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Root Causes of African Underdevelopment

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The Arndt-Corden Division of Economics
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Abstract

What are the root causes of Africa's current state of under-development? Is it the long history of slave trade, or the legacy of extractive colonial institutions, or the fallout of malaria? We investigate the relative contributions of these factors using an instrumental variable approach. The results show that malaria matters the most and all other factors are statistically insignificant. Malaria also negatively affects savings. Using a two period overlapping generation model we show that malaria impacts economic performance by increasing both mortality and morbidity. Increased mortality increases current household consumption and discourages savings. Increased morbidity adversely affects labour productivity. The combined impact is a slowdown of capital accumulation and economic growth.

Keywords: Malaria; Colonial Institutions; Slave Trade; Economic Development

JEL classification: O11; O41; O57; N0

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1. Introduction

It is well known that Africa is falling behind the rest of the world in terms of economic wellbeing. Even though global poverty is on the decline due to rapid economic growth in India, China, and other parts of the world, Africa's contribution to this decline is disappointing. Absolute poverty in many of the African nations is in fact rising (Sachs, 2005). What is the fundamental cause behind this decline? This has been a topic of research for a few decades now. Even though it is extremely difficult to summarize this voluminous literature, it is perhaps fair to say that three strands of thoughts stand out.

The first is the disease view. According to this view, malaria and other infectious diseases have fatal as well as debilitating effects on the human population in Africa. It negatively influences productivity, savings, and investments in physical and human capital and directly affects economic performance of the continent (Gallup and Sachs 2001; Bloom and Sachs 1998). According to Bloom and Sachs (1998), the high incidence of malaria in sub-Saharan Africa reduces the annual growth rate of the continent by 1.3 percentage points a year and eradication of malaria in the 1950s would have resulted into a doubling of per capita income. Sachs (2003) and Carstensen and Gundlach (2006) using a cross-national dataset (which includes African as well as non-African nations) and Lorentzen et al. (2008) using cross-national and sub-national datasets also make similar arguments about the role of diseases. Lorentzen et al. (2008) in particular argue that higher adult mortality is associated with increased level of risky behaviour, higher fertility and lower investment in physical and human capital. Acemoglu and Johnson (2006) however question these results. They find that there is no statistically significant effect of improved life expectancy on total GDP leading them to conclude that diseases do not have a direct role in development.

Despite the doubts posed by Acemoglu and Johnson (2006), a significant number of recent studies tend to support the disease view both at the macro as well as micro level. Weil (2005) and Bloom and Canning (2005) calibrating the effects of health from a range of micro estimates into a macro model show that these effects are important at the aggregate level. Kalemli-Ozcan et al. (2000) and Kalemli-Ozcan (2002) also show that lower mortality as a result of better health contributes to economic growth. In a related literature, Arndt and Lewis (2000), Bell et al. (2003), and Kalemli-Ozcan (2006) find that HIV/AIDS is reversing the trends in demographic transition in Africa and is negatively affecting growth.[1] At the micro level, Knaul (2000), Behrman and Rosenzweig (2004), Bleakley (2003), Miguel and Kremer (2004), Schultz (2002), and many others find that improved health leads to better individual economic outcomes.[2]

The second is the colonial institutions view. According to this view, the persistent effect of colonial institutions can explain the huge differences in income across all ex-colonies including Africa (Knack and Keefer, 1995; Hall and Jones, 1999; Acemoglu et al., 2001; Rodrik et al., 2004; Bhattacharyya, 2004; Nunn, 2007). The story as outlined by Acemoglu et al. (2001) goes as follows. Europeans resorted to different style of colonisation depending on the feasibility of settlement. In a tropical environment the settlers had to deal with killer malaria and hence a high mortality rate. This prevented colonisers from settling in a tropical environment and they erected extractive institutions in these colonies. These colonial institutions have persisted over time and they continue to influence the economic performance of the colonies even long after independence. Hence, the Acemoglu et al. (2001) argument is that diseases affect economic performance only indirectly through institutions. Nunn (2007) using a stylised model for Africa

[1] For an alternative view, see Young (2005) who use a calibrated simulation for South Africa to forecast that survivors of the AIDS epidemic will be economically better off than they would have been without the epidemic. The intuition in Young's model is that women become more cautious about sex due to the fear of infection. As others die out, female labour becomes more valuable and a consequent reduction in fertility leads to higher standards of living.

show that colonial extraction when severe enough can cause a society to move from a high to low production level equilibrium. Due to the stability of low level equilibrium, a society can remain trapped in this equilibrium even after the period of colonial extraction is over.

Earlier work by Easterly and Levine (1997), Sachs and Warner (1997), and Temple (1998) also reports strong link between quality of institutions and post-war growth (or the lack of it) in Africa.[3] Easterly and Levine (1997) show that ethnic diversity in Africa has led to social polarisation and the formation of several rival interest groups which increase the likelihood of selecting socially sub-optimal policies when an ethnic representative in the government fail to internalise the entire social cost of their rent seeking policies. Sachs and Warner (1997), on the other hand, stress on Africa's lack of openness to international markets and unfavourable geography as other contributors to poor growth in addition to poor quality institutions. Temple (1998) emphasizes the role of social arrangements in explaining Africa's slow growth.

Finally, a third group of explanation relates to the economic impact of Africa's engagement in slave trade. According to this view, Africa's engagement in the slave trade caused massive depopulation of the continent over two centuries. This resulted into an implosion of the continent's production possibility frontier and an unambiguous reduction in welfare. The secular decline in welfare continued over more than two centuries plunging the continent into economic backwardness (see Gemery and Hogendorn, 1979; Inikori, 1992; and Manning, 1981). In a recent paper, Nunn (2008) also reports a negative causal relationship between slave trade and current economic performance in Africa. He shows that the data is consistent with historical accounts suggesting that slave trade led to ethnic fractionalisation and weakening of political structures and hence persistently poor economic performance.

[2] Acemoglu and Johnson (2006) argue that their results are not comparable with the micro studies as the micro studies do not incorporate general equilibrium effects.

These theories, even though plausible, do not tell us how much of the variation in income across countries in Africa they can explain. One possible way to arrive at an answer is to check the relative strengths of these theories in explaining the variation when they are pitted against each other in a regression model. In this paper we investigate their relative strength by setting up a parsimonious regression model. In the regression model we use log GDP per capita in 2000 as the dependent variable and malaria risk, institutions, and log total slave exports out of Africa normalised by land area as explanatory variables. We deal with the complex causality issues involved with this strategy by using appropriate exogenous instruments for malaria risk, institutions, and total slave exports. The results show that malaria matters the most and all other factors are statistically insignificant. We explain the mechanism through which malaria impacts African development in two stages. First, we show that there is strong negative conditional correlation between malaria and savings which indicates malaria dampens savings. Second, by using a two period overlapping generation model we show that this negative relationship can be due to the increase in both mortality and morbidity. Increased mortality resulting from malaria induces households to increase current consumption and save less for the future. Increased morbidity on the other hand adversely affects productivity reducing household income and savings. This slows down capital accumulation and economic development. This discussion perhaps sheds some light on why malaria is so persistent in Africa.

Our paper is most closely related to the recent contributions by Sachs (2003), Carstensen and Gundlach (2006), and Nunn (2008). Sachs (2003) and Carstensen and Gundlach (2006) using a parsimonious model and a much larger cross-national sample (which includes both African as well as non-African nations) report direct effects of malaria prevalence on income. They also find evidence of positive effects of institutions on income. Nunn (2008) in an Africa specific study

[3] See Collier and Gunning (1999) for a survey of this literature.

show that the relationship between slave trade and current economic performance of the continent is negative and indeed causal. The major difference between Sachs (2003), Carstensen and Gundlach (2006) and our work is that we solely focus on Africa. Hence we do treat slave trade as an important explanatory variable in our model, which may not be relevant for their respective models. Furthermore, our findings with regards to institutions also differ significantly from theirs as we fail to find any statistically significant effects of institutions in Africa. Our findings are in sharp contrast to those of Nunn (2008) though. We observe that the statistical significance of the slave trade variable disappears once we include malaria and this finding is statistically robust.

