Long-term exposure to malaria and violence in Africa

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Abstract

WE NEED TO DISCUSS ON THE ABSTRACT: WHAT WE WANT TO EMPHASIZE? HOW MUCH DO WE WANT TO EMPHASIZE GENETIC IMMUNITIES IN THE PAPER? This paper explores the existence of a link between the long-term exposure to malaria and the frequency of civil conflicts in Africa. Using geographically disaggregated data at the level of grid cells the analysis provides empirical evidence for a hump-shaped relationship between the long-run stability and force of malaria transmission and the incidence of civil violence. In line with epidemiological predictions on the role of acquired immunities, cells that are characterized by intermediate malaria exposure exhibit higher conflict incidence than cells with very low or very high malaria exposure. We explore the role of the scale up in anti-malaria policy after 2005 in the context of the Roll Back Malaria program. The result provide suggestive evidence that these health interventions reduce the incidence of civil violence but only in areas where adults lack acquired immunities to malaria.

JEL-classification: D74, J1

Keywords: Malaria; Civil Violence; Economic Policies, Cell-level Data; Africa; Immunities.

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1 Introduction

Diseases and civil violence are regarded major impediments for development, particularly in the poorest regions of the world. Surveys from several African countries document that the fear of diseases (with malaria being the most frequently cited) is the main risk factor as perceived by individuals, followed by shortages of food and exposure to violence.[1] Civil violence constitutes the most common type of conflict events over the past fifty years, accounting for the largest number of conflict-related casualties. More than a third of countries in Sub-Saharan Africa have experienced medium and large scale civil conflicts in the last twenty years. Most African countries are plagued by recurrent violence at a smaller scale, involving predation and looting of local communities. While rarely making it to the international news and passing largely unnoticed, these violent events in fact involve a substantial death toll and entail poverty and underdevelopment.[2]

In this paper we provide a first empirical assessment of the largely unexplored role of long-term exposure to malaria for the likelihood of local, small-scale civil violence on the disaggregated level in Africa. Malaria represents the greatest threat for human health in Africa over the last millennia.[3] Labelled "Humanity's Burden" by epidemiological historian Webb (2009), malaria has been estimated to have killed half of the humans that ever lived (Whitfield, 2002). Still today, malaria is responsible for a large death toll in Africa, with recent estimates by Murray et al. (2012) suggesting that malaria mortality is larger than previously estimated, especially among adults. Their evidence documents a steady increase in malaria deaths after 1980 in Africa, with a peak of about 1.5 million deaths in 2004.[4] Since the launch of the "Roll Back Malaria" program by the WHO at the end of the 1990s, the effort and economic resources devoted to malaria control has been increased substantially, and private donors joined in this endeavour in the second part of last decade. Since 2005 substantial

[1] See, for instance, the Parima-Study, Doss et al. (2008) and McPeak et al. (2012).

[2] According to the Armed Conflict Location and Event Data Project, which constitutes the main data source in this paper as described in Section 4, small scale localized violence has been estimated to be responsible for the death of at least 50-70,000 people per year.

[3] Evolutionary geneticists have recently claimed that malaria affect Africans since 100,000 years in its mild form vivax and since at least 10,000 years in the deadly variant falciparum. As Webb (2009) puts it "[Malaria is] a primordial companion of our distant protohuman ancestors and an even earlier companion of the chimpanzees from which we branched off six or seven million years ago".

[4] Malaria is estimated to have caused 200 million clinical cases worldwide in 2013. See also `http://www.who.int/mediacentre/factsheets/fs094/en` and `http://www.cdc.gov/malaria/about/facts`.

interventions in terms of prevention (mosquito nets), control (insecticide spraying) and treatment (particularly with artartemisinin) have been considered responsible for a change in the trend of malaria mortality and for a sizable reduction in the number of deaths particularly among adults, see Murray et. al. (2012) and Bhatt et al. (2015) among others (see also the discussion in Section 3).

Malaria can affect the likelihood of civil violence by directly affecting the opportunity cost of violence and/or the ability to fight and the risk of being predated. In theory, lower long-term orientation and risk aversion reduces the opportunity cost of getting involved in activities with high short-run gains but high risk, such as predation and conflict, as compared to activities that pay off in the long run, such as investment and production. In this respect poor health and high mortality can tilt the trade-off faced by individuals in a standard production-predation model *a la* Skaperdas (1992), Hirshleifer (1995) or Grossman (2001). In a dynamic perspective, a lower future orientation increases defection in repeated strategic interactions, thereby reducing the likelihood of cooperation and the peaceful resolution of conflicts of interests, see, e.g., Dal Bo and Frechette (2017). Increasing evidence supports the view that individuals facing higher mortality and poor health tend to be less future oriented and less risk averse.[5] Falk et al. (2017) provide cross-country evidence for the existence of a robust negative correlation between life expectancy and individual risk aversion and patience. A cursory exploration of the link between long term malaria exposure and long-term orientation across countries delivers an even more intricate relationship. While on average, individuals living in countries with low exposure to malaria, proxied by the index of the stability of malaria transmission described below, appear to be more oriented towards the future than individuals in countries with high malaria exposure, the lowest levels of long-term orientation are observed for intermediate levels.[6]

Poor health can also weaken or kill individuals, however, thereby curbing the ability of the affected groups to fight and predate. At the same time, this may also increase the exposure to predation by others. In spite of these arguments fairly little is known empirically about the role of exposure to

[5]See, e.g., Becker and Mulligan (1997) for theoretical foundations of the link between health and time preference. Empirically, poor health has been associated with riskier behavior and increased involvement in activities that pay-off in the short run. The link between future orientation and health and mortality has been pointed out in specific empirical studies. For instance, exposure to the accident at the nuclear plant in Chernobyl in 1986 have been related to increased death rates for other risky behaviors like increased smoking, drinking and car speeding, the unsafe sex and the spread of HIV, see Lorentzen, McMillan, and Wacziarg (2008). Scattered experimental evidence confirm this patterns.

[6]See also Figure A1 in the Supplementary Appendix.

human diseases for conflicts. Moreover, the predicted direct effect of malaria on violence is therefore a priori ambiguous and whether and in which direction malaria affects civil violence is an open empirical question.

In light of the fact the most relevant demographic group involved in civil conflicts, violence and predation is (male) adults rather than children, our empirical strategy closely builds on evidence in malaria epidemiology that documents a non-linear, hump-shaped, relationship between the exposure to malaria and its health implications for adults (in terms of incidence and mortality). In particular, malaria is a disease caused by infections with plasmodium parasites, which can be transmitted only through a non-human vector: female anopheles mosquitos. Epidemiologists classify malarial areas in terms of the stability of pathogen transmission, which crucially depends on the characteristics of the vectors resident in the area and on the local bio-climatological conditions. Areas characterized by higher stability of malaria transmission imply less interrupted cycles of infections between humans and mosquitos and, therefore, higher inoculation rates (i.e., unconditional probability of being bitten by an infected mosquito).[7] A specific feature of the human physiological response to malaria is the development of protective immunities that are acquired in response to intense and uninterrupted exposure to the parasite over the years. As a result, individuals who face repeated infections early in life and survive to adulthood only experience mild malaria symptoms and low mortality risk in the face of new infections. As a consequence, in areas with highly stable malaria transmission the burden of exposure to malaria is predominantly borne by children whereas in areas with low transmission stability also adults are heavily affected. This so-called "age peak shift" phenomenon, implies that the incidence and severity of malaria among adults displays a hump-shaped relationship with the stability of malaria transmission.

The empirical strategy to explore the effect of long-term exposure to malaria on civil violence builds on this epidemiological feature. An intention-to-treat framework uses data for the "predicted", rather than actual, stability of malaria transmission. This measure is based on information about the dominant mosquito vectors in a given location and about local variation in temperatures and precipitation thresholds reflecting the suitability for vector reproduction. This index of predicted malaria stability, which has been developed by epidemiologists, has two relevant advantages for our

[7]At one extreme, when the cycle of infection is uninterrupted and stable across years, infections are endemic. At the opposite extreme, when when transmission intensity changes from year to year, infections are infrequent and take the form of epidemics (i.e. abnormal spikes in infections).

purposes. First, being based on geographical and bio-climatological features, the index is not directly affected by conflicts, contrary to malaria incidence or inoculation rates. Second, the index displays substantial regional variation across Africa. This allows us to exploit within country variation. In contrast, no systematic data on the malaria incidence, or inoculation rates among adults is available at disaggregated levels for the whole of Africa.[8] A main caveat of this approach is that the analysis is in reduced form and exploits cross-sectional variation. The main empirical threat thus comes from omitted variables. The specific non-monotonic relationship between malaria transmission and the susceptibility of adults for severe infections can nonetheless be exploited to disentangle the role of malaria from alternative determinants.

The empirical results deliver evidence for a hump-shaped association between long-term exposure, in terms of predicted malaria stability, and the occurrence of localized civil violence on the basis of grid-cell data for the whole of Africa. The association is highest for intermediate levels of malaria stability, consistent with the notion that malaria risk for adults is highest in these areas. These results are robust to the inclusion of a large set of location-specific covariates, including geo-climatological conditions, information on location and distances, natural resources, ethnic composition of the population, population density and proxies for economic development and urbanization, as well as to the inclusion of country fixed effects that subsume the role of country level factors such as institutions and colonial history.

These findings are confirmed by extensive robustness checks, including the use of information about the endemicity of malaria in the population around the year 1900. We also exploit variation in the recorded degree of innate (genetic) immunities to malaria and find that they attenuate the effect of malaria transmission on conflicts. The non-linearity of the effect is documented using both parametric and non-parametric estimates including flexible specifications. The patterns are confirmed also when using binary indicators to check whether the effect is indeed lower for high malaria transmission areas. The results hold for different measures of civil violence from different sources and different measures and types of violent conflicts both at the extensive and the intensive margin. Most of the action appears to take place in terms of battles (violent confrontations) and

[8] A large effort has been recently devoted to a systematic quantification of malaria incidence in adults but lack of data and problems of measurement errors remain serious challenges. As discussed in Sections 3 and 4 recent estimates of malaria incidence for adults are obtained from scattered survey data and meta analyses that are aggregated (sometimes using epidemiological models) to obtain country level estimates.

violence against civilians, while no robust patterns is detected in terms of riots and protests. Overall, the findings provide robust evidence of a non-monotonic effect of the malaria transmission on conflicts that is in line with the epidemiological facts and compatible with the view that high malaria risk for adults increase the likelihood of civil violence.

In a second step, we extend the analysis by exploring the role of anti-malaria policy interventions. This allows us to address two issues. A dampening, within cells, effect of anti-malaria policies on civil violence in areas with high malaria risk for adults would provide indirect evidence for the identification of the channel behind the reduced form estimation results and thus limit potential concerns about omitted variables or alternative mechanisms unrelated to malaria exposure. At the same time, this analysis sheds new light on the effectiveness of these policies above and beyond the health dimension. A potential concern for this analysis is that information on the extent of policy implementation at the local level may be subject to measurement error, which itself might be related to civil conflicts. Moreover, the implementation of such policies might be affected by outbreaks of civil violence.[9] To deal with these issues we implement a conservative strategy that exploits time variation in policy implementation. Policy coverage was negligible before 2005 and then sharply increased. The results of several exercises, including a difference-in-difference strategy, provide suggestive evidence that increases in policy coverage after 2005 were indeed associated with a reduction in civil violence, but only in the areas with high malaria risk for adults.

The remainder of the paper is structured as follows. Section 2 locates the contribution of this paper in the literature. Section 3 provides background information about malaria epidemiology that are relevant for the empirical strategy. Section 4 discusses the empirical strategy, the data and their sources. Section 5 presents the results and Section 6 concludes.

2 Literature

The project contributes to several strands of a rapidly growing literature in economics and political science that studies the geo-political, ethnic and economic determinants of civil conflicts. Substantial progress has been made during the last decade for a better understanding of the determinants of

[9] As discussed in Section 2, the epidemiological literature finds no evidence that policy implementation significantly related measures of local violence and conflicts. Still, the possibility that policies are affected by conflicts cannot be ruled out.

civil wars at the country level, mainly by exploring the role of country specific characteristics for the likelihood of civil wars. The existing findings document the relevance of variation in income and poverty, weak or non-democratic institutions, political instability and ethnic divisions across countries.[10] Ethnic polarization and genetic diversity have also been suggested as robust country specific determinants of civil conflicts.[11]

In spite of the theoretical predictions about the potential impact of mortality and health and the recurrent warnings issued by development practitioners and international organizations, there is little empirical evidence for the role of the exposure to pathogens for civil conflicts. The only existing systematic evidence for the relationship between the exposure to human pathogens and conflicts is presented by Cervellati, Sunde, and Valmori (2017) who exploit cross-country variation in the exposure to multi-host vector-transmitted, MHV, pathogens as empirical determinant of the likelihood of large scale civil wars.[12] The literature has nonetheless pointed out several limitations of cross-country analyses of civil violence, both in terms of empirical strategies available for identification and inference and for the exploration of the respective mechanisms. Exploiting disaggregated data allows us to implement a substantially refined empirical identification approach and for the exploration of the underlying channels and the existence of spatial spillovers in this paper.

The paper contributes to a recent literature that investigates the determinants of violence across grid cells at the sub-national level. Most of this work uses data from the Armed Conflict Location and Event Data Project, which offer a full coverage of violent events for the whole of Africa. Besley and Reynal-Querol (2014) document the persistence of historical conflicts across locations in Africa. Harari and La Ferrara (2016) investigate the role of weather in rain-fed agriculture. As discussed in further detail in Section 4, in terms of empirical set up the closest paper to ours is by Michalopoulos and Papaioannou (2016), who exploit cross-sectional variation in (post-colonial) country borders to

[10] See, e.g., Fearon and Laitin (2003) and Collier, Hoeffler, and Rohner (2009), among others.

