[0.094]		[0.16]
(log) Country area					-0.0013	-0.023
					[0.057]	[0.055]
Distance to coast (km)					0.49**	-0.07
					[0.20]	[0.21]
Distance to rivers (km)					-0.22***	0.012
					[0.082]	[0.14]
Year of Neolithic Transition ('000 years)					-0.044	-0.054
					[0.043]	[0.067]
Observations (countries)	170	170	168	157	148	146
R-squared	0.59	0.79	0.6	0.65	0.64	0.80
Number of controls	0	5	2	2	4	13
Joint significance of control variable (p-values for the H0: all regressors (except UV damage) are jointly insignificant):						
Continent dummies		0.00				0.00
Latitude and elevation			0.04			0.56
Temperature and precipitation				0.00		0.48
Distance to coast, rivers; timing of Neolithic transition					0.01	0.91
All controls						0.00

Notes: OLS regressions. 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 between 15 and 59), estimated by WHO (2004). UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. All regressions include a constant term. Europe excluded from the set of continent dummies. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table 4
Historical real GDP per capita (Maddison data) and biological damage due to exposure to UV radiation

	1	2	3	4	5
Dependent variable:	**Real GDP per capita in:**				
	1700	**1820**	**1900**	**1950**	**1950[a]**
(log) UV damage	-0.31	-0.27	-0.52	-1.16***	-0.89*
	[0.22]	[0.23]	[0.44]	[0.38]	[0.46]
Observations (countries)	21	40	40	110	40
R-squared	0.89	0.84	0.71	0.62	0.77
Number of controls	11	12	12	13	12

Notes: OLS regressions. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. All regressions include continent dummies, controls for latitude, elevation, temperature, precupitation, distance to coast and rivers, country area, timing of the neolithic revolution, and a constant term. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.
a: Regression with sample constrained to be the same as in year 1900 (column 3).

Table 5
Year of the fertility decline and biological damage due to exposure to UV radiation

	1	2	3	4	5	6	7	8
Dependent variable:	**Year of the fertility decline**						**(log) Real GDP per worker, 2004**	**(log) Real GDP per capita, 2004**
(log) UV damage	48.8***	28.8***	46.9***	53.4***	49.3***	23.9**		
	[3.70]	[5.17]	[4.71]	[6.60]	[3.93]	[11.7]		
Year of the fertility decline							-0.018***	-0.020***
							[0.0057]	[0.0060]
1[Continent = Africa]		40.4***				44.6***	-0.26	-0.40
		[7.49]				[10.2]	[0.53]	[0.56]
1[Continent = Asia]		32.4***				30.3***	0.38	0.40
		[6.46]				[7.75]	[0.35]	[0.36]
1[Continent = North America]		17.9**				22.3**	0.48	0.31
		[7.66]				[9.04]	[0.43]	[0.45]
1[Continent = South America]		13.3				14.2	0.40	0.34
		[11.1]				[12.3]	[0.43]	[0.45]
(log) Latitude			-1.37			-0.86	-0.023	-0.051
			[2.12]			[2.25]	[0.16]	[0.16]
Mean elevation ('000 m)			-0.07			0.18	-0.18*	-0.18*
			[1.83]			[3.60]	[0.099]	[0.10]
(area weitghted average 1980-2008) Temperature				-0.13		0.23	-0.029	-0.033
				[0.38]		[0.71]	[0.020]	[0.021]
(area weitghted average 1980-2008) Precipitation				-4.45**		4.34	-0.17	-0.15
				[1.76]		[3.86]	[0.22]	[0.23]
(log) Country area					-1.15	-0.66	0.094	0.084
					[1.05]	[0.97]	[0.059]	[0.061]
Distance to coast (km)					15.0***	8.55**	-0.75***	-0.74***
					[3.78]	[4.05]	[0.23]	[0.24]
Distance to rivers (km)					2.3	0.29	0.14	0.14
					[2.34]	[3.04]	[0.19]	[0.19]
Year of Neolithic Transition ('000 years)					0.3	1.7	0.044	0.012
					[0.75]	[1.34]	[0.062]	[0.063]
Observations (countries)	131	131	129	125	122	120	120	120
R-squared	0.61	0.75	0.62	0.64	0.67	0.78	0.63	0.65
Number of controls	0	4	2	2	4	12	12	12
Joint significance of control variable (p-values for the H0: all regressors (except UV damage) are jointly insignificant):								
Continent dummies		0.00				0.00	0.07	0.06
Latitude and elevation			0.81			0.93	0.19	0.21
Temperature and precipitation				0.04		0.48	0.26	0.22
Distance to coast, rivers; timing of Neolithic transition					0.00	0.20	0.03	0.05
All controls						0.00	0.00	0.00