Our findings are related to the literature on health and economic development (see Kalemli-Ozcan, 2002; Behrman and Rosenzweig, 2004; Bleakley, 2003; Miguel and Kremer, 2004; Weil, 2005; Acemoglu and Johnson, 2006; Lorentzen et al., 2008; and many others) to the extent that it supports the disease and development view. Results however are not comparable since there are significant differences in scale (micro or macro), approach (general equilibrium or partial equilibrium) and nature (empirical or theoretical) of these studies. Furthermore, although suggestive of the importance of diseases, some of the results related to the present day impact of HIV/AIDS in Africa may not be directly comparable with our study as we focus on estimating the causal effects of malaria.

Our analysis proceeds in four stages. In section 2, we introduce the empirical model and briefly discuss the data. We also discuss the complex causality issues associated with a study of this nature and the instrumental variable (IV) approach. In section 3, we present the empirical results. We compare our findings with Nunn (2008), Sachs (2003), and Carstensen and Gundlach (2006). These studies are the most closely related to ours. Section 4 asks the question why malaria is so persistent in Africa. In other words, what are the channels though which malaria

affects income? We empirically identify savings as an important channel. This is also demonstrated by a two period overlapping generation model. Section 5 concludes.

2. Specification and Data

In order to estimate the causal effects of malaria, colonial institutions, and slave trade on Africa's long-run economic development, we follow the literature[4] and estimate the following model.

$$\log y_i = \lambda + \alpha \mathrm{MAL}_i + \beta \mathrm{INS}_i + \gamma \mathrm{SLVX}_i + \varepsilon_i \qquad (1)$$

where y_i, MAL_i, INS_i, and SLVX_i are per capita income in country i, measure of malaria, measure of institutions, and measure of slave exports respectively. ε_i is the random error term. We are interested in the size, sign, and significance of the three coefficients $\alpha, \beta,$ and γ.

The estimation of equation (1) is based on a dataset consisting of per capita GDP levels, measure of malaria risk, measure of institutions, and measure of slave exports in (up to) 52 countries in Africa. Definition and source of all the variables used in this study is summarised in the Data Appendix. Table 1 presents summary statistics for the key variables of interest.

GDP per capita in 2000 data is from the Penn World Table 6.1. According to these figures, Tanzania is the poorest country in Africa in 2000.

Malaria risk is the percentage of population living in areas of high malaria risk in a country in 1994. It is calculated using GIS software from a digitised WHO map of the world distribution of malaria and a detailed database of world population distribution in 1994.[5] The variable lies between 0 and 1 and a higher value indicates greater risk for the population. Most of the countries in the sample registers high malaria incidence except Algeria, Tunisia and Egypt.

[4] See Acemoglu et al. (2001), Rodrik et al. (2004), Sachs (2003), Carstensen and Gundlach (2006), Nunn (2008), and many others who use similar models.

[5] For more details see http://www.earth.columbia.edu/articles/view/1932

There are at least three measures of institutional quality that has been used in the literature. Knack and Keefer (1995), Acemoglu et al. (2001), and many others use expropriation risk averaged over 1985 to 1995 from the Political Risk Services. Rodrik et al. (2004) use the rule of law index from the World Bank. Others use the executive constraint from the Polity dataset. The expropriation risk measure is perhaps the most appropriate for our purpose as we would like to capture the variation in institutions originating from different types of colonial states and state policies (see Acemoglu et al., 2001). It is also the closest to Douglass North's (1981) definition of good institutions[6] as it captures the notion of extractive state. We also check the robustness of our results using rule of law and executive constraint measures.

Slave exports data is from Nunn (2008). Nunn (2008) reports the natural log of total slaves exported out of each of the African nations normalised by land area and population in 1400.[7] According to Nunn, the maximum number of slaves exported was from Angola which accounted for 23.1 percent of the total slave exports followed by Nigeria (12.9 percent) and Ghana (10.2 percent). The least slaves exported were from Tunisia. We follow Nunn and use log total slave exports normalised by land area as our preferred measure.

Identifying good empirical proxies for each of these variables is difficult but perhaps not the most challenging part of the analysis. The major challenges are to estimate the causal effects. In order for the estimates of $\alpha, \beta,$ and γ to be interpreted as causal effects, they have to overcome some serious econometric challenges. We list them as follows.

- *Endogeneity*. Economic development is a complex phenomenon. Given the complex nature of this process, reverse causality is a real possibility. For example, rather than

[6] North (1981) defines good institutions as those that provide checks against expropriation by the government and other politically powerful groups. (see pp. 20-27)

[7] These numbers are the aggregate of Atlantic slave trade, Indian Ocean slave trade, Red Sea and Trans-Saharan slave trade. For more details see Nunn (2008).

malaria influencing development the causality may run the other way round. The rich economies can afford to invest in the research and development of drugs that cures or minimises the effect of malaria. They can also invest in public health programs to tackle malaria. Similar argument can be made about institutions. Rich nations have better institutions not because they have grown richer due to better institutions, but they can afford better institutions. Furthermore, there can be endogeneity concerns with slave trade. Societies that initially had poor domestic institutions may have selected into the slave trades. Therefore the observed negative relationship between slave exports and development may not be the causal effect (Nunn, 2008). If this is the case then OLS estimates of $\alpha, \beta,$ and γ will be biased away from zero as we will be erroneously attributing the effects of income or other factors on endogenous variables to the direct effects of these variables on income.

- *Measurement error.* The slave exports data are likely to contain significant measurement error (Nunn, 2008). One can identify the following sources. First, slave ethnicities in the dataset may have been misclassified. Slaves with similar but different ethnicities may have been classified under one ethnicity. But the possibility of a bias due to errors of this nature is minimal as the data is aggregated at the country level. Second, measurement error may arise due to the under-representation of slaves from the interior or due to the assumption used in the construction of the data that slaves shipped from a port within a country are either from that country or from countries directly to the interior. In either case, OLS estimates of $\alpha, \beta,$ and γ will be biased towards zero (Nunn, 2008). Furthermore, any random measurement error present in the data will also have the same effect on OLS estimates.

- *Omitted variable bias.* Many of the omitted time invariant deep factors (culture, ethnic makeup, colonial or legal origin, religion, climate) influencing long-run economic development can be correlated with malaria risk, institutions, and slave exports. This has the potential of biasing the OLS estimates of $\alpha, \beta,$ and γ away from zero. We control for regional fixed effects, coloniser fixed effects, and legal origin fixed effects to tackle this problem. We also test the robustness of our estimates by controlling for additional covariates. Some of the obvious ones are trade openness, Catholicism, Islam, historical schooling, ethnic fractionalisation, share of mining, foreign aid, and Gini coefficient. However, as is the case with all empirical modelling, we can never be entirely sure that we have adequately controlled for all the omitted factors.

To tackle the problems of endogeneity and measurement error, we follow the literature and use the instrumental variable (IV) estimation (see Acemoglu et al., 2001; Sachs, 2003; Carstensen and Gundlach, 2006; Nunn, 2008; and many others). A valid instrument has to satisfy the twin conditions that it is correlated with the suspected endogenous variables (malaria, institutions, and slave exports in this case) but uncorrelated with the error term or a measurement error hidden in the error term in equation 1. It is obviously a difficult task to find valid instruments. However, the literature has identified several good instruments that can serve our purpose.

It is somewhat widely accepted now that log settler mortality and log population density in1500 are good instruments for institutions.[8] They are based on the idea that Europeans resorted to different style of colonisation depending on the feasibility of settlement. In a tropical disease environment with high settler mortality and also with a large native population the intention of the colonisers were not of settlement but rapid extraction of resources. Hence, they erected

extractive institutions in these colonies. The reverse was the case in a temperate disease environment and also with a small native population. Therefore, these instruments are likely to be negatively correlated with the quality of institutions and orthogonal to the random error term since they are geography based. Hence we use these two variables as instruments for institutions.

