# EPIDEMIC SHOCKS AND CIVIL VIOLENCE: EVIDENCE FROM MALARIA OUTBREAKS IN AFRICA

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*Abstract*—This paper presents the first systematic investigation of the effect of epidemic shocks on civil violence. The identification exploits exogenous within cell × year variation in conditions that are suitable for malaria transmission using a panel database with month-by-month variation at a resolution of $1^\circ \times 1^\circ$ latitude/longitude for Africa. Suitable conditions increase civil violence in areas with populations susceptible to epidemic outbreaks. The effect is immediate, related to the acute phase of the epidemic and largest during short harvesting seasons of subsistence crops. Genetic immunities and antimalaria policies attenuate the effect. The results deliver new insights for prevention and attenuation policies and for potential consequences of climate change.

## I. Introduction

CIVIL violence is a major burden for the prospects of economic development, in particular in Africa. The reasons for outbreaks of civil violence have attracted considerable research efforts in recent years. Empirical work has focused on the role of negative economic shocks whose identification mainly relied on variation in weather and commodity prices as triggers of violence. Although health shocks and epidemic outbreaks represent existential threats for entire communities that are likely to aggravate tensions and trigger outbreaks of civil violence, their effects on civil violence have been largely neglected in the literature. A better understanding of the distinct roles of health shocks in comparison to other income shocks for the outbreaks of civil violence is paramount for the design policies to prevent civil violence.

This paper presents a first empirical investigation of the role of epidemic malaria outbreaks for civil violence in Africa. Malaria constitutes a major recurrent threat to human health. With transmission being limited to anopheles mosquitos that are sensitive to short-term changes in local bioclimatological conditions, epidemic outbreaks are geographically and temporally confined and involve a rapid increase in the number of cases and the risk of infection among susceptible populations. With estimates suggesting that between a third and half of the local population, including adults, can be infected, epidemic outbreaks represent serious shocks for entire communities. Consequently, outbreaks represent significant negative economic shocks, with costs for medical treatment that can be sizable for poor households and with a reduction in labor productivity that can be highly problematic during sensitive periods such as harvesting times of subsistence crops. While extensive reports document the sudden distress of entire communities and the risk of civil violence associated with epidemic outbreaks, to date virtually no empirical evidence exists regarding the consequences on civil violence. The preliminary results that we present indicate that spikes in malaria infections are related to significant increases in the incidence of civil violence at yearly frequencies. Infection data, however, are not suitable for a causal identification of the effect of epidemic outbreaks on civil violence as consequence of their construction.

The identification strategy applied in this paper is based on the interaction between exogenous changes in suitable conditions for malaria transmission and different levels of susceptibility of the population to malaria outbreaks (as reflected by latent prevalence of immunity among adults). This strategy makes use of the insight that "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 modifications in any of the transmission factors may completely upset equilibrium, and where the restraining influence of immunity may be negligible or absent" (MacDonald 1957, p. 45). The specific malaria epidemiology implies specific weather conditions that are required for malaria transmission and that have been isolated by algorithms developed by epidemiologists interested in early warning systems for malaria outbreaks. We use variation in these conditions at monthly frequencies at the level of cells of roughly $110 \times 110$ kilometers (at the equator). The high spatial resolution and high temporal frequency allow us to identify the effect of malaria shocks and their timing within the year in combination with variation in susceptibility, as well as the existence of attenuating and amplifying factors. The analysis is based on data from the Armed Conflict Location and Event Data Project (ACLED) for the past two decades in Africa.

The results document that the occurrence of exogenous suitable conditions for malaria transmission in a given month leads to sizable spikes in civil violence, but only in epidemic

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malaria areas that are characterized by a high susceptibility of the adult population. These findings are robust to an extensive set of sensitivity and falsification checks. These include sensitivity checks with respect to the measurement of malaria-suitable months, to the measure of susceptibility of the population, to the inclusion of controls for short-term fluctuations in weather conditions that can induce negative shocks above and beyond malaria, or to the inclusion of location-specific confounding factors. Additional results shed light on the mechanisms and the precise timing of the response to shocks within a year. The results show that violence is triggered upon impact and mostly during the acute phase of the epidemic when the risk of infection is largest. The likelihood of epidemic-related violence is even higher during harvesting months, but only in areas where latent immunities are low and only in the context of crops that exhibit short harvesting seasons. In contrast, malaria outbreaks show no effect during growing season months. We also find evidence for an attenuation of the effect of malaria shocks on violence in the presence of a higher prevalence of genetic immunities to malaria. The results of these robustness checks and additional falsification tests provide empirical support for the hypothesis that the health stress produced by sudden malaria shocks triggers civil violence among nonimmune populations and that the effect is amplified during harvesting months.

