

Root Causes of African Underdevelopment

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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 Atlantic distance, Indian Ocean distance, Saharan distance, Red Sea distance, log settler mortality, and malaria ecology as instruments. The results show that malaria matters the most and all other factors are statistically insignificant. Malaria also negatively affects savings. The results are robust even when the malaria ecology instrument is replaced by frost, humidity, and rainfall and when the latter are used as additional control variables. We find that frost alone is enough to knock off the effects of slave trade and institutions on long term development in Africa.

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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).¹ 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 global cross-national dataset 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 (2007) however question these results. They find that there is no

¹ Earlier contributions by historians also suggest that malaria indirectly affected development of the continent by causing massive depopulation in the agriculturally marginal regions (Dias, 1981 and Miller, 1982). They argue that slave trade was also an outcome of local epidemiology (particularly malaria) and poor agriculture among other things.

statistically significant effect of improved life expectancy on GDP levels leading them to conclude that diseases do not have a direct role in development.

Despite the doubts posed by Acemoglu and Johnson (2007), 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.² 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.³

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, 2008; Nunn, 2007). The story as outlined by Acemoglu et al. (2001) goes as follows.⁴ 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

² 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.

³ Acemoglu and Johnson (2007) argue that their results are not comparable with the micro studies as the micro studies do not incorporate general equilibrium effects.

⁴ An alternative story of African institutions is from Herbst (2000). He argues that due to the abundance of land in Africa, there was hardly any competition among pre-colonial states to defend a well defined territory. This prevented the development of state institutions (tax collection, defence, bureaucracy, rule of law etc.). This trend of almost no external threat continued during the colonial period. Therefore the colonisers also had very little incentive to develop good institutions. After independence the situation did not change and what we observe now is the weak institutions of contemporary Africa.

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.⁵ Hence, the Acemoglu et al. (2001) argument is that diseases affect economic performance only indirectly through institutions.⁶ Nunn (2007) using a stylised model for Africa 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 report strong link between quality of institutions and post-war growth (or the lack of it) in Africa.⁷ 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 (see Gemery and Hogendorn, 1979;

⁵ Alternatively, Glaeser et al. (2004) argue that the European settlers took human capital with them when they migrated and not institutions. Bhattacharyya (2008) show that it is impossible to empirically separate out the effects of human capital and institutions on long-run development in a cross-section model due to multicollinearity problems. However, one can successfully estimate separate effects of unbundled institutions (market creating, market regulating, market stabilising, and market legitimising institutions) and human capital on growth using panel data.

⁶ Bhattacharyya (2009) presents a unified framework to link the institutions and the diseases view. The framework is tested using case study evidence from each continent.

⁷ See Collier and Gunning (1999) for a survey of this literature.

Inikori, 1992; and Manning, 1981). The result was a significant slowdown in division of labour, demographic transition,⁸ human capital accumulation and long-run economic growth (Inikori, 1992). Depopulation also resulted into an implosion of the continent's production possibility frontier⁹ and an unambiguous reduction in welfare (Gemery and Hogendorn, 1979). The secular decline in welfare continued over more than two centuries plunging the continent into economic backwardness. In a recent paper, Nunn (2008) also report a negative causal relationship between slave trade and current economic performance in Africa. He shows that slave trade prevented state development, encouraged ethnic fractionalisation and weakened legal institutions and through these channels it affected economic development.

These competing theories¹⁰, 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. Malaria ecology from Kiszewski et al. (2004) is used as an instrument for malaria risk. Given the controversy regarding exogeneity of malaria ecology, we also use rain, humidity, and frost as alternative instruments. Our basic result survives

⁸ Faced with an increasing demand for slaves from the new world, African demand for slaves also increased. Africans preferred female slaves whereas young age male slaves were exported across the Atlantic. Result was a huge imbalance in African sex ratio, slow down of demographic transition and economic progress (Manning, 1981).

⁹ There wasn't enough labour to support capital and facilitate industrialisation in an already labour scarce continent.

¹⁰ Nunn (2008) argues that the effect of slave trade on development may work through institutions. Therefore slave trade and institutional weaknesses may not be competing explanations of African underdevelopment. Diseases and institutions however are competing theories of African underdevelopment.

this test. Institutions and slave exports are instrumented by log settler mortality¹¹ from Acemoglu et al. (2001) and distance measures from Nunn (2008) respectively. The results show that malaria matters the most and all other factors are statistically insignificant. This result survives even when we use Nunn's econometric specification and dataset. We also show that malaria dampens savings. Increases in mortality and morbidity can be possible channels through which malaria impacts African development. Increased mortality induces households to increase current consumption and save less for the future (hence the negative relationship between savings and malaria). 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.