We also follow Nunn (2008) and use overland distance from the centroid to the coast, sailing distance from the coast to the closest market of the Atlantic slave trade, sailing distance from the coast to the closest market of the Indian Ocean slave trade, overland distance from the centroid to the closest port of export for the trans-Saharan slave trade, and overland distance from the centroid to the closest port of export for the Red Sea slave trade as instruments for slave exports. Nunn (2008) argues that the distance instruments are negatively correlated with slave exports and also exogenous. Therefore they are valid instruments. However, he notes a particular concern with the overland distance measure from the centroid to the coast. This instrument is likely to be correlated with international trade which influences income (see Rappaport and Sachs, 2003) and hence is not truly exogenous. But he shows that this instrument is positively correlated with the measurement error and negatively correlated with the error term in the second stage and will only bias the IV estimates towards zero. Therefore the IV estimates with this instrument should be treated as a lower bound. Having passed what may be called the Quarterly Journal of Economics (QJE) – test, Nunn's instruments are our best hope in estimating the causal effects of slave trade on Africa's current level of development. Also using his instruments makes our results comparable with his findings. We report estimates both with and without the overland distance measure from the centroid to the coast instrument and our results are robust either way.

Finally, we follow Sachs (2003) and Carstensen and Gundlach (2006) and use malaria ecology as an instrument for malaria risk. Malaria ecology is an ecologically-based spatial index

[8] See Glaeser et al. (2004) and Albouy (2006) for a different view. Also see Acemoglu et al. (2006) reply.

and depends on climatic factors and biological properties of each regionally dominant malaria vector. Hence it is exogenous to public health interventions and economic conditions, and thus can serve as an instrumental variable in regressions of economic performance on malaria risk (Kiszewski et al., 2004).[9] Rodrik et al. (2004) doubt the exogeneity of malaria ecology as they argue that from the little information provided by Sachs (2003), it remains unclear whether malaria ecology can be influenced by human action. Another concern regarding malaria ecology

[9] Detailed information on the construction of the instrument is available online at http://www.earthinstitute.columbia.edu/articles/view/1932. The webpage writes,

"Malaria Ecology is an ecologically-based spatial index of the stability of malaria transmission based on the interaction of climate with the dominant properties of anopheline vectors of malaria that determine vectorial capacity (Kiszewski et al., 2004). Malaria is a disease of climate because a key part of the life cycle of the parasites (sporogony) depends on a high ambient temperature and their vectors require sufficient rainfall to provide breeding sites. Additionally, the intensity of malaria transmission depends on the specific mosquito species that are present and their relative attraction to humans versus animals. The Malaria Ecology variable measures the effects of ambient temperature on the force of transmission of malaria, as expressed through the length of the extrinsic incubation period, and therefore the proportion of the vector population able to survive long enough to become infectious. The index is constructed on a 0.5 degree spatial grid to derive the climatic characteristics of individual months, and then averaged over a 12-month period. The first step is to identify the distribution of anopheline species across the world using observation records and satellite-based vegetation maps to identify likely habitats where observations have not been recorded.

A dominant species is identified for each spatial zone, and for each month (in cases where there is a seasonal pattern to the dominant species). We also employ an ecological screen for the presence or absence of a vector during particular months. (For those vectors that breed mainly in temporary water, a minimum precipitation threshold of 10mm per month, lagged one month, is used to judge when the vector would be present in the site during a given month. Vectors that mainly exploit permanent or semi-permanent bodies of water were considered to be independent of seasonal fluctuations in rainfall unless empiric evidence indicated otherwise. In temperate or altitudinous regions, temperature thresholds are used to determine whether parasites can develop in mosquito vectors in a particular month, assuming that malaria parasites cannot develop when the mean monthly temperature remains below 15°C). Note that the mosquito abundance screen is ecology-based and not affected by human activity; indeed, it is worth keeping in mind that public health interventions against malaria serve to break the transmission cycle, but do not eliminate the presence of the vector itself (even until today, Anopheles mosquitoes capable of transmitting malaria can be found throughout the US and Europe, places where malaria has been largely eradicated).

The basic formula for Malaria Ecology combines climatic factors, the presence of different mosquito vector types and the human biting rate of the different mosquito vectors. The index expresses the factors that most powerfully and perennially influence the intensity of malaria transmission. It uses, therefore, a subset of the vectorial capacity equation without terms for mosquito abundance, vector competence, or recovery rate for infected people. To calculate the duration of the extrinsic incubation period "E," the index was calculated for each month, and biting activity was designated based on the average monthly temperature and Moshkovsky's degree-day-based formulae.

$\sum_{m=1}^{12} a_{i,m}^2 p_{i,m}^E / -\ln(p_{i.m})$ Where: m = month, i = identity of dominant vector, a = proportion biting people, p = daily survival rate, E = length of extrinsic incubation period in days, where: E = 111 / T-16 for P. falciparum, and E = 105 / T-14.5 for P. vivax.

The underlying index is measured on a highly disaggregated sub-national level, and then averaged for the entire country and weighted by population. Because it is built upon climatological and vector characteristics on a country-by-country basis, Malaria Ecology is exogenous to public health interventions and economic conditions, and thus can serve as an instrumental variable in regressions of economic performance on malaria risk." (7 April, 2008)

comes from a previous version of the text describing the construction of the index as it says the calculation includes mosquito abundance. Even though both critiques are technically correct, the doubts about the exogeneity of the instrument may not be justified for the following reasons. First, the index is vector-based and not affected by human activity as public health interventions against malaria only serve to break the transmission cycle, but do not eliminate the presence of the vector itself. Even until today, Anopheles mosquitoes capable of transmitting malaria can be found throughout the US and Europe, places where malaria has been largely eradicated (see Kiszewski et al., 2004). Second, observed mosquito abundance enters the index only as a screen for precipitation data, where the independently identified dominant malaria vector is assumed to be absent from the specific site under consideration if precipitation falls below a certain level per month (see Carstensen and Gundlach, 2006). Nevertheless, we use average rainfall, average humidity, and prevalence of frost as alternative instruments for malaria and our results are robust to these changes.[10] Rainfall, humidity, and lack of frost are crucial to the life cycle of the parasite and hence serve as good instruments. They are also geography based and hence exogenous to economic conditions.

In IV estimation, endogenous explanatory variables are replaced by their predicted values from the first stage equations. The first stage equations are as follows.

$$\mathrm{MAL}_i = \mu + \delta \mathrm{ME}_i + \chi \mathrm{LSM}_i + \tau \mathrm{LPD}_i + \kappa \mathrm{DC}_i + \varepsilon_{\mathrm{MAL}_i} \qquad (2)$$

$$\mathrm{INS}_i = \varphi + \eta \mathrm{LSM}_i + \theta \mathrm{LPD}_i + \sigma \mathrm{ME}_i + \nu \mathrm{DC}_i + \varepsilon_{\mathrm{INS}_i} \qquad (3)$$

$$\mathrm{SLVX}_i = \psi + \omega \mathrm{DC}_i + \phi \mathrm{ME}_i + \pi \mathrm{LSM}_i + \upsilon \mathrm{LPD}_i + \varepsilon_{\mathrm{SLVX}_i} \qquad (4)$$

[10] Average rainfall and average humidity are from Nunn (2008) and prevalence of frost is from Masters and McMillan (2001).

where ME_i, LSM_i, LPD_i, and DC_i refers to malaria ecology, log settler mortality, log population density in 1500, and the distance instruments from Nunn (2008). Equations (1) – (4) are at the core of the empirical results that we report in the next section. We also report statistical tests (Hausman test, Sargan test, and Hansen test) for the validity of instruments.

An additional concern with IV is the bias due to weak instruments. Staiger and Stock (1997) and others have shown that the consequence of weak instruments is a large-sample bias in IV as in effect the model becomes unidentified. Furthermore, the magnitude of the large-sample bias increases with the number of instruments. As the bias is primarily a large-sample issue and it also increases with sample size, it may not be a cause of concern for us as we operate with a small sample. However, we try to tackle this problem by using the Limited Information Maximum Likelihood (LIML) method. LIML is more robust to the weak instruments problem than IV (Stock and Yogo, 2005). We find that our IV results are valid even when LIML is used.