[11] See Esteban, Mayoral, and Ray (2012) and Arbatli, Ashraf, and Galor (2015), respectively. Another strand of the literature has explored the role of short-term variation in weather conditions within countries to study the role of income shocks, see, e.g., Miguel, Satyanath, and Sergenti (2004), Couttenier and Soubeyran (2014) and Berman and Couttenier (2015) and the extensive surveys by Blattman and Miguel (2010) and Couttenier and Soubeyran (2015).

[12] As consequence of their specific features, the presence (or endemicity) of MHV pathogens in a country is crucially related to country-specific characteristics. Their global distribution is influenced little by trade and economic activities. No vaccines are generally available. In addition, their reliance on multiple non-human hosts makes these pathogens highly resistant to health campaigns and eradication policies, which provides a possibility to study the relation between disease exposure and civil war.

identify the role of the "scramble" for Africa by the European colonial powers.

Some recent contributions have explored the short-term, rather than the long-term, determinants of civil violence by exploiting exogenous within-cell variation over time. Berman, Couttenier, Rohner and Thoenig (2017) exploit yearly variation in commodity prices in mining areas and document a causal increase in struggles for the control of territories. Cervellati, Esposito and Sunde (2017) use panel data to study the short run effects of variation in malaria exposure by exploiting exogenous within-cell variation in short-term weather conditions that are suitable for temporary spikes in malaria transmission. Differently from the current paper, the empirical strategies in this strand of the literature exploit within cell variation over time by including cell specific fixed effects that absorb time invariant cell-specific characteristics (including long term malaria stability and population immunities). The approach developed in the current paper therefore takes a complementary approach as it does not exploit information on short term weather shocks but on cross-grid cells variation in the long-term exposure to malaria. The current project also provides novel evidence on the largely unexplored role of resistance of the population in terms of acquired immunities, and its implications for the role of anti-malarial policies.

Most of the existing epidemiological and economic literature has concentrated attention on the reverse direction of causality, of armed conflict on the prevalence of malaria in the population. Available studies that exploit a variety of techniques and data report mixed findings. A positive association between large scale civil conflicts and malaria has been documented for the case of Afghanistan, where the pathogen was reintroduced by the massive, war-related relocation of about 100,000 people (see Kolaczinski, 2005 and Gayer et al., 2007). Evidence by Montalvo and Reynal-Querol (2007) for a large panel of countries further documents that large scale civil wars tend to increase the spread of malaria when they are associated with the displacement of large masses of people and the establishment of refugee camps. Available studies by epidemiologists that look at localized conflicts using disaggregate data and geo-statistical models find mixed evidence for the effect of violent events on malaria parasitization (as measured by health surveys) and on the implementation of anti-malarial policies (see Messina et al., 2011 and Sedda et al. 2015). Large scale conflicts and, in particular, the displacement of large numbers of individuals tend to increase the prevalence of malaria, while the relationship between localized civil violence and the prevalence of malaria is unclear.

The project also indirectly relates to the literature on the role of health and mortality for comparative development. A number of studies has investigated the implications of the overall exposure

to human diseases and measures of health for human capital accumulation and development across countries.[13] The role of malaria for cross country development is still a matter of intense debate.[14] In the attempt to improve upon cross-country studies, some recent works explore the role of diseases for African development by exploiting disaggregate data as we do in this paper. Alsan (2015) studies the role of exposure to trypanosomiasis for the pre-colonial organization of economic activity in Africa. Esposito (2015) documents the role of group-specific genetic immunities to malaria for shaping the patterns of the African slave trade. Cervellati, Chiovelli and Esposito (2016) document the role of ancestral exposure to malaria for the emergence and persistence of African ethnicities. They find that long term exposure to malaria increases the number of ethnic groups in a given cell. Depetris-Chauvin and Weil (2017) explore the role of long-term exposure to malaria for pre-colonial development in Africa. Their estimates suggest that the effect of malaria on adult mortality in the past was larger than today. In terms of pre-colonial outcomes they find no significant effect of malaria on historical population density and development. Finally, Cervellati, Esposito and Sunde (2017) document a non monotonic effect of long-term exposure to malaria on local development as measured by night lights per capita at the cell level in Africa today. The results in this paper indirectly contribute to this literature by providing evidence for a potentially relevant but largely unexplored channel through which the long-term exposure to malaria and the associated emergence of immunities also affects Africa's development performance.

3 Malaria Epidemiology: Background, Facts and Implications

From an epidemiological perspective malaria is a peculiar disease in many dimensions. In this section we provide a brief description of some of the specificities of the malaria epidemiology are very important for the implementation of the empirical analysis in this paper.

[13] See, e.g., Acemoglu and Johnson (2007), Lorentzen, McMillan, and Wacziarg (2008) or Cervellati and Sunde (2015) among others. Other studies have investigated the role of mortality and demographic dynamics for long-term development. African countries are still mostly pre-transitional in terms of the demographic transition and some works have documented the role of exposure to pathogens and mortality for long run growth during the different phases of the demographic transition, see Cervellati and Sunde (2011).

[14] Early works by Gallup et al. (1999), and Sachs (2003) attributed a major role to malaria. Their conclusions have been qualified and questioned subsequently, see Weil (2010, 2014).

Background. Malaria is caused by several types of *plasmodium* parasites of which *falciparum* is the most deadly and most common in Africa. Malaria is a vector-transmitted disease. The transmission occurs through the female *Anopheles* mosquito, which requires blood meals for ovary development. Anopheles reproduction requires water reservoirs, where the eggs are laid the larvae develop and eventually emerge as adult mosquito. The reproduction cycle and its length crucially depend on the weather conditions.[15]

The life cycle of *Plasmodium falciparum* parasites is complex and takes place both within humans and within the mosquito (that serves as both reservoir host and vector). Biting an infected human and absorbing the parasite as gametocytes (i.e., in sexual forms) from the human blood starts a cycle of growth and sexual multiplication of the parasite inside the mosquito. The cycle continues upon transmission of infection through an injection directly in the blood of a new human host where the parasites develop and multiply asexually, first in the liver and later in the the red blood cells.

Stability of Malaria Transmission and Acquired Immunities. Following the seminal work by MacDonald (1956), malaria epidemiologists classify the exposure to malaria along a stable-unstable gradient (see also Hay et al., 2008, for a discussion of the evolution of the modeling and measurement of malaria stability). At the two extremes, the literature conceptually differentiates between areas with stable malaria transmission, which are characterized by uninterrupted cycles of transmission between humans and infected mosquitos, and areas where the transmission of malaria is unstable. As will be discussed below in Section 4, the degree of stability of malaria transmission is crucially related to the local bio-climatological environment.

Areas with higher malaria stability are characterized by higher inoculation levels (that is a higher probability of being bitten by an infected mosquito) and higher endemicity of the pathogen in the population. In high malaria stability areas the mortality rates in children are large but individuals surviving repeated infections develop effective immunities. Naturally acquired immunity to falciparum malaria protects millions of people routinely exposed to Plasmodium falciparum infection from severe disease and death. "Across sub-Saharan Africa where the disease is holoendemic, most people are almost continuously infected by P. falciparum, and the majority of infected adults rarely experience overt disease. They go about their daily routines of school, work, and household chores feeling essentially healthy despite a population of parasites in their blood that would almost univer-

[15]See, e.g., Bayoh and Lindsay (2003), Christiansen-Jucht et al. (2014) and Lyons et al. (2013) for details on the requirements for reproduction and Section 4 for details on the construction of the measure of predicted malaria stability.

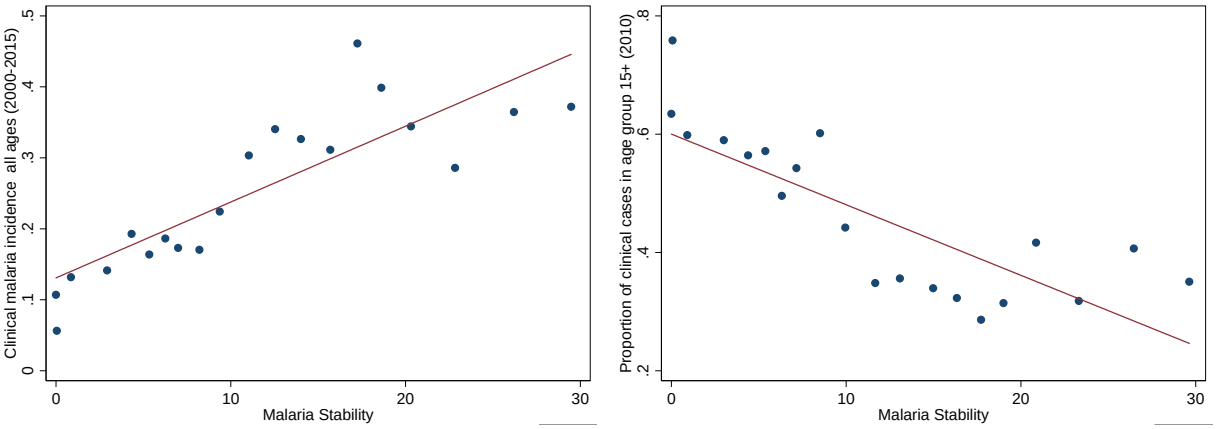
sally prove lethal to a malaria-naive visitor." (Doolan et al. 2009, p. 14.) A long term uninterrupted exposure to the pathogen is the primary driver of acquired immunities. In the words of Hay et. al. (2001) "when extrinsic development of the parasite is short (...) and when the vectors have a low mortality rate and bite humans frequently. Where such conditions are met and Plasmodium falciparum malaria transmission is stable, the prevalence of infection is high and endemicity is relatively insensitive to climatic changes. The constant high challenge to the local population stimulates strong immunity and a consequent decrease of clinical disease episodes among adults." The consequence is a greater resistance against malaria infections of adults.[16]

Areas with unstable malaria are, on the contrary, characterized by interrupted or even infrequent exposure to the pathogen. A specificity of these areas is that infections take the form of epidemics. An important point is that outbreaks of the disease in unstable transmission areas affect the population at large, including adults and can have devastating effects. According to the classic description of MacDonald (1957), "An epidemic is an acute exacerbation of disease out of proportion to the normal to which the community is subject. (...) Epidemics are common only in zones of unstable malaria, where very slight modification in any of the transmission factors may completely upset equilibrium, and where the restraining influence of immunity may be negligible or absent."

Age Patterns and Malaria Incidence in Adults. A key point for the purposes of our analysis is that the development of acquired immunities among the adult population crucially imply a changing age composition of clinical cases as a function of the stability of malaria transmission. MacDonald (1956) was one of the first to study the age distribution of malaria cases under varying transmission intensities. He emphasized that that the age peak of affected cases decreases with the level of malaria stability. Recent research on this so-called "peak shift" phenomenon has provided empirical quantifications of the shift in the age of the affected population as a function of malaria stability. Murray et al. (2012, p. 424) state that "the proportion of malaria deaths in adults is almost always more than 40 % (...) exceptions are sub-Saharan African countries with high malaria transmission." Recent evidence documents that the age peak shift pattern is particularly visible for cases of severe malaria. For instance, Griffin et. al. (2013) document that the share of severe cases

[16]Figure A2 in the Supplementary Appendix reproduces a figure from Langhorne et. al. (2008), which illustrates this in terms of the decline in the severity of infections with age due as a result of repeated infections and acquired immunity in endemic malaria areas. For individuals surviving to the age of 15, the probability of developing severe malaria is negligible and infections involve light symptoms or are even asymptomatic.

FIGURE 1: MALARIA STABILITY AND THE AGE PEAK SHIFT PHENOMENON



(a) Clinical Malaria Incidence - All Ages (b) Proportion of Clinical Cases With Age 15+

Note: The left Panel depicts the relationship between the stability of malaria transmission and clinical malaria incidence for individuals of all ages, averaged over the period 2000-2015. The right panel depicts the relationship between malaria stability and the proportion of all clinical cases that refer to the age group 15 and older. Both Figures are bin scatter plots, including a linear fit. Data sources: Kiszewski et al. (2004), Bhatt et al. (2015) and Griffin et al. (2013). See also Section 4 for details.

in children under 5 of age ranges from 60% to around 10% when passing from high to low stability of transmission. The opposite is true for the share of cases in individuals aged 15 or above, who account for a negligible fraction of cases in high stability areas and up to 60% in low transmission areas (with areas with intermediate stability of transmission displaying more balanced cases at all ages).

These specific features of malaria epidemiology have an important implication: the relationship between the level of malaria stability and the incidence of severe malaria infections (and, in the extreme, mortality) of adults is hump-shaped. On the one hand, the unconditional probability of inoculation of the pathogen is increasing in malaria transmission stability (ms). This can be seen from Figure 2(a), which plots clinical malaria incidence for individuals of all ages taken from Bhatt et al. (2015) against predicted malaria stability (labelled "Malaria Stability" in short) constructed by Kiszewski et al. (2004). On the other hand, conditional on inoculation, the probability of developing severe symptoms or even death for adults is decreasing in malaria stability, as illustrated by the data depicted in Figure 2(b).

As a consequence, the relationship between the total probability of severe symptoms or death

among adults and malaria stability is expected to be hump-shaped with a peak for intermediate malaria stability.[17] Figure 2 illustrates this pattern by depicting the relationship between the measure of predicted malaria stability and two distinct measures of adult mortality across countries in Africa. The left panel depicts the relationship between the predicted stability of malaria transmission with the recent estimates of adult mortality provided by Murray et. al. (2012).[18] Malaria is far from the only important source of mortality in Africa but it still represent a serious burden and a main determinant of adult life expectancy in Africa. The right panel illustrates the same underlying pattern in terms of the link between malaria stability and life expectancy at age 5 (i.e., excluding child mortality). Both graphs include a local polynomial that allows for a non-parametric fit of the data (with 95 percent confidence intervals) as well as a quadratic fit. In spite of the data limitation and the few observations available, the patterns document a hump-shape relation between malaria stability and adult mortality.