We contribute to the literature by showing that malaria is the most powerful explanator (at least statistically) of long-run economic development (or the lack of it) in Africa. None of the other factors (including institutions and slave trade) are statistically significant. We also provide an explanation for the persistence of malaria in Africa.¹²

Previous studies have tested statistically the merits of competing theories (institutions and diseases) of long-run development using a global sample (see Acemoglu et al. 2001; Sachs, 2003; Rodrik et al. 2004; Carstensen and Gundlach, 2006; and many others). None of them however focus on an Africa only sample. Furthermore, none of them report malaria to be the only statistically significant variable in a global sample. A common finding is that malaria and institutions are both important (see Sachs, 2003; and Carstensen and Gundlach, 2006). In that sense our result is unique and goes against previously published results. It also goes against the

¹¹ Note that the settler mortality instrument is not free from controversy either. Recently Albouy (2008) identified several problems with the construction of the original variable in Acemoglu et al. (2001) and the revised dataset published as MIT mimeo by the same authors in March 2005 and September 2006. We continue to use the original variable here to facilitate comparison with all other papers that have used this variable.

¹² An earlier version of the paper presents an overlapping generation model to outline the causal channels through which this may work. Not presented in this version but are available upon request.

results of Nunn (2008) who argue that slave trade affects Africa's current economic performance through ethnic fractionalisation and weak institutions. We notice that the direct and indirect effects of slave trade disappear when we introduce malaria as a control. This finding is robust even when we use Nunn's specification and exactly the same dataset. Furthermore, we notice that frost alone is enough to knock off the effects of slave trade and institutions on long term development in Africa.

The benefits of looking at an Africa only sample are threefold. First, it allows us to statistically scrutinize Nunn's result that slave trade has a causal effect on Africa's current economic performance and the effect works through ethnic fractionalisation and weak institutions. Second, it allows us to statistically test the strengths of competing theories of African underdevelopment¹³. Third, it allows us to focus on a continent where the majority of the bottom billion countries are located¹⁴ (Collier, 2007).

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, 2007; 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 effects of malaria.

¹³See Herbst (2000) for Africa specific institutions theory, Miller (1982) for Africa specific malaria theory, Inikori (1992) for slave trade theory.

¹⁴Note that this does not bring in selection bias as our sample contains both rich and poor countries from Africa. Previous cross-country studies have also used Africa only sample.

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 check the robustness of our results by using exactly the same specification and dataset as Nunn (2008). This study is the most closely related to ours. We also ask the question why malaria is so persistent in Africa. In other words, what are the channels through which malaria affects income? We empirically identify savings as an important channel and also provide an explanation. Section 4 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 literature¹⁵ and estimate the following model.

$$\log y_i = \lambda + \alpha \text{MAL}_i + \beta \text{INS}_i + \gamma \text{SLVX}_i + \mathbf{x}_i \boldsymbol{\Phi} + \varepsilon_i \quad (1)$$

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. \mathbf{x}_i is a row vector of additional control variables¹⁶ and ε_i is the random error term. We are interested in the size, sign, and statistical significance of the three coefficients α , β , 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

¹⁵ See Acemoglu et al. (2001), Rodrik et al. (2004), Sachs (2003), Carstensen and Gundlach (2006), Nunn (2008), and many others who use similar models.

¹⁶ We use log population density in 1500 and interior distance as additional control variables as they might influence current development through other channels (Acemoglu et al., 2002; Nunn, 2008). Acemoglu et al. (2002) and Nunn (2008) do not use them as instruments because they fail to satisfy the exclusion restrictions.

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. We also use per capita income data from Nunn in Table 7 when we check the robustness of our result using Nunn's dataset and specification. Note that Nunn uses income data from Maddison (2003).

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 1994.¹⁷ 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.

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 institutions¹⁸ 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

¹⁷ For more details see <http://www.earth.columbia.edu/articles/view/1932>

¹⁸ North (1981) defines good institutions as those that provide checks against expropriation by the government and other politically powerful groups. (see pp. 20-27)

1400.¹⁹ 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 α , β , 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 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 α , β , 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.

¹⁹ 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).

- *Measurement error.* The slave exports data are likely to contain both classical and non-classical 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 (Nunn, 2008). 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 α , β , and γ will be biased towards zero – the classical measurement error (Wooldridge, 2000). Furthermore, any random measurement error present in the data will also have the same effect on OLS estimates. Moreover, it is not possible to rule out non-classical measurement error.
- *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 α , β , 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 instruments that can serve our purpose.