3. Evidence

Table 2 reports the core results. In column 1 of panel A we start with estimating our basic model using OLS. We find that malaria negatively impacts on development, institutions are good for development, and slave exports are negatively correlated with development. We also plot the OLS partial effects (see Figure 1). The estimates however are likely to be inconsistent as OLS does not account for endogeneity or measurement error problems. In column 2 we estimate the model using IV. We notice that the negative effects of malaria survive however institutions and slave exports are statistically insignificant. The magnitude of the malaria effect is also large. A one standard deviation decrease in malaria risk increase income of an average country in Africa by 2.5 fold. To put this into perspective, the model explains approximately 60 percent of the actual variation in per capita income in Namibia and Mozambique – two countries who also share

approximately one standard deviation actual gap in malaria risk. The Hansen J test[11] and the first stage regressions reported in panel B shows that the instruments are valid however the Cragg-Donald test for weak instruments suggests that some of the instruments may be weak. Staiger and Stock (1997) and others have shown that weak instruments can cause large-sample bias in the IV estimates even when there are multiple instruments. The extent of the bias increases with the number of instruments. They suggest that F statistic of less than 10 at the first stage is a cause of concern. They recommend that cutting down on the number of instruments may help in reducing the large-sample bias. However this may not be useful for us as all instruments except malaria ecology and distance measures fail the Hall and Peixe (2000) instrument redundancy test (see panel C).[12] Stock and Yogo (2005) also show that LIML estimators are more robust to weak instruments than IV. In column 3 we report LIML estimates[13]. The results are similar to IV however the magnitude of the coefficient on malaria risk increases. We choose the IV as our preferred estimate since it is the lower bound. Furthermore, the extent of the bias due to weak instruments in IV may not be significant as we operate with a small sample and the bias is observed to be a large sample problem. We notice that the interior distance measure serves as a better instrument of colonial institutions than settler mortality. Also log population density in 1500 is a good instrument of slave exports. The population density correlation is consistent with Nunn (2008) as he shows that more slaves were exported from densely populated areas. The interior distance correlation may be due to the link that countries close to the coast trade more and more trade leads to better institutions. This story is consistent with Acemoglu et al. (2005) who

[11] Hansen J test is preferred over Hausman test as it is robust to random or cluster heteroskedasticity in standard errors.

[12] Weak instruments problem is not unique to this study and may as well be a general problem with the empirical comparative development literature as it is documented in Dollar and Kraay (2003) and elsewhere.

[13] We also use the Fuller's modified LIML estimator with $\alpha = 1$ (correction parameter proposed by Hausman et al., 2005) and we get results similar to column 2. Malaria risk is the only statistically significant variable with coefficient estimate -3.13 (se: 1.822). Results are not reported to save space but are available upon request.

show that Western European countries who had easy access to the Atlantic participated more in Atlantic trade which led to improvements in institutional quality and also rapid economic development.

Sachs (2003) predicts a 1.6 fold, 1.9 fold, and 1.8 fold increases in per capita GDP due to one standard deviation decline in malaria risk in AJR, RST, and EL samples respectively. Carstensen and Gundlach (2006) predict a 1.6 fold increase of the same. Both studies are based on a larger cross-country sample and not just limited to Africa. We find that this effect is even larger in an Africa sample as our estimates predict a 2.5 fold increase. Our results are at odds with the findings of Nunn (2008) who report slave exports have a causal effect on current development in Africa. Slave exports in our model are statistically insignificant. We also do not find statistical support for the colonial institutions view in Africa. This however does not imply refutation of these two theories. The result may be due to the weak instruments problem with log settler mortality and other widely used instruments of institutions.

In column 4 we estimate the causal effect of malaria on growth over the period 1960 to 2000. The effect is large as one standard deviation reduction in malaria yields approximately 1.2 percent growth dividends annually to an average country in Africa. This suggests that eliminating malaria alone in 1960 would have resulted in doubling of income in Africa by now. The relationship between malaria and growth is not surprising as current income levels and growth in Africa are correlated (approximately 0.7). We do not find evidence of causal effects of institutions and slave trade on growth.

Tables 3, 4, and 5 reports robustness tests with alternative instruments, with fixed effects, and with additional covariates. The alternative instruments strategy is to address the concerns about some of the instruments (especially malaria ecology). The fixed effects and the additional covariates strategies are to address the omitted variable problem. In column 1 of table 3, we

eliminate the interior distance instrument as it may be endogenous (see section 2 and also Nunn, 2008). We notice that the malaria result survives. We also replace the malaria ecology instrument with geography based instruments (rain, humidity, and frost) and the malaria result survives (see columns 2 – 5). The malaria result also survives the inclusion of coloniser fixed effects and legal origin fixed effects (see columns 1 and 3, table 4). However it vanishes when regional fixed effects are added (see column 2). This is not surprising as we find that the western region indicator dummy and the eastern region indicator dummy (which are representative of tropical Africa) are predicting negative impact on development. Therefore, the absence of malaria effect in the presence of western and eastern region indicator dummies may be because these dummies are picking up the negative malaria effect. Alternatively it may be due to deep cultural or geographic factors specific to these regions influencing both malaria and income. We are unable to separate out these effects. The malaria effect also survives the additional covariates test which is reported in table 5. The additional covariates (mining, ethnic fractionalisation, Catholicism, Islam, Gini coefficient, foreign aid, schooling, trade share)[14] are chosen on the basis of previous findings in the literature. The literature identifies these variables as important correlates of growth and development. Controlling for all additional covariates together may not be an option of choice as it weakens the power of statistical tests due to the loss of degrees of freedom.

Table 6 tests the robustness of the malaria result with alternative measures of institutions and slave exports and omission of influential observations. In column 1 we replace the expropriation risk measure of institutions with Rodrik et al.'s (2004) preferred measure the rule of law index. We notice that the malaria result survives and the magnitude of the coefficient is larger than our preferred estimate. In column 2 we replace it with executive constraints – another

[14] We also use corruption and Sachs and Warner openness index as additional covariates. The malaria result survives these tests. These results are not reported to save space.

measure of institutions used by Acemoglu et al. (2005) and many others. Our malaria result survives in this case. In column 3 we replace the log slave exports normalised by land area measure with log slave exports normalised by population. Again we notice that the malaria result survives. In column 4 we identify influential outliers using the DFITS, Cook's distance, and Welsch's distance formula (see Belsley et al. 1980) on the OLS regression reported in panel A, column 1 of table 2. The DFITS and Cook's distance formula identifies Ethiopia and Gabon as influential observations whereas the Welsch's distance formula identifies Gabon as an influential outlier. We omit these observations and estimate the model with IV. The malaria coefficient survives the test and even becomes larger in magnitude. In column 5 we use the DFBETA formula and omit Algeria, Ethiopia, Gabon, and Zambia. The malaria result survives and the coefficient becomes larger in magnitude.

4. Why Malaria is so Persistent in Africa?

Having successfully established the strong causal relationship between malaria and economic development in Africa, we now turn to another key question – why malaria is so persistent in Africa? Answer to this question may lie with the mechanism through which malaria impacts long term economic performance. In order to explore this issue we adopt the following strategies. First, we look at the conditional correlation between national savings and malaria. We observe a strong negative relationship between the two. Second, we develop an overlapping generation (OLG) model in which the household face a constant threat of death through malaria. There is also an adverse effect on their productivity from the disease. Here also we see a negative relationship between malaria and savings and hence economic growth. This helps us to better explain the persistence of malaria in Africa and also why malaria is a root cause of African underdevelopment.

4.1 Malaria and its Impact on Savings

To estimate the impact of malaria on savings, we use the following model.

$$\left(\frac{S}{Y}\right)_i = \varsigma + \vartheta \mathrm{MAL}_i + \rho \log y_i + \zeta_i \qquad (5)$$

The results are reported in Table 7. In column 1 we start off with the unconditional correlation between malaria and savings rate. The relationship is negative and statistically significant in the OLS model. A one standard deviation increase in malaria risk results into a 5.4 percentage point decline in the savings rate. Column 2 estimates the model using 2SLS. The effect remains unaltered in terms of direction and statistical significance, however, the magnitude of the effect declines to 4.2 percentage points. In column 3, we add log per capita income as a control. The result remains unaltered but the magnitude of the effect becomes bigger than column 2. A one standard deviation increase in malaria risk results into a 5.2 percentage point decline in the savings rate in this case. Column 4 estimates the model using 2SLS and the magnitude of the impact coefficient is 4.3 percentage points. Therefore malaria seems to have a negative causal effect on savings.