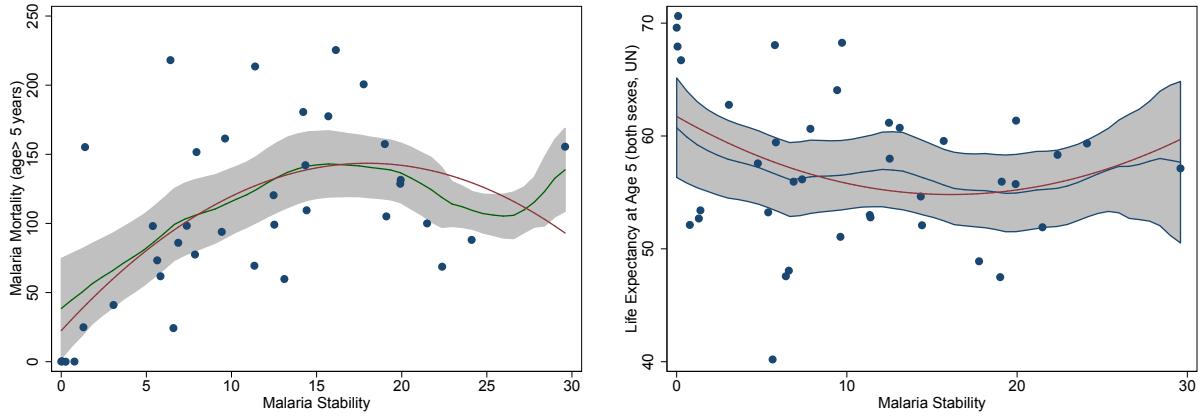
It is noteworthy that a reversal in malaria deaths and life expectancy occurs when malaria stability gets larger than 15 or 20. This is in line with the classification of areas in terms of the historical presence of the pathogen in the African population since all areas with a predicted malaria stability index above 15 are classified as endemic.[19] The high selective pressure from malaria also favored the spread of several types of genetic diseases that are malaria protective (examples include the sickle-cell disease, the Duffy-Antigen negative genotype, thalassemia, and glucose-6-phosphatase deficiency, among others). Differently from acquired immunities (that emerge over time and mostly protect adults) these innate immunities are transmitted from parents to children and tend to protect all age groups alike and therefore do not induce the age peak shift but do offer protection in the face of

[17] With $i(ms)$ denoting the unconditional probability of inoculation with the pathogen as a function of malaria transmission stability, ms, and $p(ms)$ denoting the probability of severe symptoms/death for adults conditional on inoculation, the total probability of developing severe symptoms is given by $s(ms) = i(ms) \times p(ms)$.

[18] The estimates by Murray et al. (2012), are the result of a large effort to provide systematic and comparable estimates of the evolution of malaria incidence among adults across countries. See section 4 for details.

[19] As discussed below, we use the alternative data from Lysenko and Semashko (1968), that classify categorial levels of endemicity in the African population around 1900, as robustness check. The areas with a predicted malaria stability index by Kiszewski et. al. (2004) above 15 are classified as mesoendemic, hyperendemic or holoendemic. Areas with malaria stability above 25 are classified as hyperndemic and holoendemic. See also the bin scatter plot reported in Figure A3 in the Appendix. The malaria endemicity variable is categorial with: 1 epidemic; 2 hypoendemic; 3 mesoendemic; 4 hyperendemic; and 5 holoendemic.

FIGURE 2: MALARIA STABILITY AND ADULT MORTALITY (AGED 5 OR ABOVE)



Note: The left Panel depicts the relationship between the stability of malaria transmission and the estimated mortality for individuals age 5 or above in the year 2000 across countries in Africa. The right panel depicts the relationship between malaria stability and life expectancy at age 5 in year 2000 in Africa. Both Figures plot local polynomial fits (with 95 percent confidence intervals) and a quadratic fit. Data sources: Kiszewski et al. (2004), Murray et al. (2012), and United Nations, World Population Prospects (2015 Revision). See also Section 4 for details.

increasing malaria stability and inoculation rates.[20]

Time Patterns, Treatment and Control. The specificities of malaria in terms of transmission and reproduction have shaped attempts to contain and eradicate malaria since the end of WWII. Eradication policies historically focused on preventing the vector from reproducing, by eliminating lentic water reservoirs by ways of draining swamps or eradicating the vector, e.g., by spraying insecticides like DDT. In many developed countries, like Europe, these policies were successful and malaria has been declared eradicated (although the vectors are still present and new warnings have recently been issued on the prospects of a reintroduction of the pathogen through globalization and migration). Similar attempts of eradication have been less successful in developing countries for a variety of reasons (including a reduced policy effort starting from the 1980's, the increase in the size of population at risk of malaria inoculation, and the increasing resistance of the plasmodium pathogens to the drugs traditionally administered during the last decades in Africa).

Since the late 1990's efforts to fight the disease have been increased under the Malaria Roll

[20]For robustness we also exploit information on genetic immunities to explore whether the effect of increases in malaria stability is moderated by higher genetic immunities.

Back program of the WHO. With the support of private donors and international organizations, this has led to a rapid scaling up of malaria control policies in Africa by the mid 2000's. These attempts to control malaria involved a combination of several measures, including the spraying of habitations with insecticides, distributing bed nets that have been treated with insecticides, or the administration of anti-malarial drugs to treat malaria infections. No successful eradication of Malaria has been achieved yet in Sub Saharan Africa where, in spite of the large recent efforts for treatment and control, the prospects of eradiction are still considered limited even over the medium and long term. Currently, no licensed vaccine is available.[21]

According to estimates by Murray et al. (2012), annual malaria deaths at the global level have steadily increased from below one million in 1980 to a peak of about 1.9 millions in the early 2000s. After 2005, malaria deaths eventually displayed a decrease of about 30 percent due to the systematic intervention campaigns.[22] A finding of their study that is particularly relevant in this context is that the trend reversal in mortality for adults is particularly strong in areas with intermediate and low malaria stability. The crucial role of policies for the reversal in the mortality trend is also confirmed by Bhatt et al. (2015) who provide a first attempt to map the implementation of anti-malarial policies for the whole of Africa.

4 Empirical Strategy and Data

4.1 Empirical Strategy

The analysis is based on disaggregate data for the entire African continent at the grid-cell level as primary units of observation. The estimation framework is based on the insights from the malaria epidemiology background briefly presented in Section 3. In light of local geo-climatological conditions and the the prevalence of immunity in the local population, the role of exposure to malaria can also be expected to be confined to small geographical areas, rather than entire countries. Moreover, as discussed above the relationship between malaria stability and malaria incidence and mortality in

[21] See `http://www.who.int/mediacentre/factsheets/fs094/en/` for an overview of anti-malarial policies, their effectiveness, and limitations. The most recent overview on the spread of malaria can be found in the WHO's World Malaria Report 2016 (`http://www.who.int/malaria/publications/world-malaria-report-2016/report/en/`).

[22] According to the authors, private funding specifically dedicated to malaria control, which has risen from around 150 million dollars 2000 to more than 1.2 billion dollars in 2008, was particularly important for this trend reversal.

adults is hump-shaped. We therefore implement the following empirical framework,

$$Conflict_{ic} = f(Malaria\ Stability_{ic}) + X'_{ic}\beta + \zeta_c + u_{ic}$$

where $Conflict_{ic}$ represents a measure of incidence of civil conflict in cell i in country c. The question of interest for the purpose of this paper is the link of conflicts to malaria exposure, measured by the predicted stability of malaria transmission, labelled *Malaria Stability* in short and the non linear shape of this relationship, captured by the function $f(\cdot)$. Other, cell-specific factors related to geography, climate, natural resources, location and distances, population, development and urbanization that might influence conflict, are accounted for by the inclusion of corresponding control variables, reflected in the vector X_{ic}.

Compared to a cross country framework, an analysis based on disaggregate sub-national data has the advantage of being able to account for all (observable and unobservable) country-specific features that might affect the likelihood of civil conflicts above and beyond the exposure to pathogens, subsumed in the vector ζ_c. This includes, in particular, country-specific institutions, policies and national or colonial history, and the composition of the population, which are confounders that are difficult to account for in cross-country studies. This provides a substantial improvement in terms of econometric identification relative to country-level data, following a similar approach as Michalopoulos and Papaioannou (2016). To insure external validity, we also follow the spirit of large cross-country studies and consider all locations (grid-cells) on the entire continent of Africa. The data sources discussed below are suited for this purpose as they provide disaggregate information on the cell level for different measures of long-term exposure to malaria, genetic immunities, and proxies for the implementation of anti-malarial policies. Compared to cross-country data this empirical strategy provides a balance between the external and internal validity.

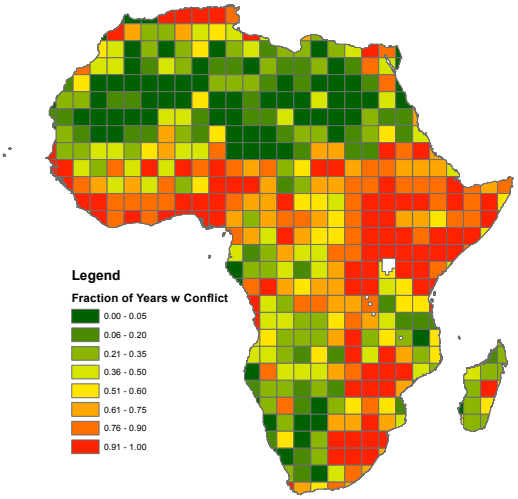
Disaggregate data also have limitations that need to be addressed in the empirical analysis. A relevant issue is about the existence of spatial dependencies in the data. The baseline analysis is conducted with 2.5×2.5 degrees cells, where one degree corresponds to about 110 kilometers at the equator. The use of this, relatively large, grid cells has the advantage of minimizing overlap and spatial dependencies. To further account for this issue, the empirical estimates also explicitly allows for standard errors that are robust for spatial dependencies (Conley standard errors).

4.2 Data

This section presents the main variable of interest. The variables, their coding as well as their respective data sources are described in detail in Tables 8 and 9.

Civil Violence. As a measure of civil violence, we use data from the Armed Conflict Location and Event Data Project (ACLED, Version 7 1997-2016), which represents the most comprehensive public collection of disaggregated data on violent events available for developing states. The data set is particularly suited for the purpose of this analysis as it provides detailed information on the geographic location of a violent event in terms of latitude and longitude. As baseline outcome variable of interest, we look at the fraction of years over the period 1997-2016 where at least one violent event took place in a given cell. Figure 3 depicts the spatial distribution of violent events in Africa.

FIGURE 3: VIOLENT EVENTS



Note: Spatial distribution of fractions of years with at least one violent conflicts over the period 1997-2016 (in $2.5^o \times 2.5^o$ cells). Data source: Armed Conflict Location and Event Data Project, ACLED (version 7, 2016).

We also explore the sensitivity of the results with respect to different coding of conflicts, including the total number of events and the intensity of violence in terms of fatalities, as well as different types of conflicts in terms of open confrontations between armed groups, violence against civilians and riots and protests. As robustness we also use alternative conflict data from the UCDP Georeferenced Event Dataset (2016) for which we code the fractions of years in conflicts and the total number of conflicts as measures of conflict.

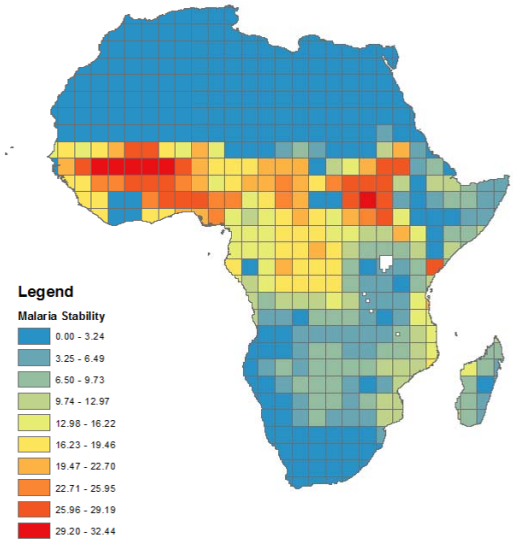
Predicted Malaria Stability. The analysis relies on the availability of a measure of the long-term exposure to malaria that is rooted in the specific features of the epidemiology of malaria. As baseline information on long-term malaria exposure we use the force of malaria transmission and stability index, which has been constructed by Kiszewski et al. (2004). This index (henceforth labelled "Malaria Stability") provides a measure of predicted malaria stability as derived from vector based models (following the seminal work by MacDonald, 1957) rather than actual malaria stability as proxied by estimated inoculation rates or prevalence of the pathogen in the population. An important advantage of this measure of malaria transmission stability that it does not exploit information about the distribution of pathogens in the human population or other relevant covariates (like population density) that are potentially directly related to human activity and conflicts. In fact, the index does not even exploit information about whether the pathogen is present or endemic in a given location, but rather about the *potential* for its transmission.

The index is built using information on the type of mosquito vectors that are prevalent in a location as well as their biological characteristics (like the typical share of blood meals from humans, the longevity of the mosquito, etc). Information on the vectors is used together with information about the average climatological conditions that favor the spread and infection of the vector according to vector based epidemiological models.[23] The index offers fine grained disaggregate information on the predicted stability of transmission in a geographic location. The data underlying this index are available with a high level of precision and geographic disaggregation and the resulting index provides full coverage of all locations without the need for spatial interpolation. Figure 4 depicts the distribution of the long-term exposure to malaria in Africa in terms of the Malaria Stability Index.