Previous studies have used log settler mortality as an instrument for institutions (Acemoglu et al., 2001; Rodrik et al., 2004; and others). It is based on the idea that European colonisers erected good institutions only in the settlement colonies. Elsewhere they erected extractive institutions. Therefore, the settler mortality instrument is likely to be negatively correlated with the quality of institutions and also orthogonal to the random error term since it is geography based. Recently this instrument has come under intense scrutiny. Glaeser et al. (2004) show that this instrument fails to satisfy the exclusion restriction since it is highly correlated with schooling. Bhattacharyya (2008) shows that it is almost impossible to separate out the effects of institutions and human capital on long-run development in a cross-section model due to multicollinearity. This is true regardless of the specifications used (see Bhattacharyya, 2008). Albouy (2008) identifies several weaknesses with regards to the construction of the instrument. In spite of the controversy regarding this instrument, we continue to use it here to facilitate comparison of our results with previous studies in the literature.

We also follow Nunn (2008) and use 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. He also uses the overland distance from the centroid to the coast and log population density in 1400 as additional instruments. However, he notes that the additional instruments may not satisfy the exclusion restrictions. Therefore, we decide not to use these additional instruments.²⁰ 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 it easier to compare our findings with Nunn (2008).

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 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).²¹ 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 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

²⁰ Our basic results are unaffected even if we use of these additional instruments.

²¹ Detailed information on the construction of the instrument is available online at <http://www.earthinstitute.columbia.edu/articles/view/1932>.

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.²² 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.²³

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

$$MAL_i = \mu + \delta ME_i + \chi LSM_i + \kappa DC_i + \mathbf{x}_i \Phi + \varepsilon_{MAL_i} \quad (2)$$

$$INS_i = \varphi + \eta LSM_i + \sigma ME_i + \nu DC_i + \mathbf{x}_i \Phi + \varepsilon_{INS_i} \quad (3)$$

$$SLVX_i = \psi + \omega DC_i + \phi ME_i + \pi LSM_i + \mathbf{x}_i \Phi + \varepsilon_{SLVX_i} \quad (4)$$

where ME_i , LSM_i , and DC_i refers to malaria ecology, log settler mortality, 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.

²² Average rainfall and average humidity are from Nunn (2008) and prevalence of frost is from Masters and McMillan (2001).

²³ One concern is that rainfall, humidity, and frost may not satisfy the exclusion restriction because they may affect development through channels other than malaria. Statistically this will bias our estimates only if the predicted value of malaria at the second stage is correlated with the error term. Hansen J test for exogeneity of instruments indicate otherwise (see Table 3). Nevertheless, we also use them as exogenous control variables which may directly influence economic performance. Our results survive this test.

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. The Staiger and Stock (1997) results rely on asymptotic properties and the asymptotic distribution theory may not necessarily apply for our small sample. However, the bias in 2SLS can't be ruled out. More importantly, the Limited Information Maximum Likelihood (LIML) estimator does not have such bias. It is also more robust to the weak instruments problem than IV (Stock and Yogo, 2005). Our basic results survive when we use the LIML estimator.

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 development, institutions are good for development, and slave exports are negatively correlated with development.²⁴ 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 5 fold. To put this into perspective, the model explains approximately 92 percent of the difference in per capita income in Namibia and Nigeria – two countries who also share approximately one standard deviation actual gap in malaria risk. The Hansen J test²⁵ and the first

²⁴ Note that including log population density in 1500 and interior distance as additional controls do not alter our malaria result in column 1. In fact institutions and slave exports become statistically insignificant.

²⁵ Hansen J test is preferred over Hausman test as it is robust to random or cluster heteroskedasticity in standard errors.

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 a useful strategy for us as all our instruments pass the Hall and Peixe (2000) instrument redundancy test (see panel C). Note that weak instruments problem is not unique to this study and may as well be a general problem with the empirical comparative development literature (Dollar and Kraay, 2003; Bhattacharyya, 2008). Stock and Yogo (2005) show that LIML estimators are more robust to weak instruments than IV. In column 3 we report Fuller's modified LIML estimates with $\alpha = 1$ (correction parameter proposed by Hausman et al., 2005) and we get results similar to IV.²⁶ The magnitude of the coefficient on malaria risk declines. The model now explains approximately 73 percent of the difference in per capita income in Namibia and Nigeria. We choose the LIML as our preferred estimate since it is the lower bound. The positive correlation between malaria ecology slave trade at the first stage is certainly noteworthy. This is consistent with the view that slave trade was also an outcome of local epidemiology, particularly malaria (see Dias, 1981 and Miller, 1982). We also notice that the interior distance is negatively correlated with colonial institutions. This may be due to the possibility that proximity to the coast leads to more trade and more trade leads to better institutions (see Acemoglu et al. 2005).

²⁶ One could argue that the presence of both slave trade and institutions in the model weakens the direct effect of institutions on long-run development. To allay this concern, we run a direct contest between malaria and institutions leaving out slave trade as a control. We estimate this model using LIML. Malaria is the clear winner with a coefficient estimate of -1.37 (se: 0.4510) and institutions are statistically insignificant.