4.2 Explaining Persistence of Malaria in Africa using OLG Model

Having established the negative relationship between malaria and savings we move to our second strategy to uncover the mechanism. We develop an OLG model.[15] The model assumes a perfectly competitive decentralised economy with a single homogeneous good for both consumption and production. The households in this economy maximise their lifetime expected utility subject to an intertemporal budget constraint. A typical household comprises of both young and old members and each member of the household lives for only two periods. The young members work in the first period and retire in the second period when they are old, and then they die. The members also consume in both periods and the consumption in the second period is

supported by their savings in the first period. Therefore at each point in time, members of only two generations are alive. There is also a positive probability that they die of malaria before they reach their old age. The probability of survival into the second period is exogenous to the model but depends inversely on unfavourable geography. Unfavourable geography is characterised by the availability of malaria vector. An important point to note here is that malaria vector population cannot be influenced by human action and hence the unfavourable geography variable is strictly exogenous. Human action can only influence the transmission cycle but is incapable of eliminating the vector as it is geography based (also see discussion in section 2). In order to maintain simplicity of the structure, we assume away the possibility of bequests or any altruistic behaviour.

Households:

The lifetime utility of a representative household of generation t can be expressed as follows.

$$U = u(c_1) + \phi u(c_2) \qquad (6)$$

Where c_1 and c_2 are the consumption of generation t when young and old respectively. We are also assuming that the household gets zero utility from death and u is concave and twice differentiable.

The survival probability ϕ of the representative household depends on the unfavourable geography vector, $\mathbf{\Gamma}$.[16]

[15] Our model is not unique and similar models are used elsewhere (see Chakraborty and Das, 2005).

[16] One argument made by Chakraborty and Das (2005) in a recent paper is that the households can influence ϕ by investing in health. Without doubt their argument is valid for other illnesses (especially HIV/AIDS). However, here we are specifically trying to model malaria and scientific evidence suggests that malaria vector is geography based and cannot be influenced by human action (see Kiszewski et al., 2004). Therefore our assumption of ϕ to be exogenous as it is dependent on $\mathbf{\Gamma}$ is realistic. However, scientific evidence also shows that the transmission cycle of malaria vectors can be influenced by human action. This becomes significantly difficult in the tropics than in the temperate. One could say that this is the non-geography component of malaria. We do not explicitly model this as the

$$\phi = \phi(\mathbf{\Gamma}) \in [0,1] \qquad (7)$$

$\mathbf{\Gamma}$ is exogenous to the model and shares an inverse relationship with ϕ. If $\mathbf{\Gamma}$ is too high then ϕ can be too low.

The representative individual supplies one unit of labour inelastically when young and receives a wage income w. Therefore the budget constraint faced by this individual in period t is given by the following expression.

$$c_1 + s = w \qquad (8)$$

Where s is the amount of the homogeneous good saved in period t. The saved homogeneous good also grows at a rate r. Therefore in period t+1the individual consumes the amount saved in period t plus the growth in the homogeneous good. So the consumption in the second period is as follows.

$$c_2 = Rs \text{ where } R = 1 + r \qquad (9)$$

Using (9) one can rewrite the budget constraint as

$$c_1 + \frac{c_2}{R} = w \qquad (10)$$

For analytical convenience we assume that

$$u(c) = \frac{c^{1-\sigma}}{1-\sigma}, \sigma \in (0,1) \qquad (11)$$

Each household treat w and R as given and maximise their lifetime utility subject to the budget constraint. This yields the following Euler equation.

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aim is to support the IV estimates which capture the geography based causal effects of malaria on development. Even though it is theoretically possible to break the transmission cycle so that it outweighs the negative effects of $\mathbf{\Gamma}$ on ϕ, it can be prohibitively expensive to the extent that it is beyond the scope of a private investor (perhaps Bill and Melinda Gates are exceptions) or a household. One can look into these aspects by explicitly modeling non-geography component of malaria but we choose not to do so as this is not the focus of our study.

$$\frac{c_2}{c_1} = (\phi R)^{\frac{1}{\sigma}} \qquad (12)$$

Also from the first order conditions we get

$$c_1 = \frac{w}{1+\phi^{\frac{1}{\sigma}} R^{\frac{1}{\sigma}-1}} \;\&\; s = \frac{c_2}{R} = \frac{w\phi^{\frac{1}{\sigma}} R^{\frac{1}{\sigma}-1}}{1+\phi^{\frac{1}{\sigma}} R^{\frac{1}{\sigma}-1}} \qquad (13)$$

Production:

Every period the economy produces a single homogeneous good which can be consumed, saved, or invested. The output is produced using physical capital K and labour L. The production technology $\theta F(K, L)$ is neoclassical in nature and satisfies the Inada conditions. The parameter θ is less than 1 and is indicative of the morbidity effects of malaria. Due to morbidity of labour the economy cannot operate at the frontier of its production technology. It always underperforms. θ is also negatively dependent on $\mathbf{\Gamma}$. If geography is too unfavourable then θ is extremely low.

Under these conditions, in competitive product and factor markets, the economy wide wage and interest rates are:

$$w = \theta[f(k) - kf'(k)] \text{ and } r = \theta f'(k) \qquad (14)$$

where $k = \frac{K}{L}$. For simplicity, we assume no depreciation of capital.

Dynamics:

Aggregate savings in period t is used as aggregate capital stock for production in period t+1 in this economy. Assuming logarithmic preferences $(\sigma = 1)$ and Cobb-Douglas production technology, we get the dynamic equation of the economy as

$$k_{t+1} = \frac{\theta\phi(1-\alpha)}{(1+n)(1+\phi)} k_t^{\alpha} \qquad (15)$$

At steady state $k_{t+1} = k_t = k^*$ and therefore the steady state level of capital stock is given by the following:

$$k^* = \left[\frac{\theta\phi(1-\alpha)}{(1+n)(1+\phi)} \right]^{\frac{1}{1-\alpha}} \qquad (16)$$

From the above expression we can see that a low probability of survival (ϕ) into the second period and a low value of θ results into a low level of steady state capital stock and hence per capita income. In other words, high mortality and morbidity due to unfavourable geography may very well create an environment of a low level equilibrium trap. The low probability of survival effect works through the low savings channel. This certainly fits well with what we observe in the data. The causal effect of malaria on economic development in Africa may very well be working through low θ and low ϕ. This perhaps explains the long-term persistence of malaria in the continent.

5. Concluding Remarks

In this paper we investigate the relative strength of the malaria, colonial institutions, and slave trade view of African underdevelopment. The results show that malaria matters and all other factors are statistically insignificant. We are able to estimate the causal effect of malaria on development by exploiting the variation in malaria that is dependent on geography and can't be influenced by human action. We also show using an OLG model that malaria impacts African development by increasing both mortality and morbidity. Increased mortality induces households to increase current consumption and save less for the future. Increased morbidity on the other hand adversely affects productivity reducing household income and savings. This slows down capital accumulation and economic growth. This discussion also sheds some light on why malaria is so persistent in Africa.

The results when compared with Sachs (2003) and Carstensen and Gundlach (2006) reveal that the causal effect of malaria is larger in Africa compared to the same in a larger cross-country sample. Our results are in sharp contrast to Nunn (2008) who report a negative effect of slave exports on development in Africa. We notice that the slave exports variable is statistically insignificant when malaria is introduced into the model. The results however should not be interpreted as a refutation of colonial institutions and slave exports hypotheses. Failure to reject the null may not necessarily imply that colonial institutions and/or slave trade have no role. However it does imply that given the available measures of these variables and instruments, these views does not seem to have statistical support in Africa when one controls for the effect of malaria.

The paper is related to the large literature on health and development (see review in section 1) to the extent that it supports the disease view. In that sense it contributes to the growing evidence that disease control and health matter in development. It also contributes to the literature by providing a theoretical explanation of why malaria is so persistent in Africa.

References

Acemoglu, D., S. Johnson, and J. Robinson. (2001). "The colonial origins of comparative development: an empirical investigation," American Economic Review, 91(5), 1369-1401.

Acemoglu, D., S. Johnson, and J. Robinson. (2005). "The Rise of Europe: Atlantic Trade, Institutional Change and Economic Growth," American Economic Review, 95(3), 546-579.

Acemoglu, D., S. Johnson, and J. Robinson. (2006). "Reply to the Revised (May 2006) version of David Albouy's "The Colonial Origins of Comparative Development: An Investigation of the Settler Mortality Data"," manuscript, September.

Acemoglu, D., and S. Johnson. (2006). "Diseases and Development: The Effect of Life Expectancy on Economic Growth," NBER Working Paper No. 12269, May.