To check the robustness of the findings and the persistence of the effect we use several alternative data sources. In particular, we use information on the spatial distribution of historical malaria

[23]When constructing this index, Kiszewski et al. (2004) assigned to each region a dominant vector of *Anopheles* mosquitoes (for countries with different dominant vectors, mosquitoes were assigned to sub-regions), and used this information together with the respective biting rates of humans of the prevalent vectors and the specific bio-climatological conditions in each location to measure the force of malaria transmission and stability. This involves a positive (although non linear role) of temperature in the previous month and a threshold level of precipitation. The climatic data employed are averages of monthly observations between 1901 and 1990. The final index is indirectly informative on the number of months that are predicted to be compatible with malaria transmission in each location. In our sample the Malaria Stability index ranges from 0 (absence of a sustainable environment for malaria transmission) to about 34 (high potential for malaria transmission).

FIGURE 4: STABILITY OF MALARIA TRANSMISSION



Note: Spatial distribution of malaria ecology index (in $2.5^o \times 2.5^o$ cells). Data source: Kiszewski et al. (2004).

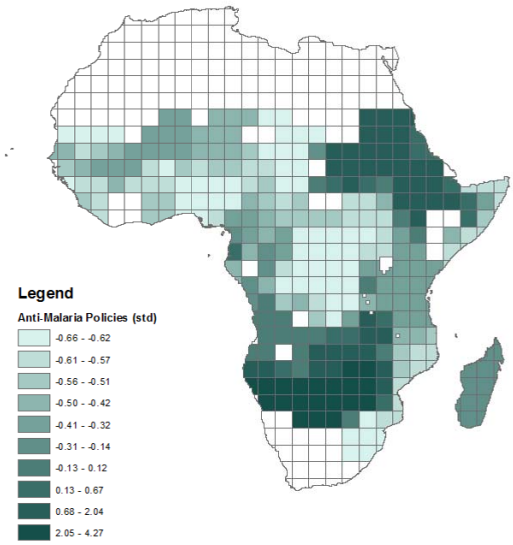
endemicity around 1900 constructed by Lysenko and Semashko (1968) and digitalized by Hay et al. (2004). A corresponding map is presented in Figure A4 in the Supplementary Appendix.

Anti-Malarial Interventions. As a final step, we investigate the potential impact of major anti-malaria interventions. Besides providing insights on the effectiveness of policy initiatives beyond the health domain, this analysis also provides further support for the identification of the role of malaria exposure for civil violence and the mechanism behind this link. To this end, the analysis makes use of a valuable and still under-exploited source of information. Major anti-malaria interventions were essentially absent before 2000, when the average coverage per cell was less than 0.5% of the population. The coverage increased thereafter, and anti-malaria policies were effectively implemented on a large scale by several African states by 2005 in the context of the implementation of the Roll Back Malaria initiative, developed around the United Nations Millennium Development Goals (MDGs).[24] The data are from Bhatt et al. (2015), who provided a first attempt for a comprehensive measure of the scale-up of coverage of main malaria control interventions. These interventions include insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy

[24]Starting from 2005, the average coverage had increased by more than five-fold, with some cells revealing a coverage at the order of 20%.

(ACT).

Figure 5: Anti-Malaria Policy Interventions



Note: Spatial distribution of the coverage of Anti-malarial Policies in the population in $2.5^o \times 2.5^o$ grid-cells. The picture depicts the average coverage of anti-malaria policy interventions (in terms of insecticide-treated bed nets, indoor residual spraying and artemisinin-based combination therapy) in the population (in $2.5^o \times 2.5^o$ cells) in 2005 (see text for details). Data source: Bhatt et al. (2015).

The descriptive statistics of the main variables are contained in Table 1, details regarding data sources and coding can be found in Table 8 in the Appendix.[25]

Table 1: Summary Statistics – Main Variables

Variable	**Mean**	**Std. Dev.**	**Min.**	**Max.**	**N**
Fraction of Years with Conflicts 1997-2016 (ACLED)	0.514	0.345	0	1	442
Malaria Stability (demeaned)	0	8.863	-8.066	24.369	442
Malaria Endemicity	2.461	1.737	0	5	441
Av. Policy Coverage.	0.026	0.039	0	0.165	383

Other Variables and Covariates. The empirical analysis conditions on an extensive set of covariates. To account for the mechanical effect of the share of land in a each cell, which differs

[25]Descriptive statistics for all variables used in the empirical analysis can be found in the Supplementary Appendix in Table A1.

across cells depending on the latitude, the analysis always controls for the natural logarithm of cell area. To streamline the presentation of the empirical results, the different variables are grouped under different headings in the results tables.

The set *Geography/Climate* includes the (log of) area occupied by seas, oceans, lakes and rivers, average and standard deviation of temperature and precipitation (over the period 1901-2000), average elevation and ruggedness of the terrain, and the local vegetation environment by including the Normalized Difference Vegetation Index (NDVI). The controls for *Location and Distances* include latitude and longitude (in some specifications also their second order polynomials), the (log) of the distances (in km) to the cost, to the capital and to the closest country border, the (log) distance to Addis Ababa as a proxy for genetic diversity, as well as an indicator for cells that are split across two (or more) countries. *Natural Resources* accounts for the land suitability for agriculture using two alternative measures (average land suitability in a cell from Ramankutty et. al. (2002) and the caloric suitability index for all crops available in the old world after 1500 from Galor and Özak (2016)), presence of diamond mines, petrol fields, and mineral facilities or deposits. *Ethnic Groups and Country Borders* reflects control for the number of ethnic groups in a cell and an indicator whether a cell is populated by an ethnic group that is divided by country borders. *Population, Night Lights and Roads* includes a set of control variables that, in different ways, are informative about the distribution of population and economic activity, such as population density, the intensity of lights at nights and the presence of a primary roads. A relevant caveat for the inclusion of this latter group of variables is that they might be affected jointly with civil conflict by other omitted variables. While their inclusion may therefore lead to problems of bad controls, exploring the role of these covariates can nonetheless be informative about the potential channels linking malaria to conflicts. Detailed information about the variables and their sources are contained in Table 9 in the Appendix.

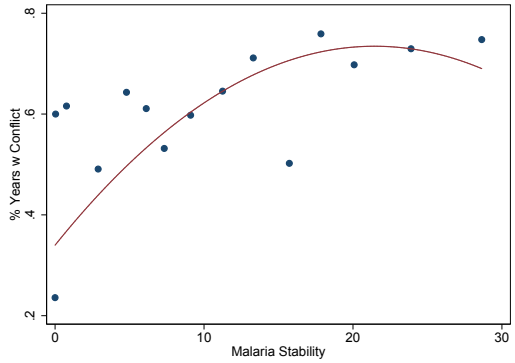
5 Results

5.1 Predicted Malaria Stability and Civil Violence: Baseline Results

Linear Specification. As discussed in Section 4, the advantage of using an index of predicted malaria stability as measure of long-run malaria exposure is its exclusive reliance on biological and geo-climatological specificities of malaria transmission and its availability at high resolution. Figure 6

provides a bin-scatter plot of the unconditional relationship between malaria stability and the fraction of years over the period 1997-2016 during which a cell experienced at least one civil conflict. The graph shows a positive unconditional relationship between malaria exposure and conflict incidence. As suggested by the quadratic fit, however, the relationship becomes weaker for higher levels of malaria stability.

FIGURE 6: STABILITY OF MALARIA TRANSMISSION AND CIVIL CONFLICTS



Note: Scatter plot (bins) of unconditional relationship between malaria stability and civil violence (in $2.5^o \times 2.5^o$ cells); quadratic fit. Data sources: Kiszewski et al. (2004) and ACLED 7.

The shape of the unconditional relationship should be taken as purely suggestive as the analysis does not condition on any of the (many) potentially relevant covariates. To investigate the relationship more rigorously, we conduct regression analysis of the prevalence of conflict in a cell during the period 1997-2016 on the exposure to malaria, in terms of the index value of the malaria stability index *Malaria Stability* of the cell, using a linear specification. The results for different specifications of the estimation equation with regard to control variables are shown in Table 2. Standard errors robust to clustering at the country level are shown in parentheses, Conley-robust standard errors to account for potential spatial clustering are reported in square brackets.

The results shown in Column (1) replicate the unconditional relationship of Figure 6 and show a significant positive association. The results in Column (2) are obtained with a specification that only contains country fixed effects and also suggest a positive association between malaria exposure and civil conflicts. However, once controls for geographic and climatological features of the respective cell are added to the specification, the coefficient becomes quantitatively much weaker and the significant association vanishes, see Column (3). Additional specifications include further controls relating to economic potential and natural resources as in Column (4), controls for the fact that a cell belongs to

Table 2: Malaria Exposure and Conflicts: Linear Specification

Dependent Variable	Fraction of Years with Conflicts (1997-2016) - Cell Level						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.016***	0.014***	0.009	0.006	0.004	0.004	0.006
	(0.003)	(0.004)	(0.006)	(0.005)	(0.006)	(0.006)	(0.006)
	[0.002]	[0.002]	[0.004]	[0.003]	[0.004]	[0.004]	[0.004]
Controls:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	442	442	442	442	442	442	442
Adjusted R-squared	0.183	0.426	0.598	0.619	0.625	0.643	0.670

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors clustered at the country level in parentheses. Conley standard error (300 km cut-off) in squared brackets. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

more than one country (cells with a country border) and for cells that are populated by more than one ethnic group as in Column (5), population density as in Column (6), or for economic development (in the form of access to primary roads and illumination at night) as in Column (7). Despite the risk of subsequently adding potentially 'bad controls' that might be affected by problems of endogeneity or reverse causality, the estimation delivers very similar coefficient estimates, with no evidence for any coherent link between malaria exposure, measured by the malaria stability index, and civil conflict using a linear regression framework.

Non-Linear Specifications. The epidemiological features of malaria imply that high malaria stability is associated with higher inoculation rates, whereas these higher inoculation rates also imply an increasing prevalence of acquired immunities among adults. A key implication is, as discussed above, a non-monotonic relation between malaria stability and the effective malaria burden faced by adults with higher malaria risk for adults in cells with low and intermediate levels of malaria stability compared to cells with high (or no) malaria transmission stability. We investigate the prediction of a

hump-shaped form of $f(\cdot)$ by estimating flexible, non-linear, specifications that allow for a non-linear relation between malaria exposure and civil conflict.

Table 3 presents the results from estimating the same empirical specifications as before in terms of the control variables, but with the malaria stability index entering as second-order polynomial. Throughout all specifications, the estimates reveal a significant positive but concave relationship between malaria exposure and civil conflict, as indicated by the positive coefficient on the linear malaria stability index and by the negative coefficient on the square term. Both coefficients are affected very little when subsequently adding further covariates. In particular, compared to the baseline specification with geographic and climatological controls in Column (3), specifications with controls that are more likely to be affected by endogeneity problems only deliver slightly smaller coefficients (about 25 percent). Since clustered standard errors are similar and even slightly larger compared to Conley standard errors, we only report the former in the remaining tables.

With a coefficient of 0.0212 for the linear term and -0.0007 for the quadratic term as in Column (2), the maximum of the effect for the most extensive specification is around a malaria stability index of 15. In terms of magnitude, the predicted effect for cells located at the the peak of the hump-shape relationship (which correspond to a malaria ecology index of 15) is about 22.5 percentage points larger than the predicted effect in cells with minimum malaria stability (at zero) or at malaria stability of 30 (where the maximum malaria stability in the sample is a cell with an index of around 34). Compared to an unconditional average fraction of years with conflicts of 0.514, this corresponds to a variation in the effect of almost 50 percent of the unconditional mean across the range of malaria stability.

5.2 Discussion and Robustness

The baseline results provide a first indication for a hump-shape relation between malaria exposure and conflicts that is consistent with the role of acquired immunities discussed in Section 3. Several exercises are performed to check the robustness of the baseline patterns and to the explore the predicted role of human immunities in mediating the relationship between malaria stability and conflicts.

Flexible Specifications. Additional robustness checks confirm the main result for more flexible specifications that futher account for spatial correlations in terms of a second order polynomial in

Table 3: Malaria and Conflicts: Non-linear Specification

Dependent Variable	Fraction of Years with Conflicts 1997-2016 - Cell Level						
	Malaria Stability						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.023***	0.021***	0.019***	0.016***	0.014**	0.013**	0.015**
	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
	[0.003]	[0.004]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]
(Malaria Stability)^2	-0.001***	-0.001**	-0.001***	-0.001***	-0.001***	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]
Controls:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	442	442	442	442	442	442	442
Adj. R-squared	0.217	0.433	0.606	0.627	0.633	0.650	0.677

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors clustered at the country level in parentheses. Conley standard error (300 km cut-off) in squared brackets. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

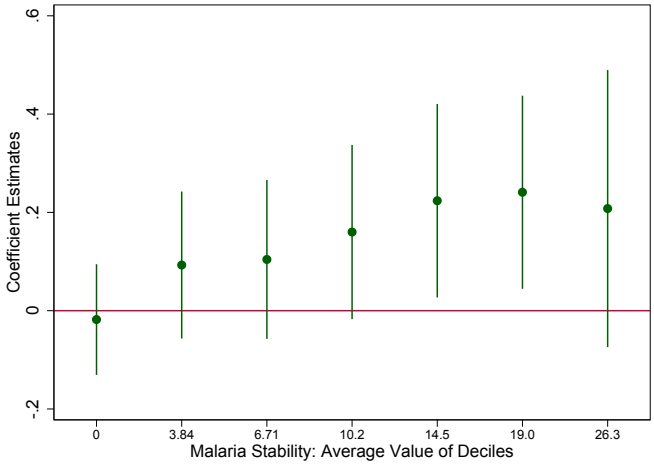
absolute latitude and longitude.[26] As a first more flexible alternative to the quadratic specification of the function $f(Malaria\ Stability)$, we allow for different coefficients for malaria stability for each decile of the data on the malaria stability support. The corresponding coefficient estimates for an empirical specification that includes all controls as in specification (7) of Table 2 are depicted in Figure 7.[27] The pattern emerging from this figure is hump-shaped, with the largest and significantly positive coefficient estimates for deciles with average malaria stability of around 14.5 and 19. As a final way to visualize the existence of a hump-shape link between predicted malaria stability and conflicts, we plot the unexplained variation that is left after controlling for all covariates (such as

[26]See Table A2 in the Supplementary Appendix.