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 global sample and they find both institutions and malaria are statistically significant. We find that the malaria effect is even larger (our preferred LIML estimate predicts a 3.6 fold increase) in an Africa only sample and all other factors are statistically insignificant. Our results are at odds with the findings of Nunn (2008) who report that slave exports have a causal effect on current development in Africa via state development, ethnic fractionalisation and weakened legal institutions. We do not find any statistical evidence of direct and indirect effects of slave trade on Africa's current development. To be completely sure we also check the robustness of our result using Nunn's specification and dataset (see Table 7). Our malaria result survives. We also do not find statistical support for the colonial institutions view in Africa. This is regardless of the specification and sample.

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 1.5 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 fail to 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.²⁷ The alternative instruments strategy is to address the concern

²⁷ Note that we estimated all the specifications reported in tables 3, 4, and 5 using LIML. The results are qualitatively the same.

that malaria ecology is not exogenous. The fixed effects and the additional covariates strategies are to address the omitted variable problem. In table 3 columns 1 – 4 we replace the malaria ecology instrument with geography based instruments (rain, humidity, and frost) and the malaria result survives.²⁸ One concern is that rain, humidity, and frost may not satisfy the exclusion restriction as they might influence income through channels other than malaria. To address this concern we use these variables as additional controls in columns 5 – 7. The malaria result survives in column 5. The large standard errors and statistical insignificance of all variables in columns 7 and 6 may be due to small sample size, degrees of freedom problems and multicollinearity (Dollar and Kraay, 2003; Bhattacharyya, 2008). 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, it can very well be the case that these dummies are picking up the negative malaria effect. Multicollinearity between malaria and the regional dummies can also be an issue here as we notice large standard error on malaria estimate. 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 in majority of cases (7 out of 8) which is reported in table 5. The additional covariates (mining, ethnic fractionalisation, Catholicism, Islam, Gini coefficient, foreign aid, schooling, trade share)²⁹ are chosen on the basis of previous findings in the literature. The

²⁸ For curiosity sake we also check the robustness of our malaria result using *logmort2* from Albouy (2008) as an instrument for institutions instead of Acemoglu et al.'s (2001) settler mortality. Our malaria result survives and all other variables are statistically insignificant.

²⁹ 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.

literature identifies these variables as important correlates of growth and development. Controlling for all additional covariates together may not be an option 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 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. The malaria coefficient survives the test. 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.

In table 7 we test the robustness of our malaria result using Nunn's specification and data. In column 1 we estimate Nunn's preferred specification³⁰ (see table 5, column 6 of Nunn, 2008, p. 31). Our estimate of -0.20 is marginally different from Nunn's -0.188.³¹ In column 2 we add

³⁰ This specification is estimated without interior distance and log population density in 1400 instruments.

³¹ Surprisingly, we get very different first stage estimates. We are somewhat puzzled with this outcome as we are using exactly the same specification, dataset and sample of countries as Nunn. Not reported here to save space but available upon request.

malaria into this specification and the statistical significance of the slave trade variable disappears. Nunn argues that the effect of slave trade may be working through institutions. Column 3 checks this possibility by adding institutions into the mix. The malaria effect survives and neither institutions nor slave trade are statistically significant. In column 4 we replace malaria ecology with the geography based instrument humidity. The malaria result survives.³² In columns 5 – 7 we check whether the indirect effects of slave trade can survive the malaria test. Nunn argue that slave trade works through pre-colonial state development, rule of law, and ethnic fractionalisation (see table 8 of Nunn, 2008, p. 37). None of these variables are statistically significant in the presence of malaria. In columns 8 and 9 we use a more direct approach to test the robustness of the slave trade result of Nunn (2008) and institutions result of Acemoglu et al. (2001). We check what happens to these results when we use frost as an additional control variable. Note that we do not use frost as an instrument to address the concern that it may not satisfy the exclusion restriction. Also note that we choose not to use the controversial malaria ecology variable. It appears that frost alone is enough to knock off the slave trade and institutions results.³³ This further reinforces our point that the slave trade and institutions results are extremely weak for the continent of Africa. Malaria is the only statistically significant variable.

Next, we ask the 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 table 8 we report a strong negative relationship between national savings and malaria in Africa even after controlling for income. This is perhaps indicative of the fact that

³² Note that the result is qualitatively the same if we use humidity as an additional control and not as an instrument.