Notes: OLS regressions. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. 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 between 15 and 59), estimated by WHO (2004). All regressions include a constant term. Europe and Oceania excluded from the set of continent dummies. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table 6
Placebo regressions

	1	2	3	4	5	6	7	8	9	10	11
		Other eye diseases				**Other diseases**			**Infectious, parasitic, tropical-clustered diseases**		
Dependent variable: (log)	**Cataract**	Trachoma	Onchocer-chiasis	Glaucoma	Referactive errors	Hear loss	All sense organ diseases	Skin cancer	HIV/AIDS	Malaria	Hookworm
(log) UV damage	0.80*	0.09	-0.40	0.23	-0.20	-0.17	-0.06	0.10	0.41	-0.11	0.60
	[0.43]	[0.64]	[0.43]	[0.14]	[0.13]	[0.10]	[0.12]	[0.32]	[0.74]	[0.52]	[0.40]
Observations (countries)	146	146	146	146	146	146	146	146	146	146	146
R-squared	0.80	0.52	0.45	0.84	0.57	0.74	0.79	0.63	0.70	0.80	0.84
Number of controls	13	13	13	13	13	13	13	13	13	13	13

Notes: OLS regressions. Incidence of all diseases is measured as the number of years lost due to disability, for incident cases of each disease (expressed as a rate per 100,000 people between 15 and 59), estimated by WHO (2004). UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. All regressions include a constant term, and control for continent dummies, latitude, elevation, temperature, precipitation, distance to coast and rivers, and the year of the Neolithic revolution. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table 7
Real product per capita (2005) and biological damage due to exposure to UV radiation

	1	2	3	4
Dependent variable:	**(log) Real product per capita by geographic cell (1x1 degrees), 2005**			
(log) UV damage	-0.30**	-0.35***	-0.38***	-0.16**
	[0.15]	[0.09]	[0.14]	[0.07]
(log) Latitude	0.19**			0.18*
	[0.09]			[0.1]
(log) Elevation	-0.03**			-0.07
	[0.01]			[0.04]
Temperature		0.00		-0.01
		[0.01]		[0.01]
Precipitation		-0.03		0.01
		[0.03]		[0.03]
(log) Area size			0.02**	0.03**
			[0.01]	[0.01]
Distance to ocean			-0.05	-0.05*
			[0.03]	[0.03]
Distance to major navigable river			0.03	0.02
			[0.04]	[0.04]
Observations	16,978	17,083	17,056	16,953
R-squared	0.93	0.93	0.93	0.93
Country fixed effects	Yes	Yes	Yes	Yes
Number of countries (clusters)	162	162	159	159

Notes: OLS regressions. Each variable observation is for a geographic cell of 1 degree latitude by 1 degree longitude. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. Real product and control variables from the GECON 3.4 dataset, Yale University. All regressions include a constant term. Robust standard errors, clustered at the country level, reported in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table A1
Summary statistics