Albouy, D. (2006). "The Colonial Origins of Comparative Development: An Investigation of the Settler Mortality Data," manuscript, May.

Alesina, A., A. Devleeschauwer, W. Easterly, S. Kurlat, and R. Wacziarg. (2003). "Fractionalization," Journal of Economic Growth, 8, 155-194.

Arndt, C., and J. Lewis. (2000). "The Macro Implications of HIV/AIDS in South Africa: A Preliminary Assessment," South African Journal of Economics, LXVIII, 856-887.

Behrman, J., and M. Rosenzweig. (2004). "The Returns to Birthweight," Review of Economics and Statistics, 86(2), 586-601.

Bell, C., S. Devarajan, and H. Gersbach. (2003). "The Long-Run Economic Costs of AIDS: Theory and an Application to South Africa," working paper, June.

Belsley, D., E. Kuh, and R. Welsch. (1980). "Regressions Diagnostics: Identifying Influential Data and Sources of Collinearity," New York: John Wiley & Sons.

Benavot, A. and P. Riddle (1988). "The Expansion of Primary Education, 1870-1940: Trends and Issues." Sociology of Education, 61(3), 191-210.

Bhattacharyya, S. (2004). "Deep Determinants of Economic Growth," Applied Economics Letters, 11(9), 587-590.

Bleakley, H. (2003). "Disease and Development: Evidence from the American South," Journal of the European Economic Association, 1(2-3), 376-386.

Bloom, D. and D. Canning. (2005). "Health and Economic Growth: Reconciling the Micro and Macro Evidence," working paper, February.

Bloom, D. and J. Sachs. (1998). "Geography, Demography, and Economic Growth in Africa," Brookings Papers on Economic Activity, 2, pp. 207-295.

Carstensen, K. and E. Gundlach (2006). "The Primacy of Institutions Reconsidered: Direct Income Effects of Malaria Prevalence," World Bank Economic Review, 20(3), 309-339.

Chakraborty, S., and M. Das (2005). "Mortality, Human Capital and Persistent Inequality," Journal of Economic Growth, 10, pp. 159-192.

Collier, P. and J.W. Gunning. (1999). "Explaining African Economic Performance," Journal of Economic Literature, 37(1), 64-111.

Dollar, D., and A. Kraay. (2003). "Institutions, Trade, and Growth," Journal of Monetary Economics, 50, 133-162.

Easterly, W., and R. Levine. (1997). "Africa's Growth Tragedy: Policies and Ethnic Divisions," Quarterly Journal of Economics, 112(4), pp. 1203-1250.

Frankel, J., and D. Romer. (1999). "Does trade cause growth?" American Economic Review, 89(3), 379-399.

Gallup, J., and J. Sachs. (2001). "The Economic Burden of Malaria," American Journal of Tropical Medicine and Hygiene, 64(1-2), pp. 85-96.

Gemery, H. and J. Hogendorn. (1979). "The Economic Costs of West African Participation in the Atlantic Slave Trade: A Preliminary Sampling for the Eighteenth Century," In Gemery, H. and J. Hogendorn (eds.), The Uncommon Market: Essays in the Economic History of the Atlantic Slave Trade, Academic Press: New York.

Glaeser, E., R. LaPorta, F. Lopez-de-Silanes, and A. Shleifer. (2004). "Do Institutions Cause Growth?" Journal of Economic Growth, 9, 271-303.

Hall, R., and C. Jones. (1999). "Why do some countries produce so much more output per worker than others?" Quarterly Journal of Economics, 114(1), 83-116.

Hall, A., and F. Peixe. (2000). "A consistent method for the selection of relevant instruments," Econometric Society World Congress.

Hausman, J., J. Stock, and M. Yogo. (2005). “Asymptotic Properties of the Hahn-Hausman Test for Weak Instruments” Economics Letters, 89(3), 333-342.

Inikori, J. (1992). “The Chaining of a Continent: Export Demand for Captives and the History of Africa South of the Sahara, 1450-1870,” Institute of Social and Economic Research, University of the West Indies, Mona, Jamaica.

Kalemli-Ozcan, S. (2002). “Does the Mortality Decline Promote Economic Growth?” Journal of Economic Growth, 7(4), 311-349.

Kalemli-Ozcan, S. (2006). “AIDS, Reversal of the Demographic Transition and Economic Development: Evidence from Africa,” University of Houston, mimeo.

Kalemli-Ozcan, S., H. Ryder, and D. Weil (2000). “Mortality Decline, Human Capital Investment, and Economic Growth,” Journal of Development Economics, 62, 1-23.

Kiszewski, A., A. Mellinger, A. Spielman, P. Malaney, S. Sachs, and J. Sachs. (2004). “A Global Index Representing the Stability of Malaria Transmission,” American Journal of Tropical Medicine and Hygiene, 70(5), pp. 486-498.

Knack, S., and P. Keefer. (1995). “Institutions and Economic Performance: Cross-Country Tests using Alternative Institutional Measures,” Economics and Politics, 7, 207-227.

Knaul, F. (2000). “Health, Nutrition, and Wages: Age at Menarche and Earnings in Mexico,” in W. Savedoff and T. Schultz, eds., Wealth from Health: Linking Social Investments to Earnings in Latin America, Washington: Inter-American Development Bank.

LaPorta, R., F. Lopez-de-Silanes, A. Shleifer, and R. Vishny. (1999). “The Quality of Government,” Journal of Law, Economics and Organization, V15, N1, 222-279.

Lorentzen, P., J. McMillan, and R. Wacziarg. (2008). “Death and Development,” Journal of Economic Growth, forthcoming.

Manning, P. (1981). “The Enslavement of Africans: A Demographic Model,” Canadian Journal of African Studies, 15(3), 499-526.

Masters, W., and M. McMillan. (2001). “Climate and Scale in Economic Growth,” Journal of Economic Growth, 6(3), 167-186.

Miguel, E., and M. Kremer. (2004). “Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities,” Econometrica, 72(1), 159-217.

North, D. (1981). “Structure and Change in Economic History,” New York: Norton.

Nunn, N. (2007). “Historical Legacies: A Model Linking Africa's Past to its Current Underdevelopment", Journal of Development Economics, 83(1), 157-175.

Nunn, N. (2008). “The Long-Term Effects of Africa’s Slave Trades,” Quarterly Journal of Economics, 123(1), 139-176.

Rappaport, J., and J. Sachs. (2003). “The United States as a Coastal Nation,” Journal of Economic Growth, 8, 5-46.

Rodrik, D., A. Subramanian, and F. Trebbi. (2004). “Institutions Rule: the Primacy of Institutions over Geography and Integration in Economic Development,” Journal of Economic Growth, 9, 131-165.

Sachs, J. (2003). “Institutions Don’t Rule: Direct Effects of Geography on Per Capita Income,” NBER Working Paper No.w9490, February.

Sachs, J. (2005). “The End of Poverty: How We Can Make it Happen in our Lifetime,” Penguin Books, London.

Sachs, J. and A. Warner. (1997). “Sources of Slow Growth in African Economies,” Journal of African Economies, 6(3), 335-376.

Schultz, P. (2002). “Wage Gains Associated with height as a Form of Health Human Capital,” American Economic Review, 92(2), 349-353.

Staiger, D. and J. Stock. (1997). “Instrumental Variables Regression with Weak Instruments,” Econometrica, 65, 557-586.

Stock, J. and M. Yogo. (2005). “Testing for Weak Instruments in Linear IV Regression,” in D. Andrews and J. Stock, eds., Identification and Inference for Econometric Models: Essays in Honor of Thomas Rothenberg, Cambridge: Cambridge U y Press, 2005, pp. 80–108.

Temple, J. (1998). “Initial Conditions, Social Capital and Growth in Africa,” Journal of African Economies, 7(3), 309-347.

Weil, D. (2005). “Accounting for the Effects of Health on Growth,” NBER working paper 11455, July.

Young, A. (2005). “The Gift of the Dying: The Tragedy of AIDS and the Welfare of Future African Generations,” Quarterly Journal of Economics, CXX(2), 423-466.