³³ The results are even more unfavourable for slave trade and institutions if we control for malaria ecology, log population density in 1500, frost, rainfall, and humidity. This result holds if we eliminate log population density in 1500 from the mix. The only exception is the case when log population density in 1500, frost, rainfall, and humidity are used as additional controls. Slave trade is marginally significant with p-value 0.09. This is not surprising as it is very close to Nunn's original specification. Institutions however are statistically insignificant. STATA codes for these variants are downloadable from the author's website <http://rspas.anu.edu.au/~sambit/>.

malaria influences long-run development in Africa through the savings channel. Malaria increases mortality and morbidity. High mortality rate induces households to save less and consume more. Morbidity reduces productivity shrinking household's income and the ability to save. The result is a low level equilibrium trap and persistent poverty. This perhaps helps explain the persistence of malaria in Africa and also why malaria is a root cause of African underdevelopment. An earlier version of this paper explains this mechanism using an OLG model.

4. Concluding Remarks

In this paper we investigate the relative strength of malaria, colonial institutions, and slave trade view of African underdevelopment. The results show that malaria matters the most (at least statistically) and all other factors are statistically insignificant in an Africa only sample. This is different from Sachs (2003) and Carstensen and Gundlach (2006) who show that malaria and institutions are both important in a global sample. It is also at odds with Nunn (2008) as we do not find any statistical support for his claim that slave trade affects current development in Africa directly and indirectly (through institutions and ethnic fractionalisation). This is true even when we use Nunn's specification and dataset. One way to interpret our result is 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 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. One should not forget that the process of long-run

economic development is complex and cannot be adequately captured using reduced form models. However it does imply that the recent claims of strong statistical support in favour of causal effects of institutions (see Acemoglu et al., 2001) and slave trade (see Nunn, 2008) on long-run development in Africa is at the very least statistically weak.

The paper is related to the large literature on health and development to the extent that it supports the disease view. In that sense it contributes to the growing evidence that disease control and health matters in development.