	Obs	Mean	Std. Dev.	Min	Max
A. Cross country data					
Real GDP per worker, 2004	170	24,726	24,423	934	118,730
Real GDP per capita, 2004	160	11,622	12,961	354	70,788
UV damage, av. 1990, 2000	170	201.0	77.1	31.8	298.5
1[Continent = Africa]	170	0.31	0.46	0	1
1[Continent = Asia]	170	0.25	0.43	0	1
1[Continent = Oceania]	170	0.05	0.21	0	1
1[Continent = North America]	170	0.11	0.32	0	1
1[Continent = South America]	170	0.07	0.26	0	1
Latitude (degrees)	170	18.8	24.8	-41.8	65.0
Elevation mean (km)	168	1.4	1.0	0.0	4.3
Temperature (area weighted 80-08, C degrees)	157	17.6	8.3	-4.5	28.9
Precipitation (area weighted av 80-08, '000 mm)	157	1.0	0.7	0.0	3.3
Country area ('000 sq km)	157	0.83	2.09	0.00	17.20
Distance to coast (km)	153	0.40	0.46	0.01	2.37
Distance to rivers (km)	153	1.00	1.09	0.02	9.41
Year of Neolithic Transition ('000 years)	153	4.8	2.4	0.4	10.5
B. Geo gridded data (1x1 degree lat lon)					
Real gross cell product per capita, 1990 ('000 USD)	18,527	12.2	23.4	0.00	45.9*
Real gross cell product per capita, 1995 ('000 USD)	17,341	10.8	24.8	0.18	39.6*
Real gross cell product per capita, 2000 ('000 USD)	17,379	11.8	25.4	0.00	43.7*
Real gross cell product per capita, 2005 ('000 USD)	17,108	13.8	26.2	0.00	52.3*
UV damage, av. 1990, 2000	19,099	149.8	95.1	8.5	428.6
Latitude (degrees)	19,105	31.4	31.8	-56	83
Elevation (m above sea level)**	19,105	690.8	803.3	3.9	6,350
Temperature (average 1980-2008, C degrees)	19,105	9.3	14.7	-33.0	30.9
Precipitation (av 1980-2008, '000 mm)	19,105	0.72	0.70	0.01	6.86
Area (sq km)	19,105	6,995	3,711	0.9	12,415
Distance to ocean (km)	19,077	0.8	0.7	0.0	2.98
Distance to major river (km)	19,074	1.7	1.3	0.0	9.99

Notes. *: 99th percentile reported, instead of maximum value.
**: Elevation + 50 m reported (transformation to take log values).

Table A2
Correlates of biological damage due to exposure to UV radiation - Cross country data

	1	2	3	4	5
Dependent variable:			**(log) UV damage**		
1[Continent = Africa]	1.19***				0.48***
	[0.068]				[0.074]
1[Continent = Asia]	0.94***				0.40***
	[0.081]				[0.064]
1[Continent = Oceania]	1.09***				0.71***
	[0.11]				[0.15]
1[Continent = North America]	1.05***				0.38***
	[0.12]				[0.092]
1[Continent = South America]	1.10***				0.42***
	[0.089]				[0.074]
(log) Latitude		-0.36***			-0.021
		[0.046]			[0.019]
Mean elevation ('000 m)		0.031			0.16***
		[0.032]			[0.027]
(area weitghted average 1980-2008) Temperature			0.056***		0.046***
			[0.0039]		[0.0047]
(area weitghted average 1980-2008) Precipitation			0.028		-0.029
			[0.029]		[0.026]
(log) Country area				0.028	-0.052***
				[0.027]	[0.012]
Distance to coast (km)				-0.18	0.011
				[0.12]	[0.043]
Distance to rivers (km)				0.071	-0.038**
				[0.073]	[0.019]
Year of Neolithic Transition ('000 years)				-0.065***	0.0022
				[0.014]	[0.0087]
Observations (countries)	170	168	157	148	146
R-squared	0.71	0.46	0.72	0.14	0.93

Notes: OLS regressions. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. All regressions include a constant term. Europe excluded from the set of continent dummies. Robust standard errors in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table A3
Correlates of biological damage due to exposure to UV radiation - Geo gridded data (1x1 degree lat lon)

	1	2	3	4
Dependent variable:		**(log) UV damage**		
(log) Latitude	-0.43**			-0.15**
	[0.18]			[0.06]
(log) Elevation	0.12***			0.18***
	[0.02]			[0.02]
Temperature		0.03***		0.03***
		[0.01]		[0.01]
Precipitation		-0.03		-0.07***
		[0.03]		[0.02]
(log) Area size			0.04***	0.01**
			[0.01]	[0.00]
Distance to ocean			0.06	0.06
			[0.04]	[0.04]
Distance to major navigable river			-0.30***	-0.15***
			[0.07]	[0.04]
Observations	18,989	19,099	19,068	18,960
R-squared	0.89	0.90	0.90	0.95
Country fixed effects	Yes	Yes	Yes	Yes
Number of countries (clusters)	189	190	186	185

Notes: OLS regressions. Each variable observation is for a geographic cell of 1 degree latitude by 1 degree longitude. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. All control variables from the GECON 3.4 dataset, Yale University. All regressions include a constant term. Robust standard errors, clustered at the country level, reported in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table A4
Real product per capita (1990) and biological damage due to exposure to UV radiation