[27]Notice that since around 40% of all cells in the sample exhibit a malaria stability index of 0, the four lowest deciles coincide at an average value of 0, which explains that only seven coefficients are plotted.

the specification as in Column (7) of Table 2 but without malaria stability) against malaria stability non parametrically by means of local polynomial regression. The results provide further suggestive evidence of a non monotonic relationship.[28]

FIGURE 7: MALARIA STABILITY AND CIVIL CONFLICT: SEMI-PARAMETRIC ESTIMATES



Note: Results of regressions of civil violence (in $2.5^o \times 2.5^o$ cells) by malaria stability, allowing for different coefficients by decile. Notice that as consequence of the distribution, three deciles take value 0 and form the reference, group, while the fourth decile (for which the first coefficient is estimated) exhibits an average malaria stability index of 0.41.

Alternative Long-Run Mechanisms. THE PART ON INDIRECT CHANNELS IS A BIT LONG. IF WE NEED TO CUT I WOULD DO IT HERE. Variation in long-term malaria exposure may affect civil violence indirectly by affecting relevant conflict-related outcomes of the process of long term development, for instance through the quality of institutions. Along the lines of the argument proposed by Acemoglu, Johnson, and Robinson (2001), regions that are associated with low exposure might have been governed by colonizers that implemented inclusive institutions, whereas in regions with intermediate exposure and therefore high risk of epidemics among adult colonizers might have implemented extractive institutions. In regions with very high malaria exposure, European colonization might also have been severely hampered or even prevented. One element of this argument that does not square with the epidemiological literature and the baseline results is that it refers to mortality of European settlers. Contrary to African populations, these settlers might have displayed mortality rates that were monotonic (and not hump-shaped) in the intensity of malaria transmission, suggesting the emergence of extractive institutions, and therefore high conflict

[28]See Figure A6 in the Supplementary Appendix.

intensity, in areas with high, rather than low to intermediate, malaria stability. To the extent that colonization history is accounted for by the historically-determined national borders contained in the country fixed effects, the effect of (national) institutions is already absorbed in the empirical analysis. Following Michalopoulos and Papaioannou (2016), we also explicitly account for whether a given cell hosts an ethnic group that has been partitioned by country borders, and whether the cell is intersected by a country border, which additionally accounts for systematic influences of colonization strategy unrelated to the malaria channel. To explore the issue further, we also conducted an analysis for more homogeneous sub-samples with respect to colonization history and European settlement patterns. In particular, we replicated the analysis while excluding Northern Africa, which has a different colonization history, excluding North and South Africa, or excluding countries with significant numbers of European settlers (which might indicate suitable conditions for long-term settlements and hence incentives to implement inclusive rather than extractive institutions). The results consistently document the same non-linear relationship between long-run malaria exposure and conflict as in the main sample.[29]

Another indirect channel refers to the long term effect of malaria exposure population composition. Cervellati, Chiovelli and Esposito (2016) provide arguments and evidence that highly malarial areas are characterized by larger ethnic diversity, which could in turn affect civil conflicts. To explore this channel we explicitly account for the number of ethnic groups in the cells. Controlling for ethnic diversity reduces slightly the role of malaria stability (and the unreported effect of ethnic diversity on conflicts is positive and significant), suggesting that this is not the main, or the only, driver of the findings. Another long-term effect of malaria stability can be related to population density, the incentives for urbanization and economic development in general. Depetris-Chauvin and Weil (2017) find no evidence of a significant link between malaria and pre-colonial population density and proxies for development. Cervellati, Esposito and Sunde (2017) suggest the existence of a link between malaria exposure and light density at nights. The main patterns are robust to controlling for population density, night light intensity and access to primary roads.

Alternative Measures of Long Term Exposure to Malaria: Malaria Endemicity in 1900. We also replicated the analysis using a novel measure of malaria endemicity in the population around 1900 as alternative measure of malaria exposure. This measure accounts for the prevalence of the pathogen in the respective populations during the early stages of European colonization and

[29]See Table A4 in the Online Appendix.

thus provides valuable information regarding potential endogeneity issues. Estimation results from the same empirical specifications with respect to the other control variables deliver consistent evidence for a non-linear, hump-shaped relationship between malaria exposure and conflict prevalence.[30] Additional unreported results also suggest that the prevalence of genetic immunities to malaria infections, which is increasing in long-term exposure, has no effect on conflict by themselves but has an attenuating effect on the relation of malaria stability, and therefore the effect of latent inoculation rates, on conflict.

Alternative Conflict Measures. To investigate the robustness of the results with respect to the measure of civil violence, the share of years with any violent conflict in a given cell, we replicate the estimation using alternative measures of conflict at the extensive and intensive margin. Estimation results for measures including the log of the number of total conflicts (+1), for the fraction of years with any conflict in a given cell constructed from an alternative source, the UCDP GED data set, for the severity of civil violence at the intensive margin, the log number of fatalities reported in the ACLED data, or for a binary variable that takes value 1 if there has been at least one conflict in a cell during the period 1997-2016, and zero otherwise, as dependent variables all confirm the concave shape of the association of malaria stability with conflicts.[31] With reference to the behavioral patterns and channels that are responsible for the results, we investigated the robustness of the relationship for different types of conflicts, including the fraction of years during 1997 and 2016 in which a cell experienced conflicts that relate to large-scale confrontations ("battles"), to violence against civilians, or to riots or protests (all from the ACLED data). For all these subcategories of violent events, the estimates reveal a non-linear, concave association between malaria exposure and civil violence, with the weakest results for riots and protests.[32]

[30]The results are shown in Table A3 in the Supplementary Appendix. The peak of the effect is obtained for an index level of 3.5 when considering an average linear coefficient of 0.245 and a quadratic coefficient of 0.035. Quantitatively, the effect of variation in malaria endemicity is even slightly larger than one obtained with malaria stability, where the predicted conflict for cells at the peak is 43 percentage points larger than for cells with no endemicity.

[31]See Table A6 in the Supplementary Appendix for the respective results, which are even quantitatively similar with a peak of the effect around a malaria stability index of 15.

[32]See Table A7 in the Supplementary Appendix.

5.3 Exploring the Role of Anti-Malarial Policies

In a last step, we explore the role of anti-malarial policies. The results of this analysis are informative about the scope of policy, since one would expect antimalarial interventions to moderate violence mainly in areas with a high malaria risk for adults that characterized by a low level of acquired immunities (as documented before). In addition, this analysis sheds light on the identification of the effect of malaria exposure by providing an indirect test of the exclusion restriction that implicitly assumes that the hump-shape effect of the malaria stability index on conflict does not reflect an alternative (geographical or bio-climatological) feature that drives conflict. If it were the case that the exclusion restriction does not hold and other channels are at work, then anti-malaria policies should not lead to any reduction in violence. TEO: I DO NOT UNDERSTAND THE WORDING IN TERMS OF EXCLUSION RESTRICTION. WE DO NOT HAVE AN IV. CAN WE HAVE A BETTER WORDING? We use novel data on anti-malarial policies in terms of the population coverage with insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) and investigates the link between these policies and conflict prevalence, as well as their interaction with the exposure to malaria.

Given the cross-sectional nature of the data a first concern refers to the existence of third factor that correlates with both conflict prevalence and policy coverage. Our interest is not on the role of policies *per se*, but rather on the role of the interaction term between policies in high risk areas. This effect can be estimated consistently even if a third factor correlates with the policy variable, as long as the policy variable is jointly independent of the malaria exposure, see Bun and Harrison (2014) and Nizalova and Murtazashvili (2016). A potentially more serious concern is the possibility that policy implementation is prevented or complicated by the occurrence of conflicts. As mentioned in Section 2 this concern is well funded in the context of civil wars and conflicts involving substantial displacements of people and the presence of refugee camps, see in particular Montalvo and Reynal-Querol (2007) for cross-country data on civil wars. However, no consistent patterns have been detected in terms of the effect of localized small scale violence (see Messina et al., 2011 and Sedda et al. 2015).

Information on policy coverage is available at the sub-national level. The data nonetheless reveal strong country-level patterns, which are visible also in the spatial distribution of policy coverage depicted in Figure 5 reported in Section 4. These country-level patterns likely reflect the decisions

to implement these policies, as well as data patterns related to coverage and documentation. The geographic coverage of the data, which originally stem from health surveys and field studies, is related to country-level factors and smoothed by spatial interpolation. Both features, the variation at country level and the geographic clustering, imply that the policy data are characterized by higher spatial autocorrelation and geographic clustering than the data for conflicts and covariates, which are available at high levels of spatial disaggregation. This also poses a challenge for the use of policy data with excessively small grid cells.[33] Moreover, the local deployment of anti-malaria policies might be influenced by localized civil conflicts.

In light of this, we first consider the coverage of anti-malarial policies at the country level. In the context of an estimation framework that exploits cell-level data, this implies loosing information about within-country variation in policy coverage. At the same time, using country level policy coverage limits the potential of reverse causality running from the occurrence of localized violent events to policy coverage in a given cell. The use of country-level variation in policy coverage in interaction with local long-term malaria exposure (which is related to exogenous geographic and bio-climatic conditions) provides an a first identification strategy that exploits exogenous variation at the country level in interaction with local conditions. Finally, as discussed in Section 4, the coverage with anti-malaria policies was essentially zero by the year 2000, and most interventions were implemented around or after 2005. We use coverage data for the year 2005 and exploit variation in conflicts pre and post 2005.

Preliminaries. We begin by exploring the role of localized conflicts before 2005 on the coverage of anti-malaria policies. To this end, we regress the coverage of anti-malaria policies in 2005 on prevalence during the period 1997-2005. The results, shown in Table 4, reveal no statistically significant pattern linking conflict prevalence before policy implementation and policy coverage in 2005 in any of the specifications. Conflicts explain virtually no variation in coverage of anti-malaria policies at the country level.[34] This finding provides a first suggestive indication about the validity of the identification strategy as it reveals no systematic robust influence of local conflict activity before 2005 on country-level policy coverage in 2005.

THERE IS SOMETHING WRONG WITH THIS TABLE? THE FIRST TWO COLUMNS ARE

[33]This was also an important reason for conducting the analysis using 2.5^o as baseline.

[34]Similar patterns emerge for the effect of conflicts over the period 1997-2005 on policy coverage at the cell level as reported in Table A8 in the Supplementary Appendix.

TABLE 4: VIOLENCE BEFORE 2005 AND ANTI-MALARIA POLICIES AT COUNTRY LEVEL IN 2005

Dependent Variable	Anti-Malaria Policies - Country Level in 2005						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Conflicts (1998-2004)	-0.147	-0.137	-0.311	-0.250	-0.254	-0.159	-0.140
	(0.298)	(0.224)	(0.257)	(0.238)	(0.248)	(0.243)	(0.241)
Sets of Covariates:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	No	No	No	No	No	No
Observations	383	383	383	383	383	383	383
Adj. R-squared	-0.001	0.268	0.296	0.339	0.336	0.349	0.354

Notes: OLS estimates. The dependent variable is the coverage with anti malaria policies at country level in 2005 in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015). Conflicts (1997-2005) measures the fraction of years 1997-2005 with a conflict event in the cell (ACLED 1997-2016). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

DIFFERENT BUT THE REPORTED SPECIFICATION IS THE SAME. MAYBE COUNTRY FE ARE INCLUDED FROM COLUMN 2?

High Malaria Risk Areas for Adults: Dichotomous Measurement. To move a step forward in the investigation it is useful to provide a dichotomous coding of high malaria risk for adults. The hump-shaped pattern of the association between malaria exposure and civil conflict can be interpreted as being related to the high malaria risk areas for adults. In view of the epidemiological literature discussed and the evidence presented in Section 3, the risk of serious symptoms or even death for adults is low in areas with very low transmission stability due to the low inoculation rates or in areas with very high transmission stability due to the acquired immunity.

We construct a corresponding dichotomous measure by coding a binary variable that takes value one only in the low to intermediate malaria stability areas. We code cells as high risk for malaria infections of adults if the stability index takes positive but low values, and as low risk for malaria infection either if malaria stability is zero or takes sufficiently high values. This requires the definition

of a threshold above which malaria stability is large. Following the epidemiological evidence on the hump shape link between malaria stability and mortality we code high stability areas for levels of malaria stability above 15. This is also compatible with the view that above this level cells tend to display large historical endemicity (see Figure A3 in the Supplementary Appendix) and that the selective pressure from the pathogen in the past was large enough to induce the development of widespread genetic immunities (see Figure A7 in the Supplementary Appendix).

Table 5 presents the estimation results for specifications with the same set of control variables as before, but with a binary measure for malaria risk that has been constructed as described. The results confirm that conflict prevalence is significantly higher in cells with high malaria risk as compared to cells with a low malaria risk.[35] The results document that the hump-shape effect of malaria stability on conflicts can be detected also with a simple dichotomous measure. Despite being coarse, the binary measure has the advantage of not relying on information on the intensive margin of malaria stability and allowing us to explore the role of policies along the lines of a difference-in-difference design as discussed next.

The effect of anti-malarial policies in high and low areas for adults. As a first step in the investigation of the potential joint effect of malaria risk and anti-malaria interventions on conflict we explore the role of policy coverage in 2005 for subsequent conflicts.