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

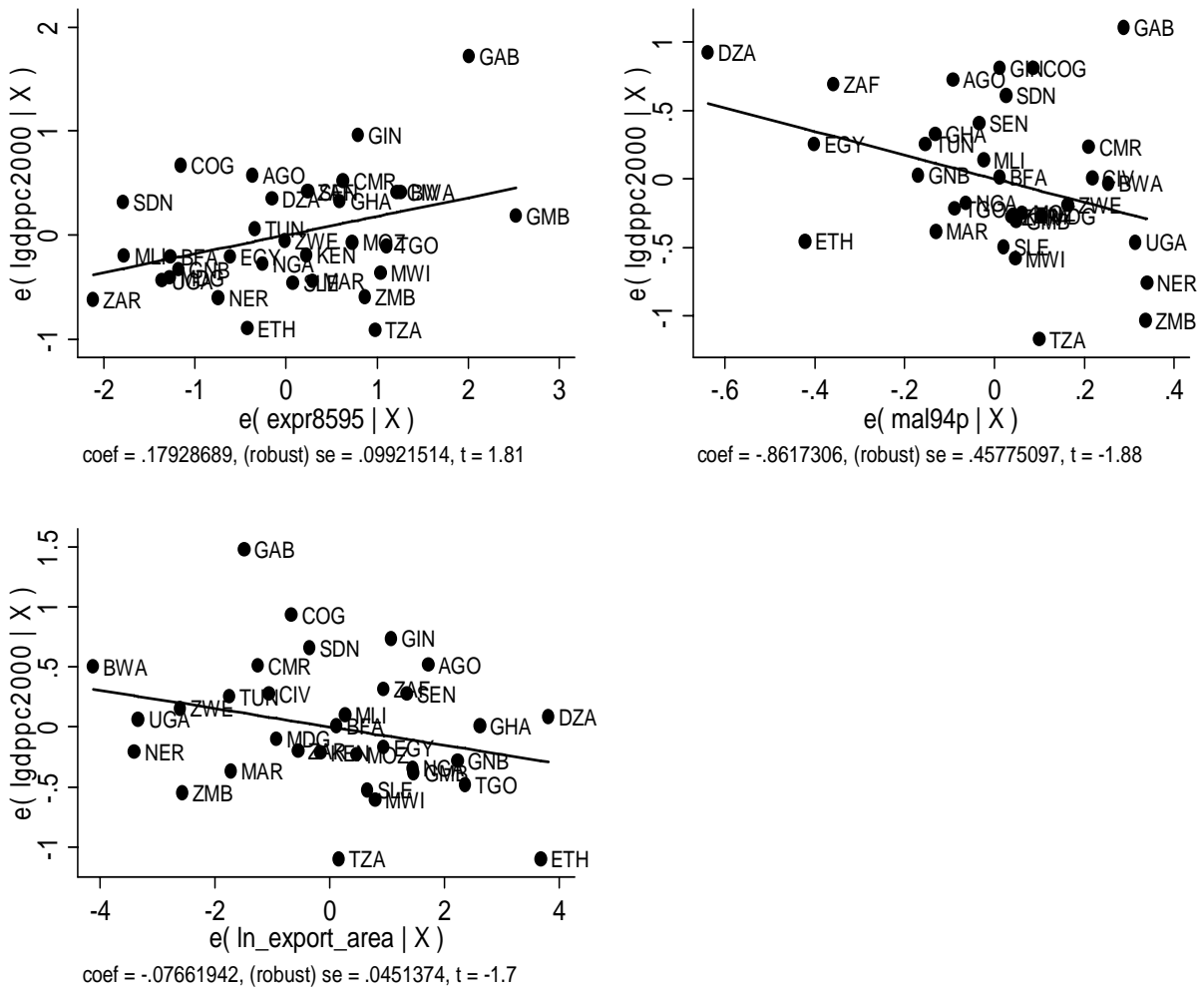


Table 1. Descriptive Statistics

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 $\log y_i = \lambda + \alpha MAL_i + \beta INS_i + \gamma SLVX_i + \varepsilon_i$				
Dependent Variable	Log per Capita GDP in 2000			Growth during 1960 – 2000
	OLS estimate obs= 33 (1)	2SLS estimate obs= 27 (2)	LIML Fuller estimate obs= 27 (3)	2SLS estimate obs= 27 (4)
Malaria Risk (MAL_i)	-0.86* (0.4576)	-4.19** (2.105)	-3.3** (1.758)	-0.04* (0.0244)
Expropriation Risk in 1985 to 1995 (INS_i)	0.18* (0.0992)	0.29 (0.6543)	0.16 (0.5251)	0.004 (0.0049)
Log total slave exports normalised by land area ($SLVX_i$)	-0.08* (0.0451)	0.39 (0.3043)	0.25 (0.2505)	0.002 (0.0033)
Log per capita income in 1960 R^2	0.59			-0.005 (0.0049)
Hansen J test (p)		0.92	--	0.63
Hausman/Sargan test (p)		--	0.71	--
Cragg-Donald test (p)		0.97	--	--
Additional Controls	--	LPD _i , IDC _i		
Instruments		ME, LSM, ADC, IODC, SDC, RDC		
Panel B: The First Stage Regressions				
Dependent Variables	MAL_i obs= 27 (1)	INS_i obs= 27 (2)	$SLVX_i$ obs= 27 (3)	
Malaria Ecology (ME_i)	0.02** (0.0080)	-0.03 (0.0277)	0.18** (0.0863)	
Log Settler Mortality (LSM_i)	0.12* (0.0663)	-0.08 (0.2906)	0.63 (0.6501)	
Log Population Density in 1500 (LPD_i)	0.09 (0.0745)	-0.26 (0.3741)	1.64* (0.8451)	
Interior Distance (IDC_i)	-0.00004 (0.00009)	-0.002*** (0.0007)	-0.001 (0.0022)	
Atlantic Distance (ADC_i)	-0.07 (0.0588)	-0.12 (0.2149)	-0.36 (0.6597)	
Indian Distance ($IODC_i$)	-0.02 (0.0454)	-0.13 (0.1680)	0.24 (0.6589)	
Saharan Distance (SDC_i)	0.12 (0.0969)	-0.55* (0.3154)	2.2* (1.162)	
Red Sea Distance (RDC_i)	-0.18* (0.0912)	0.19 (0.3920)	-1.6* (0.8680)	
R^2	0.82	0.58	0.64	
F-stat	54.75	6.65	3.88	
Panel C: Instrument Redundancy tests				
Instruments tested	ME	LSM	IDC, ADC, IODC, SDC, RDC	
LM test statistic	7.7	6.92	19.57	
p-value	0.05	0.07	0.08	
Degrees of freedom	3	3	12	

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. Fuller's modified LIML estimator with $\alpha = 1$ (correction parameter proposed by Hausman et al., 2005) is used in column 3, panel A. 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 test statistic follows F-distribution under the null with degrees of freedom = N-L, L1 (N-number of observations, L-total instruments, L1-excluded instruments). 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. The endogenous regressors are MAL_i , INS_i , and $SLVX_i$. The abbreviations used in the table are ME_i : Malaria ecology; LSM_i : Log settler mortality; LPD_i : Log population density in 1500; IDC_i : Interior Distance; ADC_i : Atlantic Distance; $IODC_i$: Indian Ocean Distance; SDC_i : Saharan Distance; and RDC_i : Red Sea Distance.