Figure 1: Partial Correlation Plot: Root Causes of African Underdevelopment

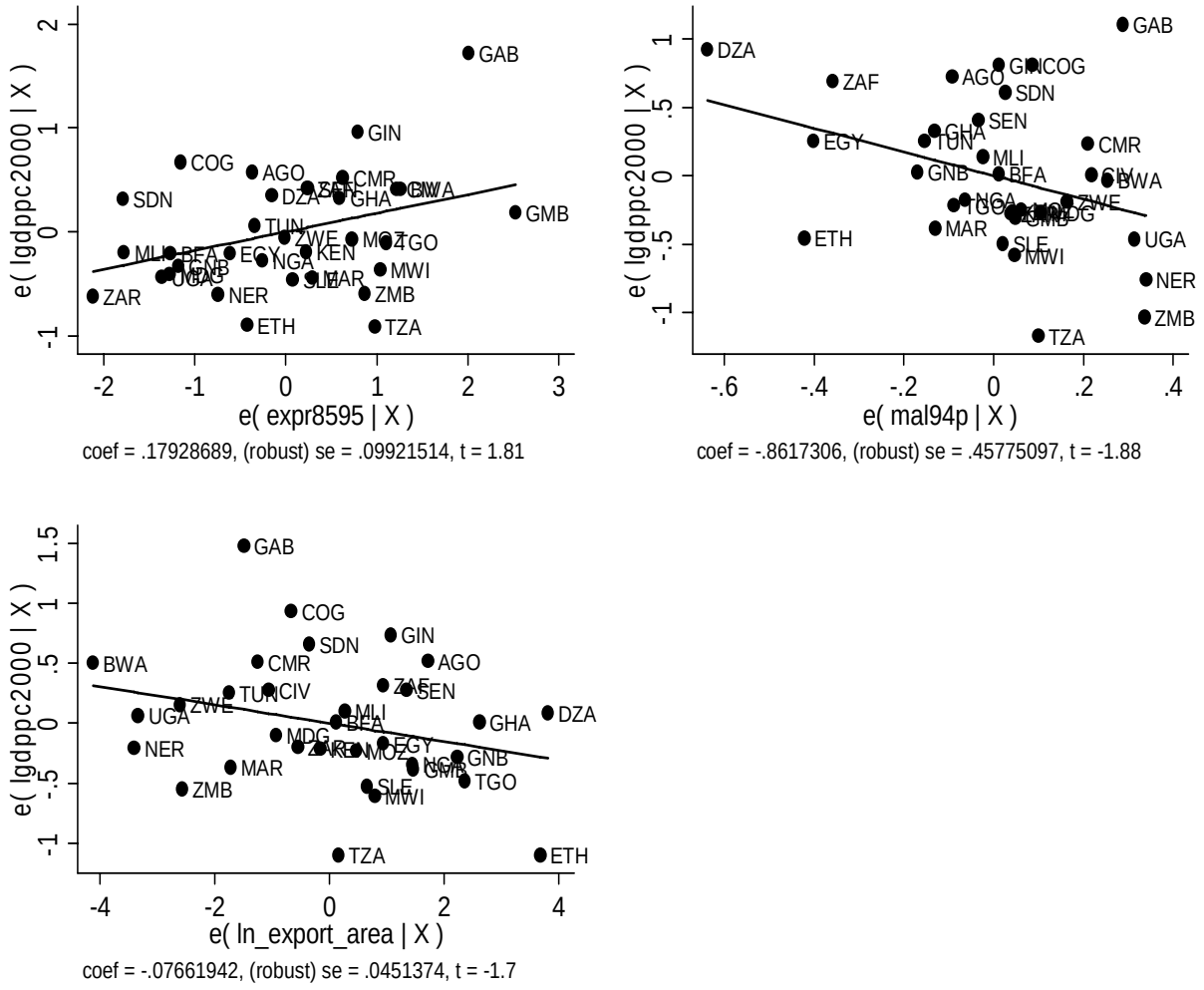

Table 1. Descriptive Statistics

Variable	Number of obs.	Mean	Standard Deviation	Minimum	Maximum
Log GDP per capita in 2000 ($\log y_i$)	46	7.46	0.815	6.19	9.24
Malaria Risk (MAL_i)	49	0.77	0.386	0	1
Expropriation Risk in 1985 to 1995 (INS_i)	35	5.82	1.30	3	8.27
Log total slave exports normalised by land area ($SLVX_i$)	52	3.26	3.89	-2.30	8.82

Table 2. Malaria as a Root Cause of African Underdevelopment: Core Results

Panel A: The Model $\log y_i = \lambda + \alpha \text{MAL}_i + \beta \text{INS}_i + \gamma \text{SLVX}_i + \varepsilon_i$				
Dependent Variable	**Log per Capita GDP in 2000**			**Growth during 1960 – 2000**
	OLS estimate obs= 33 (1)	2SLS estimate obs= 28 (2)	LIML estimate obs= 28 (3)	2SLS estimate obs= 28 (4)
Malaria Risk (MAL_i)	-0.86*	-2.41***	-4.93**	-0.03*
	(0.4576)	(0.8869)	(2.3614)	(0.0150)
Expropriation Risk in 1985 to 1995 (INS_i)	0.18*	0.10	-0.07	0.001
	(0.0992)	(0.1652)	(0.3396)	(0.0019)
Log total slave exports normalised by land area (SLVX_i)	-0.08*	0.16	0.52	0.0003
	(0.0451)	(0.1212)	(0.3310)	(0.0021)
Log per capita income in 1960				-0.004
				(0.0029)
R^2	0.59			
Hansen J test (p)		0.47	--	0.63
Hausman/Sargan test (p)		--	0.77	--
Cragg-Donald test (p)		0.89	--	--
Instruments		ME, LSM, LPD, IDC, ADC, IODC, SDC, RDC		

Panel B: The First Stage Regressions			
Dependent Variables	MAL_i obs= 28 (1)	INS_i obs= 28 (2)	SLVX_i obs= 28 (3)
Malaria Ecology (ME_i)	0.02*	-0.02	0.15
	(0.0094)	(0.0292)	(0.0980)
Log Settler Mortality (LSM_i)	0.10	0.01	0.48
	(0.0675)	(0.3232)	(0.6601)
Log Population Density in 1500 (LPD_i)	0.10	-0.33	1.77**
	(0.0760)	(0.3975)	(0.8948)
Interior Distance (IDC_i)	-0.00003	-0.002***	-0.001
	(0.00009)	(0.0007)	(0.0022)
Atlantic Distance (ADC_i)	-0.09	-0.01	-0.55
	(0.0666)	(0.2461)	(0.6952)
Indian Distance (IODC_i)	-0.05	-0.0003	0.007
	(0.0495)	(0.2063)	(0.6793)
Saharan Distance (SDC_i)	0.08	-0.31	1.78
	(0.1054)	(0.3843)	(1.275)
Red Sea Distance (RDC_i)	-0.14	-0.01	-1.25
	(0.0909)	(0.4413)	(0.8973)
R^2	0.79	0.45	0.59
F-stat	41.38	3.14	3.01

Panel C: Instrument Redundancy tests						
Instruments tested	**ME**	**LSM**	**LPD**	**IDC, ADC, IODC, SDC, RDC**	**ENGFRAC**	**EURFRAC**
LM test statistic	12.36	5.49	4.14	27.64	3.06	3.58
p-value	0.006	0.14	0.25	0.02	0.38	0.31
Degrees of freedom	3	3	3	15	3	3

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation. All the regressions reported above are carried out with an intercept. Both Hansen J test and Hausman/Sargan test p-values are reported. In both cases, the null hypotheses are that the instruments are jointly exogenous. Cragg-Donald test p-values for weak instruments are also reported. The null hypothesis in this case is that the instruments are jointly weak. The LM statistic for instrument redundancy tests are distributed as chi-squared under the null hypothesis that the specified instruments are redundant with degrees of freedom equal to the number of endogenous regressors times the number of instruments being tested.