Table 6 presents the results for regressions of the prevalence of cell-level civil conflict after 2005 on specifications with an interaction term between malaria risk, measured using the same binary indicator variable as before, and the coverage of anti-malaria policies at the country level in 2005. The estimates again show a positive effect of high malaria risk (in terms of a binary indicator) and conflict, which is statistically significant in most specifications. Quantitatively, this effect is smaller than in the baseline results of Table 5. The coverage of anti-malaria policies at the country level has a significantly negative influence on the effect of malaria risk on cell-level civil conflict.[36] A higher policy coverage tends to reduce conflict in cells with high risk of epidemic malaria outbreaks among adults. This effect is sizable in comparison to the main effect. Moreover, this specification explains a considerable part of the variation in conflicts at the cell level.[37] THE NEXT SENTENCE IS

[35]Table A9 in the Supplementary Appendix presents results a coding of high malaria risk areas in the alternative range $\in (5, 25]$. The results suggest that the baseline coding provides a conservative measure of high risk for adults.

[36]The effect of anti-malaria policies at the country level is not identified in specifications with country fixed effects.

[37]The placebo results for predicting policies in 2005 by violence before 2005 also holds when allowing for a heterogeneous effect of conflicts in high and low stability areas by interacting the conflict incidence with the respective binary

Table 5: Malaria and Conflicts: Binary Measure for High Malaria Risk

Dependent Variable	Fraction of Years with Conflicts 1997-2016 - Cell Level						
	Binary Measure with Malaria Stability $\in (0, 15]$						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria - High Risk DV $\in (0, 15]$	0.181**	0.200**	0.083*	0.090*	0.083*	0.068*	0.029
	(0.069)	(0.075)	(0.047)	(0.047)	(0.047)	(0.039)	(0.038)
Observations	442	442	442	442	442	442	442
Adj. R-squared	0.082	0.405	0.618	0.622	0.629	0.645	0.668
Controls:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). "Malaria - High Risk DV" is a binary variable that identifies cells with high risk of epidemic malaria outbreaks among adults, reflected by levels of the index of malaria strength and stability by Kiszewski et al. (2004) in the interval $(0, 15]$. Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

NOT FULLY CLEAR TO ME: These findings complement the findings regarding immunity in light of the fact that immunity and resistance are low in these high risk areas, making the populations particularly vulnerable to malaria outbreaks in these regions. The results are robust to controlling for the prevalence of conflict in the respective cell before 2005.[38]

As a final step, we explore the role of anti-malaria policies in a simple differences-in-differences framework. Identification in such a framework comes from the timing and intensity of the policy treatment both of which may be potentially affected by conflicts. To limit potential endogeneity problems with the actual timing of policies we restrict attention to a long difference set up by looking at two sub-periods: before 2005 (where policy coverage was essentially zero) and after 2005. To limit problems of endogeneity and measurement at the intensive margin, we create a time varying

indicator, see Table A10 in the Supplementary Appendix, or when using a binary indicator for policies, see Table A11 in the Supplementary Appendix.

[38]See Table A12 in the Supplementary Appendix.

Table 6: Malaria, Policies (Country Level) and Violent Events

Dependent Variable	Fraction of Years with Conflicts (2006-2016) - Cell Level						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria - High Risk DV $\in (0, 15]$	0.129*	0.127**	0.067**	0.044*	0.040	0.038	0.015
	(0.067)	(0.058)	(0.026)	(0.026)	(0.026)	(0.026)	(0.028)
Malaria - High Risk DV $\in (0, 15]$	-0.052	-0.082***	-0.071***	-0.088***	-0.087***	-0.090***	-0.077***
$\times$ Pol. Country 2005	(0.042)	(0.024)	(0.018)	(0.015)	(0.016)	(0.017)	(0.016)
Sets of Controls:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	383	383	383	383	383	383	383
Adjusted R-squared	0.050	0.360	0.552	0.570	0.574	0.578	0.599

Note: OLS estimates. The dependent variable is the fraction of years 2006-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria - High Risk DV" is a dummy that identifies cells with low to intermediate levels of stability of malaria transmission given by levels of the index of malaria strength and stability by Kiszewski et al. (2004) in the interval $(0, 15]$. Anti malaria policies measures the average coverage in a country of main malaria control interventions in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

binary indicator variable that takes value one after 2005 only in the cells in which policy coverage was sufficiently large (we take the sample average as the respective threshold). The diff-in-diff is then implemented by comparing the differential effect in areas with high malaria risk for adults (i.e., areas characterized by low to intermediate malaria stability) and in areas with low malaria risk for adults (locations with zero or large malaria stability). Given the epidemiological evidence we should expect no sizable effect of policies on health and mortality of adults in low malaria risk cells (that is in cells with high and zero malaria transmission).

More specifically, we regress the fraction of years with conflicts and, alternatively, the number of yearly conflict-related fatalities, on the cell level for the period before and after 2005, on a binary measure of policy coverage, an indicator for the period after 2005 (the "treatment" period), and an interaction with the (time-invariant) binary indicator characterizing cells with high malaria risk for

adults. Since the coverage of anti-malaria policies is essentially zero before 2005, this specification corresponds to estimating the differential effect of the implementation of anti-malaria policies with high coverage in cells with high and low malaria stability.

The results, reported in Table 7, reveal that conflict prevalence increased in the second sample period, but not differentially by malaria exposure. While policy coverage appears to have no consistent relation with conflict prevalence in cells with low malaria risk, civil conflict prevalence is significantly reduced by anti-malaria policies in cells with high malaria risk. The findings are suggestive of the role of policy implementation in reducing violent conflicts, but only in areas with high risk (low resistance) for adults and not in areas with low risk (high resistance).

Table 7: Antimalarial Policies, Malaria Stability and Violent Events: Diff-in-Diff

Dependent Variable	Frac. of Years with Conflicts		Ln Average Yearly Fatalities	
	(1)	(2)	(3)	(4)
Policies DV	0.063	-0.012	-0.079	-0.033
	(0.051)	(0.061)	(0.363)	(0.419)
Policies DV×Malaria High Risk DV	-0.078*	-0.124**	-0.626**	-0.627*
	(0.044)	(0.048)	(0.302)	(0.331)
Post 2005	0.100**	0.090**	0.662***	0.267
	(0.039)	(0.046)	(0.209)	(0.329)
Cell FE	Yes	Yes	Yes	Yes
Data Source	ACLED	UCDP GED	ACLED	UCDP GED
Observations	612	612	612	612
Adjusted R-squared	0.778	0.710	0.655	0.561

Note: OLS estimates. The sample is composed by 306 cells for which information on policies is available for two time periods (2000-2005 and 2006-2015). The dependent variable is the fraction of years with (at least) a conflict event in the two periods, in Column 1-2 and the average number of fatalities in the two periods, in Column 3-4. Both variables are constructed using data from ACLED 7 in columns 1 and 3 and using UCDP GED in columns 3 and 4. "Post 2005" is an indicator variable taking value 1 in the second period (2006-2015). Malaria High Risk DV is a binary indicator taking value one for the cells in the low to intermediate malaria stability of transmission measured in terms of an index by Kiszewski et al. (2004) $\in (0, 15]$. Policy DV is an indicator variable taking value 1 if policies in the cell are higher than the mean level of policies in the sample in the period 2000-2015. Anti malaria policies measures the average coverage in a country of main malaria control interventions in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

@UWE: NICOLA ASKED TO INCLUDE ALSO A QUANTIFICATION OF THE EFFECT OF THE POLICY ANALYSIS.

6 Concluding Remarks

This paper explores the existence of a link between the long-term exposure to malaria and the occurrence of civil conflicts in Africa. The empirical analysis exploits disaggregate data at the level of 2.5 degree grid cells for the whole of Africa. The empirical strategy builds on insights from the epidemiology of malaria, which have documented systematic variation in the age composition of severe malaria infections for different levels of inoculation rates. Adult mortality from malaria peaks in areas with low to intermediate levels of malaria stability where the risk of being bitten by infected mosquitos is non-negligible and where the adult population is not immune. The results document a hump-shaped relationship between the intensity of malaria transmission and civil violence. This finding consistently emerges for alternative measures of long term malaria exposure and for different estimation strategies including both parametric and non parametric regressions. The hump-shaped relation is found for different measures of civil violence, including the likelihood of violence and its intensity (also in terms of fatalities). The effect is sizable for violent confrontations between armed groups and violence against civilians while we find no significant evidence for riots and protest. The results provide some indications of a moderating effect of genetic immunities to malaria. DO WE WANT TO EMPHASIZE THIS? ALSO FROM THE TEXT IT IS NOT CLEAR TO ME IF WE WANT TO EMPHASIZE/REPORT THE RESULTS ON GENETIC IMMUNITIES. THE FIGURES AND THE TABLE ARE STILL PRESENT BUT NO LINK TO THEM EXISTS ANY MORE IN THE TEXT.

We also explored the role of health policies using information on the substantial scale up in anti-malarial interventions after 2005 in Africa in the context of the Roll Back Malaria program. By exploiting information about the timing of interventions we provide suggestive evidence that the implementation of anti-malarial policies led to a reduction in civil violence but only in areas where adults are more at risk. No evidence of sizable effects can be detected in areas with high malaria transmission where acquired immunities in adults already offer substantial protection against infection even in the absence of health policies. The findings therefore suggests that the effect is likely heterogenous and concentrated in areas where adults, rather than children, are mostly at risk of malaria infections.

In terms of mechanism, the results are compatible with the possibility that the effect of malaria works through a direct, rather than an indirect, channel and is linked to the actual incidence of the

disease in the adult population. The current analysis essentially relies, however, on cross-sectional variation across different locations and does not allow us to reach final conclusions. Further analysis is definitively needed to identify the actual channel linking variation in malaria exposure, or short term malaria shocks, to civil violence.

The findings are suggestive of the existence of a largely overlooked, but potentially very relevant, side effect of antimalarial interventions. The empirical results points at a potentially important effects of anti-malarial policies above and beyond health conditions. The evidence is compatible with the possibility that the development of a malaria vaccine might provide an effective instrument also to reduce health-related conflicts. Lack of data availability prevented us to perform meaningful cost-benefit analysis, however. Definitively more work is therefore needed for a deeper understanding of the link between variation in malaria exposure and the likelihood of violence and for designing appropriate and effective policy interventions.

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Table 8: Main Variables: Description and Data Sources

Measures of Violence

ACLED. Baseline measure is the fraction of years with at least one conflict event in the cell over the period 1998-2015. Alternative measures for conflicts over the same time span are: a dummy taking value one if there is at least one conflict in the cell in the period, the total number of conflicts and the (log) number of fatalities. In terms of conflict types we code the fractions of years with battles, with violence against civilians and riots or protests. Source: ACLED Version 7 (1997-2016), ACLED - Armed Conflict Location and Event Data Project.

UCDP. The baseline measure is the fraction of years with at least one conflict event in the cell over the period 1998-2015. As robustness we also code the total number of events. Source: UCDP Georeferenced Event Dataset (GED) Global version 17.1 (2016).

Measures of Long Term Exposure to Malaria

Malaria Stability Index. Index measuring the predicted force and stability of malaria transmission based on biological characteristics of diverse vector mosquitoes and their interaction with local climate. Data source: Kiszewski, Mellinger, Spielman, Malaney, Sachs, and Sachs (2004).

Historical Malaria Endemicity. Prevalence of malaria in the population in 1900. Lysenko and Semashko (1968) and digitalized by Hay (2004).

Genetic Immunities. Average predicted frequency of genetic immunities to malaria in terms of sickle haemoglobin alleles, G6PD deficiency and Duffy blood group in the general population. Source: Piel et al. (2013) and Howes et al. (2011, 2013).

Anti-Malaria Policies

Average Coverage. Anti-Malaria Policies (av. coverage) is the average coverage in the cell in the year for three major anti-malaria policies: Insecticide treated bednet coverage (ITN), Indoor residual spraying coverage (IRS) and Artemisinin-based combination therapy coverage (ACT). Bhatt et al. (2015), retrieved from http://www.map.ox.ac.uk/.

Max coverage. Cell year average coverage of the policy with the higher coverage. Bhatt et al. (2015), retrieved from http://www.map.ox.ac.uk/.

Table 9: Covariates: Description and Data Sources

Geography:

Cell Area. Nat. logarithm of the cell area.

Total Water Area. Total area occupied by water in the cell (seas, oceans, lakes and rivers). Digital Chart of the World inwater shapefile and the Digital Chart of the World oceans and sea shapefile.

Average Temperature. Average annual cell temperature (1961-1990). FAO/IIASA, 2011-2012. Global Agro-ecological Zones (GAEZ v3.0). FAO and IIASA.

Average Precipitation. Average cell monthly precipitation mm/month (1961-1990). CRU CL 2.0 data from New, Lister, Hulme, and Makin (2002).

Mean Elevation. Average cell elevation. National Oceanic and Atmospheric Administration (NOAA) and U.S. National Geophysical Data Center, TerrainBase, 1.0, Boulder, Colo.

Ruggedness. Average ruggedness (Terrain Ruggedness Index, 100 m). Terrain Ruggedness Index devised by Riley, DeGloria, and Elliot (1999). http://diegopuga.org.

Variation in Precipitation. Standard deviation of the av. yearly precipitation years 1900-2000. Buggle and Durante (2017).

Variation in Temperature. Standard deviation of the av. yearly temperature years 1900-2000. Buggle and Durante (2017).

Vegetation. Normalized Difference Vegetation Index (NDVI). Average for year 1999. COPERNICUS Global Land Service. http://land.copernicus.eu/global.

Location and Distances:

Absolute Latitude and Longitude. From centroid of the cell. Constructed with ArcGIS.