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

Dependent Variable	Log per capita GDP in 2000						
	2SLS estimate obs= 27 (1)	2SLS estimate obs= 27 (2)	2SLS estimate obs= 25 (3)	2SLS estimate obs= 25 (4)	2SLS estimate obs= 27 (5)	2SLS estimate obs= 27 (6)	2SLS estimate obs= 25 (7)
Malaria Risk (MAL_i)	-2.38** (0.9327)	-3.6** (1.887)	-3.45** (1.632)	-1.95* (1.188)	-3.35** (1.425)	-6.3 (10.70)	-0.26 (3.363)
Expropriation Risk in 1985 to 1995 (INS_i)	0.36 (0.5166)	0.33 (0.5721)	0.51 (0.7613)	0.61 (0.6554)	0.29 (0.5764)	1.89 (5.629)	0.52 (0.5102)
Log total slave exports normalised by land area ($SLVX_i$)	0.11 (0.1475)	0.29 (0.2864)	0.33 (0.2358)	0.13 (0.2306)	0.24 (0.1989)	0.85 (2.031)	0.12 (0.1705)
Hansen J test (p)	0.53	0.94	0.93	0.64	0.53	0.97	0.28
Additional Controls	LPD_i, IDC_i				$LPD_i, IDC_i,$ Rain	$LPD_i, IDC_i,$ Rain, Humidity	$LPD_i, IDC_i,$ Rain, Humidity, frost
Instruments	Replacing ME by Rain	Replacing ME by Humidity	Replacing ME by Frost	Replacing ME by Rain, Humidity, and Frost	ME, LSM, 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. 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. The endogenous regressors are MAL_i , INS_i , and $SLVX_i$. The abbreviations used in the table are ME_i : Malaria ecology; LSM_i : Log settler mortality; LPD_i : Log population density in 1500; IDC_i : Interior Distance; ADC_i : Atlantic Distance; $IODC_i$: Indian Ocean Distance; SDC_i : Saharan Distance; and RDC_i : Red Sea Distance.

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

Dependent Variable	Log per capita GDP in 2000		
	2SLS estimate obs= 27 (1)	2SLS estimate obs= 27 (2)	2SLS estimate obs= 27 (3)
Malaria Risk (MAL_i)	-2.36*** (0.6808)	1.43 (1.654)	-3.99** (1.729)
Expropriation Risk in 1985 to 1995 (INS_i)	-0.19 (0.4147)	0.23 (0.2615)	0.07 (0.3751)
Log total slave exports normalised by land area ($SLVX_i$)	0.09 (0.0915)	-0.03 (0.1034)	0.36 (0.2436)
Hansen J test (p)	0.28	0.09	0.92
Additional Controls	LPD _i , IDC _i		
Fixed Effects	Coloniser Fixed Effects	Region Fixed Effects	Legal Origin Fixed Effects
Instruments	ME, LSM, 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. The endogenous regressors are MAL_i , INS_i , and $SLVX_i$. The abbreviations used in the table are ME_i: Malaria ecology; LSM_i: Log settler mortality; LPD_i: Log population density in 1500; IDC_i: Interior Distance; ADC_i: Atlantic Distance; IODC_i: Indian Ocean Distance; SDC_i: Saharan Distance; and RDC_i: Red Sea Distance.

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

Dependent variable	Log per Capita GDP in 2000							
	2SLS estimate obs= 27 (1)	2SLS estimate obs= 27 (2)	2SLS estimate obs= 26 (3)	2SLS estimate obs= 26 (4)	2SLS estimate obs= 19 (5)	2SLS estimate obs= 27 (6)	2SLS estimate obs= 11 (7)	2SLS estimate obs= 26 (8)
MAL_i	-3.29** (1.464)	-3.2** (1.496)	-1.36* (0.8251)	-2.39*** (0.8453)	-2.31*** (0.8267)	-3.22 (2.199)	-1.51*** (0.4838)	-2.22*** (0.6315)
INS_i	0.20 (0.4987)	0.32 (0.5814)	0.43 (0.4931)	0.02 (0.5876)	0.33 (0.4141)	0.47 (0.6639)	0.06 (0.0849)	0.04 (0.1488)
$SLVX_i$	0.25 (0.1943)	0.29 (0.2359)	-0.01 (0.1422)	0.09 (0.1216)	0.16 (0.1341)	0.32 (0.3083)	-0.05 (0.0491)	0.09 (0.0694)
Hansen J test	0.67	0.61	0.08	0.41	0.45	0.84	0.95	0.42
Control Variables	LPD _i , IDC _i							LPD _i
Additional Covariates	Mining	Ethnic Fractionalisation	Catholicism	Islam	Gini Coefficient	Foreign Aid	Schooling in 1900	Trade Share
Instruments	ME, LSM, ADC, IODC, SDC, RDC							All Instruments plus CONST and IDC _i

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). The endogenous regressors are MAL_i , INS_i , and $SLVX_i$. The abbreviations used in the table are ME_i: Malaria ecology; LSM_i: Log settler mortality; LPD_i: Log population density in 1500; IDC_i: Interior Distance; ADC_i: Atlantic Distance; IODC_i: Indian Ocean Distance; SDC_i: Saharan Distance; and RDC_i: Red Sea Distance.