Table 3. Malaria and African Underdevelopment: Robustness with Alternative Instruments

Dependent Variable	**Log per capita GDP in 2000**				
	2SLS estimate obs= 28 (1)	2SLS estimate obs= 28 (2)	2SLS estimate obs= 28 (3)	2SLS estimate obs= 25 (4)	2SLS estimate obs= 25 (5)
Malaria Risk (MAL_i)	-2.63***	-1.46***	-1.96**	-1.97***	-1.17**
	(0.8605)	(0.5225)	(0.9147)	(0.7218)	(0.5434)
Expropriation Risk in 1985 to 1995 (INS_i)	0.02	0.18	0.14	0.27*	0.33***
	(0.2637)	(0.1191)	(0.1554)	(0.1558)	(0.0999)
Log total slave exports normalised by land area ($SLVX_i$)	0.17*	-0.005	0.07	0.13	0.01
	(0.1081)	(0.0731)	(0.1322)	(0.0929)	(0.0891)
Hansen J test (p)	0.41	0.20	0.34	0.22	0.09
Instruments	without IDC	Replacing ME by Rain	Replacing ME by Humidity	Replacing ME by Frost	Replacing ME by Rain, Humidity, and Frost

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation. All the regressions reported above are carried out with an intercept. P-values of Hansen J tests are reported. The null hypothesis is that the instruments are jointly exogenous.

Table 4. Malaria and African Underdevelopment: Robustness with Fixed Effects

Dependent Variable	**Log per capita GDP in 2000**		
	2SLS estimate obs= 28 (1)	2SLS estimate obs= 28 (2)	2SLS estimate obs= 28 (3)
Malaria Risk (MAL_i)	-1.96**	1.25	-2.49***
	(0.7789)	(1.521)	(0.8708)
Expropriation Risk in 1985 to 1995 (INS_i)	0.06	0.19*	0.08
	(0.1643)	(0.1078)	(0.1701)
Log total slave exports normalised by land area ($SLVX_i$)	0.07	-0.005	0.18
	(0.0892)	(0.0797)	(0.1148)
Hansen J test (p)	0.18	0.19	0.66
Fixed Effects	Coloniser Fixed Effects	Region Fixed Effects	Legal Origin Fixed Effects
Instruments	ME, LSM, LPD, IDC, ADC, IODC, SDC, RDC		

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation. Coloniser fixed effects, region fixed effects, and legal origin fixed effects are dummies representing colonial origin, region, and legal origin respectively.

Table 5. Malaria and African Underdevelopment: Robustness with Additional Covariates

Dependent variable	**Log per Capita GDP in 2000**							
	2SLS estimate obs= 28 (1)	2SLS estimate obs= 28 (2)	2SLS estimate obs= 27 (3)	2SLS estimate obs= 27 (4)	2SLS estimate obs= 20 (5)	2SLS estimate obs= 28 (6)	2SLS estimate obs= 11 (7)	2SLS estimate obs= 27 (8)
MAL_i	-2.53*** (0.7732)	-2.22*** (0.8659)	-1.13* (0.6616)	-2.15*** (0.8184)	-1.22** (0.5854)	-2.29*** (0.7652)	-1.67*** (0.4836)	-1.92*** (0.6632)
INS_i	0.03 (0.1527)	0.11 (0.1595)	0.22 (0.1414)	0.10 (0.1610)	0.28 (0.2831)	0.16 (0.1283)	-0.02 (0.1379)	0.03 (0.1477)
$SLVX_i$	0.14 (0.1014)	0.14 (0.1149)	-0.05 (0.0895)	0.09 (0.1159)	-0.01 (0.0857)	0.16 (0.1081)	-0.006 (0.0542)	0.06 (0.0774)
Hansen J test	0.57	0.43	0.04	0.29	0.15	0.36	0.51	0.30
Additional Covariates	Mining	Ethnic Fractionalisation	Catholicism	Islam	Gini Coefficient	Foreign Aid	Schooling in 1900	Trade Share
Instruments	ME, LSM, LPD, IDC, ADC, IODC, SDC, RDC							All Instruments plus CONST

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation. All the regressions reported above are carried out with an intercept. The instrument CONST is constructed openness from Frankel and Romer (1999).

Table 6. Alternative Measures and Influential Observations tests

Dependent Variable	Log per capita GDP in 2000				
	2SLS estimate obs= 34 (1)	2SLS estimate obs= 32 (2)	2SLS estimate obs= 28 (3)	2SLS estimate obs= 26 (4)	2SLS estimate obs= 24 (5)
MAL_i	-3.69**	-2.15***	-3.71***	-2.57***	-2.93***
	(1.681)	(0.3453)	(1.412)	(0.6789)	(0.8270)
INS_i			0.11	-0.04	-0.10
			(0.2256)	(0.1316)	(0.1405)
Rule of Law Index	-0.93				
	(0.9144)				
Executive Constraint		-0.04			
		(0.1128)		0.13	0.15*
$SLVX_i$	0.24	0.10*		(0.0809)	(0.0897)
	(0.1626)	(0.0600)			
Log total slave exports normalised by population			0.41*		
			(0.2347)		
Hansen J test (p)	0.96	0.14	0.89	0.64	0.85
Omitted Influential Outliers				ETH, GAB	DZA, ETH, GAB, ZMB
Instruments	ME, LSM, LPD, IDC, ADC, IODC, SDC, RDC				

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation. Coloniser fixed effects, region fixed effects, and legal origin fixed effects are dummies representing colonial origin, region, and legal origin respectively. Influential observations are omitted using the following standard rules. In column 4, omit if at least $|DFITS_i| > 2\sqrt{\frac{k}{n}}$, $|Cooksd_i| > \frac{4}{n}$, and $|Welschd_i| > 3\sqrt{k}$ holds (see Belsley et al. 1980). In column 5, an additional formula is used which is $|DFBETA_i| > 2/\sqrt{n}$. Here n is the number of observation and k is the number of independent variables including the intercept. All the distance formulas are calculated from the OLS version of the model.

Table 7. Malaria and National Savings

The Model $\left(\frac{S}{Y}\right)_i = \varsigma + \vartheta \mathrm{MAL}_i + \rho \log y_i + \zeta_i$				
Dependent Variable	**Gross Savings as percentage of GDP in 2000** $\left(\frac{S}{Y}\right)$			
	OLS Estimate obs = 42	2SLS Estimate obs = 42	OLS Estimate obs = 40	2SLS Estimate obs = 40
MAL_i	-15.21***	-12.29**	-15.22***	-12.56**
	(3.674)	(4.997)	(3.923)	(5.248)
$\log y_i$			2.58	2.96
			(2.069)	(1.919)
R^2	0.30		0.33	
F-Stat	16.67	5.76	7.85	3.74
P-value	0.0002	0.0211	0.0014	0.033
Instruments		ME		ME

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation.

Data Appendix

Log per capita GDP in 2000 ($\log y_i$): Penn World Table (PWT) 6.1.

Expropriation Risk (INS_i): risk of “outright confiscation and forced nationalization" of property, ICRG.

Executive constraint in 2000: A seven category scale, 1 to 7, with a higher score indicating more constraint, Polity IV.

Rule of Law Index: see Rodrik et al. (2004) for details.

Malaria Risk: Percentage of the population at risk of malaria transmission in 1994, CID datasets, Harvard University.

Malaria Ecology (ME): Kiszewski et al. (2004).

Log total slave exports normalised by land area ($SLVX_i$): see Nunn (2008).

Log total slave exports normalised by population: see Nunn (2008).

Log Settler Mortality (LSM): Acemoglu et al. (2001).

Log Population Density in 1500 (LPOPDEN): Acemoglu et al. (2001).

ENGFRAC: fraction of the population speaking English, Hall and Jones (1999).

EURFRAC: fraction of the population speaking other European languages, Hall and Jones (1999).

Interior Distance (IDC_i), Atlantic Distance (ADC_i), Indian Distance ($IODC_i$), Saharan Distance (SDC_i), and Red Sea Distance (RDC_i): Nunn (2008).

Frost: Masters and McMillan (2001), see Carstensen and Gundlach (2006) for details.

Rain: Minimum of monthly average rainfall, Nunn (2008).

Humidity: Maximum of monthly afternoon average humidity (%), Nunn (2008).

Legal origin: LaPorta et al. (1999).

Schooling in 1900: Benavot and Riddle (1988).

Log trade share in 2000: WDI online.

CONST: Constructed openness, Frankel and Romer (1999).

Ethnic Fractionalisation: Alesina et al. (2003).

Mining: Share of mining in GDP, Hall and Jones (1999).

Catholicism and Islam: LaPorta et al. (1999).

Gini coefficient: World Bank.

Foreign Aid and National Savings: WDI online

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