Ln Distance Coast and Ln Distance Border. Nat. logarithm of the average cell distance to closest coast and to the closer border, respectively. Coastline shapefile from Global Self-consistent Hierarchical High-resolution Geography Version 4.2.2 January 1, 2013.

Ln Distance Capital. Nat. logarithm of the average cell distance to the country capital. World Capital shapefile.

Ln Distance River. Nat. logarithm of the average cell distance to the closest river. Major Rivers World Selected (p3w) shapefile (from www.naturalearth.com).

Ln Distance Adis Ababa. Nat. logarithm of the geodesic distance to Adis Ababa.

Natural Resources:

Land Suitability. Average land suitability. Ramankutty (2002) .

Caloric Suitability Index. Potential agricultural output (measured in calories) post-1500. Galor and Ozak, (2016).

Diamond Mines. Indicator variable taking value 1 if at least one petrol field is located in the cell, 0 otherwise. Gilmore et al. (2005)

Petrol Fields. Indicator variable taking value 1 if at least a diamond mine is located in the cell, 0 otherwise. Lujala et al. (2007).

Mines. Indicator variable taking value 1 if at least a mineral facilities or mineral deposit is located in the cell, 0 otherwise. U.S. Geological Survey, U.S. Department of the Interior.

Shared Cells, Population and Economic Activities:

>1 Country. Indicator variable taking value one if the cell is split across two countries, 0 otherwise. Constructed with ArcGIS.

Ethnic Groups. Number of ethnic groups in the cell. Geo-referencing of ethnic groups dataset (GREG). https://icr.ethz.ch/data/greg/

Partitioned Cell. Indicator variable taking value one if the cell there is an ethnic group that have been partitioned by a country border, 0 otherwise. Constructed with ArcGIS.

Ethnic groups. A dummy indicating if more than one ethnic group is observed in a cell (constructed with a ArcGIS). "Geo-referencing of ethnic groups" (GREG) database.

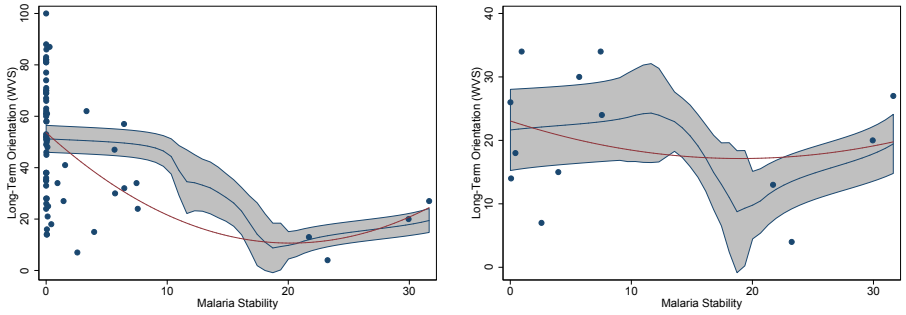
Population. Average population in year 1995. Center for International Earth Science Information Network - CIESIN - Columbia University, FAO, and Centro Internacional de Agricultura Tropical - CIAT. 2005. Gridded Population of the World, Version 4 (GPWv3): Population Count Grid. Palisades, NY: NASA Socioeconomic Data and Applications Center (SEDAC). http://sedac.ciesin.columbia.edu/data/set/gpw-v3-population-count.

Night Lights. Average night light intensity in year 2000. NOOA National Geophysical Data Centre.

Primary Roads. Indicator variable taking value one if the cell is crossed by at least one primary road. World Roads Shapefile, Esri.

Supplementary Appendix: Additional Figures

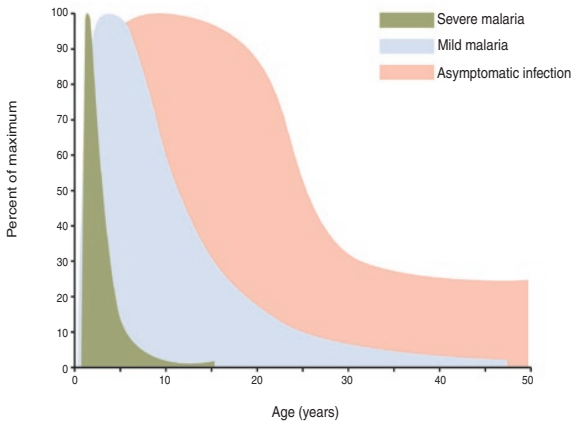
Figure A1: Exposure to Malaria and Long-Term Orientation



(a) World (b) Africa

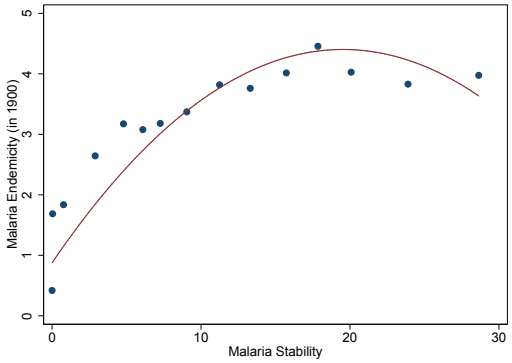
Note: Scatter plot, quadratic fit and kernel-weighted local polynomial regression results. Sources: World Values Survey (taken from Galor and Özak, 2016) and Kiszewski et al. (2004). See Section 4 for details.

Figure A2: Severity of Malaria Infections by Age



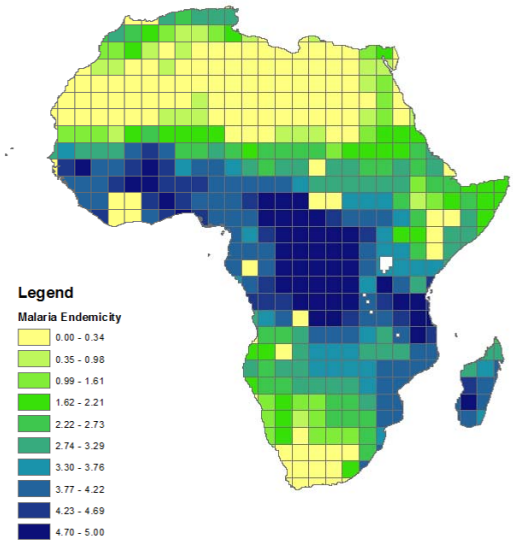
Note: In Malaria Endemic Areas. Figure extracted from Langhorne et al. (2008, Figure 1b).

Figure A3: Stability of Malaria Transmission and Malaria Endemicity



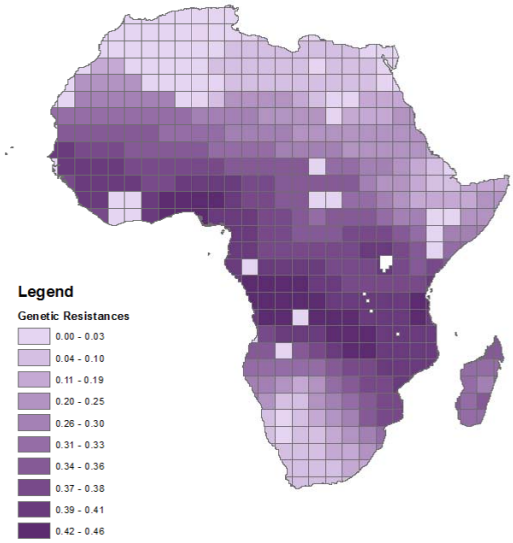
Note: Bin Scatter plot of malaria stability and malaria endemicity in 1900 (in $2.5^o \times 2.5^o$ cells). Data sources: Kiszewski et al. (2004) and Hay et al. (2004).

Figure A4: Endemicity of Malaria in the Population in 1900



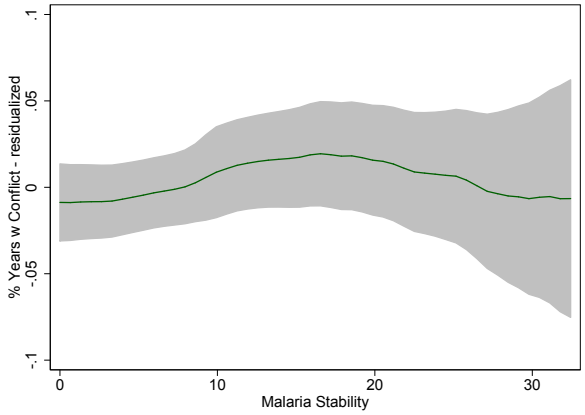
Note: Spatial distribution of the endemicity around 1900 (in $2.5^o \times 2.5^o$ cells). Data source: Hay et al. (2004).

Figure A5: Genetic Immunities



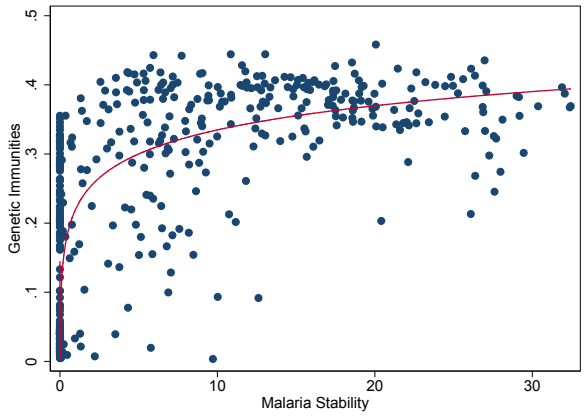
Note: Spatial distribution of the prevalence of genetic immunities (in terms of average prevalence of sickle-cell trait, G6PD, and Duffy antigen negative) in the population (in $2.5^o \times 2.5^o$ cells). Data sources: Piel et al.(2013) and Howes et al. (2011 and 2013).

Figure A6: Malaria Stability and Civil Conflict: Local Polynomial Fit of residual variation



Note: Kernel-weighted local polynomial regression of civil violence (in $2.5^o \times 2.5^o$ cells) by malaria stability, conditional on all controls as in specification (7) of Table 2 but without malaria stability.

FIGURE A7: MALARIA EXPOSURE AND GENETIC IMMUNITIES



Note: Scatter plot of average prevalence of genetic immunities in the population against malaria ecology (malaria transmission and stability index constructed by Kiszewski et al., 2004) in $2.5^o \times 2.5^o$ cells.

Supplementary Appendix: Additional Tables and Results

Table A1: Summary Statistics

Variable	Mean	Std. Dev.	Min.	Max.	N
Fraction of Years with Conflicts 1997-2016 (ACLED)	0.514	0.345	0	1	442
Fraction of Years with Conflicts 1989-2015 (UCDP GED)	0.220	0.261	0	1	442
Av. Conflict Fatalities p.a.	1482.353	7301.889	0	119965	442
Conflict Fatalities DV	7.05	6.335	0	20	442
Battle DV	6.253	6.234	0	20	442
Strategic Conflict DV	3.717	4.385	0	20	442
Riots/Protests DV	6.165	5.991	0	20	442
Violence ag. Civilians DV	6.919	6.392	0	20	442
Malaria Stability (demeaned)	0	8.863	-8.066	24.369	442
Malaria Stability^2	78.370	100.218	0.004	593.858	442
Malaria Endemicity	2.461	1.737	0	5	441
Cell Area	20.194	0.596	17.122	20.468	442
Total Water Area	2199.295	2663.489	0.685	14581.728	442
Av. Precipitation	57.68	53.935	0.112	265.381	442
Av. Temperature	23.806	3.47	13.78	29.966	442
Precipitation (std.)	148.642	100.169	0.217	513.099	442
Temperature (std.)	0.126	0.069	0.024	0.349	442
Mean Elevation	554.99	426.575	-1533.861	2008.812	442
Ruggedness	63454.443	69394.01	1270.976	364646.719	442
Norma. Diff. Vegetation Index	144.192	48.559	16.321	231.409	442
Latitude	5.154	18.379	-33.868	35.75	442
Longitude	18.941	16.448	-23.75	57.75	442
Distance to Capital (ln)	12.83	0.945	8.303	14.394	442
Distance to Coast (ln)	12.133	2.009	0	14.129	442
Distance to cl. Border (ln)	10.64	2.111	0	12.887	442
Distance to River (ln)	0.938	0.737	0.116	2.655	442
Distance to Addis Ababa (ln)	7.893	0.625	4.475	8.814	442
Land Suitability Agr.	0.255	0.253	0.001	0.946	442
Caloric Suitability I.	913.569	791.366	0	2555.548	442
Diamond Mines	0.199	0.4	0	1	442
Mineral Mines	0.862	0.345	0	1	442
Petrol Fields	0.208	0.406	0	1	442
>1 Country	0.523	0.5	0	1	442
# Ethnic Groups	3.342	2.326	1	11	442
Partitioned Cell	0.738	0.44	0	1	442
Population dens. (1995)	32.759	74.358	0.031	595.528	442
Night Lights (1995)	2.647	0.985	2.001	13.151	442
Primary Roads	0.437	0.497	0	1	442
Av. Genetic Immunity	0.26	0.141	0.004	0.458	442
Av. Policy CoverageAv.	0.026	0.039	0	0.165	383

Table A2: Malaria and Conflicts: Non-linear Specification (Robustness)

Dependent Variable	Fraction of Years with Conflicts 1997-2016 - Cell Level						
	Malaria Stability						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.023***	0.021***	0.018***	0.016***	0.014**	0.013**	0.014**
	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
(Malaria Stability)^2	-0.001***	-0.001**	-0.001***	-0.001***	-0.001***	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Controls:							
Geography/Climate (ext.)	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	442	442	442	442	442	442	442
Adj. R-squared	0.217	0.433	0.606	0.627	0.633	0.650	0.677

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Each specification controls for the natural logarithm of the cell area. Geography/Climate (ext.) includes second-order polynomials of absolute latitude and longitude to flexibly account for spatial correlations unrelated to malaria. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors clustered at the country level in parentheses. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A3: Malaria Endemicity and Conflicts: Non-linear Specification