Table 6. Alternative Measures and Influential Observations tests

Dependent Variable	Log per capita GDP in 2000				
	2SLS estimate obs= 27 (1)	2SLS estimate obs= 25 (2)	2SLS estimate obs= 27 (3)	2SLS estimate obs= 25 (4)	2SLS estimate obs= 23 (5)
MAL_i	-4.08** (1.893)	-3.82*** (1.423)	-3.97* (2.262)	-2.72*** (0.7063)	-3.92** (1.793)
INS_i			0.42 (0.7933)	-0.03 (0.2977)	0.11 (0.3580)
Rule of Law Index	0.17 (0.7219)				
Executive Constraint		-0.19 (0.2040)			
$SLVX_i$	0.37 (0.3051)	0.29 (0.1846)		0.13 (0.0969)	0.25 (0.1990)
Log total slave exports normalised by population			0.48 (0.4206)		
Hansen J test (p)	0.87	0.47	0.99	0.32	0.79
Controls	LPD _i , IDC _i				
Omitted Influential Outliers				ETH, GAB	DZA, ETH, GAB, ZMB
Instruments	ME, LSM, 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. 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. The endogenous regressors are MAL_i , INS_i , and $SLVX_i$. The abbreviations used in the table are ME_i: Malaria ecology; LSM_i: Log settler mortality; LPD_i: Log population density in 1500; IDC_i: Interior Distance; ADC_i: Atlantic Distance; IODC_i: Indian Ocean Distance; SDC_i: Saharan Distance; and RDC_i: Red Sea Distance.

Table 7. Robustness with Nunn's Specification and Data

Dependent Variable	Log income in 2000								
	obs = 52 (1)	obs = 48 (2)	obs = 27 (3)	obs = 27 (4)	obs = 46 (5)	obs = 27 (6)	obs = 48 (7)	obs = 43 (8)	obs = 25 (9)
MAL _i		-1.2*** (0.4443)	-1.67*** (0.5749)	-1.00* (0.5861)	-1.39*** (0.4587)	-2.6* (1.611)	-1.42** (0.6328)		
INS _i			-0.04 (0.0864)	0.12 (0.1718)					0.58 (0.7179)
SLVX _i	-0.20*** (0.0429)	-0.05 (0.0624)	0.05 (0.0749)	-0.05 (0.0721)	-0.04 (0.0567)	0.11 (0.1403)	-0.04 (0.0629)	-0.34 (0.2126)	
Pre-colonial state development					-0.26 (0.4171)				
Rule of Law						-0.77 (0.7163)			
Ethnic fractionalisation							0.37 (0.7170)		
Coloniser fixed effects	YES	YES	YES	YES	YES	YES	YES	YES	YES
Sargan test	0.29	0.20	0.13	0.19	0.47	0.57	0.18	0.70	Exact Identification
Controls								Frost	Frost
Instruments	ADC, IODC, SDC, RDC	ME, ADC, IODC, SDC, RDC	ME, LSM, ADC, IODC, SDC, RDC	Humidity, LSM, ADC, IODC, SDC, RDC	ME, ADC, IODC, SDC, RDC	ME, LSM, ADC, IODC, SDC, RDC	ME, ADC, IODC, SDC, RDC	ADC, IODC, SDC, RDC	LSM

Notes: ***, ** and * indicates significance level of 1%, 5% and 10% respectively against a two sided alternative. Figures in the parentheses are cluster standard errors (except for column 1) and they are robust to arbitrary heteroskedasticity and arbitrary intra-group correlation. Coloniser fixed effects are dummies representing colonial origin. The dependent variable is from Nunn who use Maddison's figures for per capita GDP in 2000. The endogenous regressors are MAL_i, INS_i, SLVX_i, and Rule of Law. The abbreviations used in the table are ME_i: Malaria ecology; LSM_i: Log settler mortality; IDC_i: Interior Distance; ADC_i: Atlantic Distance; IODC_i: Indian Ocean Distance; SDC_i: Saharan Distance; and RDC_i: Red Sea Distance.

Table 8. Malaria and National Savings

The Model $\left(\frac{S}{Y}\right)_i = \zeta + \vartheta \text{MAL}_i + \rho \log y_i + \zeta_i$				
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*** (3.674)	-12.29** (4.997)	-15.22*** (3.923)	-12.56** (5.248)
$\log y_i$			2.58 (2.069)	2.96 (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. The endogenous regressor is MAL_i . The abbreviations used in the table is ME_i : Malaria ecology.

Data Appendix

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

Log income in 2000: Nunn (2008), originally from Maddison (2003).

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.

Pre-colonial state development: Nunn (2008).

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 (LPD): Acemoglu et al. (2001).

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.

Corruption: ICRG, PRS dataset.

Sachs and Warner openness: Sachs and Warner (1997).