	1	2	3	4
Dependent variable:	**(log) Real product per capita by geographic cell (1x1 degrees), 1990**			
(log) UV damage	-0.36**	-0.32***	-0.45***	-0.21***
	-0.14	-0.08	-0.16	-0.06
(log) Latitude	0.12**			0.12**
	-0.05			-0.06
(log) Elevation	-0.01			-0.06
	-0.01			-0.04
Temperature		-0.01		-0.02
		-0.01		-0.01
Precipitation		0.01		0.03
		-0.03		-0.04
(log) Area size			0.03*	0.03*
			-0.01	-0.02
Distance to ocean			-0.03	-0.05
			-0.05	-0.03
Distance to major navigable river			-0.01	-0.02
			-0.05	-0.04
Observations	18,404	18,514	18,485	18,377
R-squared	0.94	0.94	0.94	0.94
Country fixed effects	Yes	Yes	Yes	Yes
Number of countries (clusters)	182	183	179	178

Notes: OLS regressions. Each variable observation is for a geographic cell of 1 degree latitude by 1 degree longitude. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. Real product and control variables from the GECON 3.4 dataset, Yale University. All regressions include a constant term. Robust standard errors, clustered at the country level, reported in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table A5
Real product per capita (1995) and biological damage due to exposure to UV radiation

	1	2	3	4
Dependent variable:	**(log) Real product per capita by geographic cell (1x1 degrees), 1995**			
(log) UV damage	-0.32**	-0.35***	-0.44***	-0.21***
	[0.15]	[0.09]	[0.15]	[0.07]
(log) Latitude	0.16**			0.16*
	[0.07]			[0.08]
(log) Elevation	-0.02			-0.06
	[0.02]			[0.04]
Temperature		0.00		-0.01
		[0.01]		[0.01]
Precipitation		-0.01		0.02
		[0.03]		[0.03]
(log) Area size			0.03**	0.03*
			[0.01]	[0.02]
Distance to ocean			-0.02	-0.03
			[0.04]	[0.03]
Distance to major navigable river			-0.01	-0.02
			[0.03]	[0.03]
Observations	17,230	17,335	17,307	17,204
R-squared	0.93	0.93	0.93	0.93
Country fixed effects	Yes	Yes	Yes	Yes
Number of countries (clusters)	163	163	159	159

Notes: OLS regressions. Each variable observation is for a geographic cell of 1 degree latitude by 1 degree longitude. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. Real product and control variables from the GECON 3.4 dataset, Yale University. All regressions include a constant term. Robust standard errors, clustered at the country level, reported in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.

Table A6
Real product per capita (2000) and biological damage due to exposure to UV radiation

	1	2	3	4
Dependent variable:	**(log) Real product per capita by geographic cell (1x1 degrees), 2000**			
(log) UV damage	-0.28*	-0.30***	-0.38**	-0.15**
	[0.15]	[0.09]	[0.16]	[0.08]
(log) Latitude	0.17**			0.16*
	[0.08]			[0.09]
(log) Elevation	-0.02*			-0.06
	[0.01]			[0.04]
Temperature		-0.01		-0.01
		[0.01]		[0.01]
Precipitation		-0.02		0.02
		[0.03]		[0.03]
(log) Area size			0.02**	0.03*
			[0.01]	[0.01]
Distance to ocean			-0.03	-0.03
			[0.03]	[0.03]
Distance to major navigable river			0.00	-0.01
			[0.04]	[0.04]
Observations	17,265	17,370	17,342	17,239
R-squared	0.93	0.93	0.93	0.93
Country fixed effects	Yes	Yes	Yes	Yes
Number of countries (clusters)	165	165	161	161

Notes: OLS regressions. Each variable observation is for a geographic cell of 1 degree latitude by 1 degree longitude. UV damage is an index of Erythemal exposure, constructed as the daily average 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 UV exposure daily data for 1990 and 2000 produced by NASA. Real product and control variables from the GECON 3.4 dataset, Yale University. All regressions include a constant term. Robust standard errors, clustered at the country level, reported in brackets. ***, ** and * denote statistical significance at 1, 5 and 10% levels, respectively.