Dependent Variable	Fraction of Years with Conflicts 1997-2016 - Cell Level						
	Malaria Endemicity						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Endemicity	0.250***	0.284***	0.284***	0.284***	0.284***	0.238***	0.238***
	(0.050)	(0.066)	(0.066)	(0.066)	(0.066)	(0.057)	(0.057)
(Malaria Endemicity)^2	-0.032***	-0.037**	-0.037**	-0.037**	-0.037**	-0.030**	-0.030**
	(0.010)	(0.015)	(0.014)	(0.014)	(0.014)	(0.013)	(0.013)
Controls:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	441	441	441	441	441	441	441
Adjusted R-squared	0.309	0.517	0.518	0.518	0.518	0.564	0.564

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Panel B: "Malaria Endemicity in 1900" is the index of average historical malaria endemicity by Lysenko and Semashko (1968) and digitalized by Hay et al. (2004). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors clustered at the country level in parentheses. Conley standard error (8 degrees cut-off) in squared brackets. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A4: Accounting for Settlement by Europeans

Dependent Variable	Fraction of Years with Conflicts 1997-2016 - Cell Level					
Sample (Both Panels)	Excluding North Africa		Excl. North and South Africa		Excl. Countries Settled Europeans	
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Stability	0.022***	0.016**	0.027***	0.019***	0.028***	0.018***
	(0.006)	(0.006)	(0.004)	(0.005)	(0.003)	(0.006)
(Malaria Stability)^2	-0.001**	-0.001**	-0.001***	-0.001**	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Observations	359	359	333	333	362	362
Adjusted R-squared	0.200	0.645	0.287	0.661	0.302	0.723
Controls:						
Geography/Climate	No	Yes	No	Yes	No	Yes
Distances	No	Yes	No	Yes	No	Yes
Resources	No	Yes	No	Yes	No	Yes
>1 Country	No	Yes	No	Yes	No	Yes
# Ethnic Groups	No	Yes	No	Yes	No	Yes
Partitioned Cell	No	Yes	No	Yes	No	Yes
Population dens.	No	Yes	No	Yes	No	Yes
Lights, Roads	No	Yes	No	Yes	No	Yes
Country FE	No	Yes	No	Yes	No	Yes

Notes: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. Panel A: "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. Specifications in Column (1) and (2) do not contain cells located in North Africa (Morocco, Algeria, Tunisia, Lybia and Egypt), in Column (3) and (4) cells located in North Africa and in South Africa, in Column (5) and (6) cells located in countries where 100,000 inhabitants with European ancestry (South Africa, Angola, Namibia, Madagascar, Tunisia and Morocco). See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A5: Malaria, Genetic Immunities, and Conflict

Dependent Variable	Fraction of Years with Conflicts (1997-2016) - Cell Level						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.019**	0.020	0.017*	0.017**	0.014*	0.021**	0.021**
	(0.008)	(0.012)	(0.009)	(0.007)	(0.008)	(0.008)	(0.008)
Genetic Immunities (Normalized)	-0.036	0.079	-0.029	-0.089	-0.059	0.061	0.061
	(0.143)	(0.444)	(0.287)	(0.228)	(0.238)	(0.220)	(0.220)
Malaria Ec.×Gen. Imm. (Nrm.)	-0.005	-0.011	-0.022	-0.023*	-0.020	-0.029**	-0.029**
	(0.015)	(0.026)	(0.016)	(0.013)	(0.013)	(0.012)	(0.012)
Sets of Covariates:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	442	442	442	442	442	442	442
Adj. R-squared	0.180	0.424	0.599	0.623	0.627	0.673	0.673

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Genetic immunities measures the average prevalence of the sickle cell trait (Piel et al., 2013) and of the Duffy negative phenotype (Howes et al., 2011) in the population. Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A6: Alternative Measures of Conflict Intensity

	Panel A: Log Total Conflicts ACLED (1997-2016)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.122***	0.125***	0.123***	0.101***	0.089**	0.082**	0.093**
	(0.024)	(0.032)	(0.038)	(0.036)	(0.038)	(0.037)	(0.035)
(Malaria Stability)^2	-0.005**	-0.005***	-0.006***	-0.006***	-0.005***	-0.005***	-0.005***
	(0.002)	(0.002)	(0.001)	(0.001)	(0.002)	(0.001)	(0.002)
Adj. R-squared	0.159	0.398	0.590	0.617	0.626	0.645	0.676
	Panel B: Fraction of Years with Conflict UCDP GED (1989-2015)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.011***	0.011**	0.009**	0.007*	0.006	0.005	0.006
	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
(Malaria Stability)^2	-0.000	-0.000	-0.001**	-0.001***	-0.001***	-0.001**	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. R-squared	0.115	0.430	0.531	0.551	0.555	0.574	0.581
	Panel C: Log Total Fatalities ACLED (1997-2016)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.177***	0.175***	0.205***	0.181***	0.162***	0.155***	0.165***
	(0.031)	(0.043)	(0.050)	(0.049)	(0.054)	(0.053)	(0.052)
(Malaria Stability)^2	-0.006**	-0.006***	-0.008***	-0.008***	-0.007***	-0.007***	-0.007***
	(0.003)	(0.002)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)
Adj. R-squared	0.217	0.509	0.624	0.637	0.649	0.663	0.676
	Panel D: At least one Conflict ACLED (1997-2016)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.009***	0.008**	0.007**	0.004	0.004	0.004	0.004
	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
(Malaria Stability)^2	-0.000***	-0.000**	-0.000**	-0.000	-0.000	-0.000	-0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. R-squared	0.120	0.169	0.207	0.224	0.222	0.222	0.226
Observations (all panels)	442	442	442	442	442	442	442
Controls (all Panels):							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A7: Alternative Types of Conflicts ACLED (1997-2016)

	Panel A: Fraction of Years with Battles (1997-2016)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.020***	0.018***	0.020***	0.019***	0.017***	0.016**	0.017***
	(0.003)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)
(Malaria Stability)^2	-0.001*	-0.001**	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. R-squared	0.247	0.540	0.654	0.670	0.672	0.694	0.703
	Panel B: Fraction of Years with Violence Against Civilians ACLED (1997-2016)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.022***	0.017***	0.014**	0.012**	0.010	0.009	0.010*
	(0.003)	(0.005)	(0.006)	(0.005)	(0.006)	(0.006)	(0.006)
(Malaria Stability)^2	-0.001***	-0.001**	-0.001***	-0.001***	-0.001**	-0.001**	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. R-squared	0.209	0.455	0.612	0.642	0.650	0.678	0.697
	Panel C: Fraction of Years with Riots/Protests (1997-2016) ACLED (1997-2016)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria Stability	0.022***	0.010*	0.007	0.005	0.004	0.003	0.004
	(0.003)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)
(Malaria Stability)^2	-0.001***	-0.000	-0.000**	-0.000*	-0.000*	-0.000	-0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. R-squared	0.209	0.316	0.516	0.536	0.540	0.566	0.596
Observations (all panels)	442	442	442	442	442	442	442
Controls (all Panels):							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A8: Violence before 2005 and Anti-Malaria Policies after 2005

Dependent Variable	Anti-Malaria Policies (2005)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Conflicts (1998-2004)	-0.274	-0.142	-0.225	-0.134	-0.152	-0.084	-0.103
	(0.330)	(0.216)	(0.236)	(0.205)	(0.204)	(0.204)	(0.200)
Sets of Covariates:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes
Observations	306	306	306	306	306	306	306
Adj. R-squared	0.019	0.404	0.453	0.505	0.509	0.514	0.530

Notes: OLS estimates. Anti malaria policies measure the average coverage in each grid-cell of main malaria control interventions in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015). Conflicts (1997-2005) measures the fraction of years 1997-2005 with a conflict event in the cell (ACLED 1997-2016). Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A9: Alternative Binary Measure for High Malaria Risk

Dependent Variable	Fraction of Years with Conflicts 1997-2016 - Cell Level						
	Binary Measure with Malaria Stability $\in (5, 25]$						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria - High Risk DV $\in (5, 25]$	0.221***	0.152**	0.074*	0.080*	0.081*	0.078*	0.083**
	(0.053)	(0.069)	(0.037)	(0.041)	(0.044)	(0.041)	(0.036)
Observations	442	442	442	442	442	442	442
Adj. R-squared	0.099	0.383	0.616	0.619	0.627	0.646	0.672
Controls:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	Yes	Yes	Yes	Yes	Yes	Yes

Note: OLS estimates. The dependent variable is the fraction of years 1997-2016 with a conflict event in the cell (ACLED 7). See text for details. "Malaria Stability" is the index of malaria strength and stability by Kiszewski et al. (2004). "Malaria - High Risk DV" is a binary variable that identifies cells with high risk of epidemic malaria outbreaks among adults, reflected by levels of the index of malaria strength and stability by Kiszewski et al. (2004) in the interval $(5, 20]$. Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A10: Violence before 2005 and Policies (Country Level in 2005)

Dependent Variable	Anti-Malaria Policies - Country Level in 2005						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Conflicts (1998-2004)	-0.195	0.108	-0.079	-0.024	0.000	0.123	0.140
	(0.245)	(0.242)	(0.157)	(0.184)	(0.191)	(0.204)	(0.209)
Conflicts (1998-2004)	-0.155	-0.458	-0.415	-0.400	-0.445	-0.483	-0.494
×Malaria - High Risk DV $\in (0, 15]$	(0.443)	(0.415)	(0.394)	(0.387)	(0.355)	(0.349)	(0.349)
Malaria - High Risk DV $\in (0, 15]$	0.573	0.302	0.152	0.133	0.156	0.162	0.172
	(0.399)	(0.297)	(0.264)	(0.240)	(0.238)	(0.241)	(0.244)
Sets of Covariates:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	No	No	No	No	No	No
Observations	383	383	383	383	383	383	383
Adjusted R-squared	0.055	0.273	0.300	0.343	0.340	0.355	0.360

Note: OLS estimates. The dependent variable is the coverage with anti malaria policies at country level in 2005 in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015). Conflicts (1997-2005) measures the fraction of years 1997-2005 with a conflict event in the cell (ACLED 1997-2016). "Malaria - High Risk DV" is a dummy that identifies cells with low to intermediate levels of stability of malaria transmission given by levels of the index of malaria strength and stability by Kiszewski et al. (2004) in the interval $(0, 15]$. Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A11: Policies (Country Level in 2005) and Violence before 2005

Dependent Variable	A-M. Policies - Binary Indicator (Country in 2005)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Conflicts (1998-2004)	0.094	0.109	-0.021	-0.011	0.078	0.048	0.039
	(0.111)	(0.085)	(0.094)	(0.093)	(0.094)	(0.094)	(0.084)
Sets of Covariates:							
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	No	No	No	No	No	No	No
Observations	383	383	383	383	383	383	383
Adjusted R-squared	0.016	0.252	0.382	0.406	0.421	0.455	0.502

Notes: OLS estimates. The dependent variable is a binary indicator taking value 1 if the coverage with anti malaria policies at country level in 2005 in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015) was above the country average in 2005. Conflicts (1997-2005) measures the fraction of years 1997-2005 with a conflict event in the cell (ACLED 1997-2016). "Malaria - High Risk DV" is a dummy that identifies cells with low to intermediate levels of stability of malaria transmission given by levels of the index of malaria strength and stability by Kiszewski et al. (2004) in the interval $(0, 15]$. Each specification controls for the natural logarithm of the cell area. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

Table A12: Anti-Malaria Policies Robustness: Controlling for Past Conflict

Dependent Variable	Fraction of Years with Conflicts (2006-2016) - Cell Level						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Malaria - High Risk DV $\in (0, 15]$	0.016	0.045**	0.039**	0.032*	0.032	0.032	0.022
	(0.042)	(0.022)	(0.018)	(0.019)	(0.019)	(0.019)	(0.018)
Malaria - High Risk DV $\in (0, 15]$	-0.020	-0.043**	-0.050***	-0.062***	-0.062***	-0.062***	-0.057***
$\times$ Pol. Country 2005	(0.013)	(0.016)	(0.010)	(0.011)	(0.012)	(0.012)	(0.012)
Sets of Controls:							
Conflicts (1997-2005)	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Geography/Climate	No	No	Yes	Yes	Yes	Yes	Yes
Distances	No	No	Yes	Yes	Yes	Yes	Yes
Resources	No	No	No	Yes	Yes	Yes	Yes
>1 Country	No	No	No	No	Yes	Yes	Yes
# Ethnic Groups	No	No	No	No	Yes	Yes	Yes
Partitioned Cell	No	No	No	No	Yes	Yes	Yes
Population dens.	No	No	No	No	No	Yes	Yes
Lights, Roads	No	No	No	No	No	No	Yes
Country FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	383	383	383	383	383	383	383
Adj. R-squared	0.599	0.686	0.721	0.721	0.720	0.719	0.723

Notes: OLS estimates. The dependent variable is the fraction of years 2006-2016 with a conflict event in the cell (ACLED 1997-2016). See text for details. "Malaria - High Risk DV" is a dummy that identifies cells with low to intermediate levels of stability of malaria transmission given by levels of the index of malaria strength and stability by Kiszewski et al. (2004) in the interval $(0, 15]$. Anti malaria policies measures the average coverage in a country of main malaria control interventions in terms of insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), and artemisinin-based combination therapy (ACT) as of 2005 (Bhatt et al., 2015). Each specification controls for the natural logarithm of the cell area and the fraction of years with conflicts in the period 1997-2005. See the text and Tables 8 and 9 for a description of each variable and their sources. See Table A1 for descriptive statistics. The unit of observation is a 2.5 x 2.5 degree cell. Robust standard errors in parentheses clustered at the country level. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.