Investigating the role of health policies, we find that the effect of malaria shocks is attenuated in areas with more extensive coverage of prevention and better treatment facilities. We illustrate and quantify the implications of the results by way of a counterfactual simulation that is based on the thought experiment of the introduction of an effective malaria vaccine. The results suggest that the eradication of malaria could bring sizable reductions in civil violence, particularly in epidemic areas. Using existing long-term climate projections, we also investigate the role of climate change as a potential exacerbating factor. Projecting the corresponding changes in exposure to malaria over the next two decades reveals that climate change may induce heterogenous effects on the risk of malaria-driven conflicts across different locations. Together, the findings have far-reaching policy implications by documenting indirect effects of malaria interventions and providing relevant information for targeting short-term intervention campaigns as well as for long-term policies.

This paper contributes to the recent literature on the subnational drivers of civil violence that makes use of georeferenced databases, like ACLED, and conducts empirical analysis at the level of grid cells.[1] Work has focused on estimating the effect of economic shocks on civil violence in reduced-form analyses that exploit exogenous variation in either commodity prices (Berman & Couttenier, 2015; Berman et al., 2017; Berman, Couttenier, & Soubeyran, 2021; McGuirk & Burke, 2020, among others) or weather conditions (Almer, Laurent-Lucchetti, & Oechslin, 2017; Harari & La Ferrara, 2018). We complement these studies by investigating the hitherto empirically unexplored role of malaria shocks using data at monthly frequency, which allows us to account for all shocks that affect conflicts from one year to the other.[2] This also allows us to explore the underlying channels by considering within-year dynamics of epidemics and interactions between latent epidemics and agricultural production cycles.

This paper contributes to a small, nascent literature on the role of exposure to diseases for civil conflicts.[3] Cervellati, Sunde, and Valmori (2017) show that the disease environment and the occurrence of epidemics affects the likelihood of civil wars at the country level. Cervellati et al. (2018) document a long-run cross-sectional correlation between exposure to malaria and conflicts in Africa. Esposito and Gonzales-Torres (2020) study the role of Ebola outbreaks in three Western African countries from 2014 to 2016 for riots and demonstrations. This paper complements these studies by documenting the role of epidemic malaria outbreaks for civil violence in the whole of Africa, exploiting exogenous shocks at monthly frequencies over a period of two decades. The analysis also documents an amplified risk of violence when malaria-suitable months coincide with short harvesting periods of important subsistence crops, which has been suggested previously (Arrow, Panosian, & Gelband, 2004) but has not been documented empirically. The results also suggest that specific health policies can help the prevention of civil violence.

Finally, the paper contributes to an ongoing debate about the link between climate and conflicts (Hsiang & Meng, 2014; O'Loughlin, Linke, & Witmer, 2014; Buhaug et al., 2015; von Uexkull et al., 2016; Breckner & Sunde, 2019; for surveys, see Burke, Hsiang & Miguel, 2015, and Levy, Sidel, & Patz, 2017, for surveys). Our results point to a largely neglected channel: the changing exposure to epidemics. By combining the estimation results with existing long-term projections of temperature and precipitation at the grid-cell level, our counterfactual analysis suggests very uneven impacts across different locations, with some areas facing an increase in the likelihood of malaria-related violence and other areas facing a decline in the risk of malaria-related violence as a consequence of an increasingly dry climate.

Section II discusses the basics of malaria epidemiology and the conceptual framework and discusses anecdotal evidence. Section III presents data and empirical strategy,

[1] The role of aggregate shocks for civil conflict was initially investigated by exploiting cross-country panel data (see Berman & Couttenier, 2015, for a survey).

[2] Technically, the analysis exploits exogenous month-by-month variation in weather conditions within cells in combination with variation in the susceptibility of the population to outbreaks, while conditioning on cell × year and calendar month fixed effects thereby implicitly accounting for seasonal effects and for all unobserved factors that affect violence in a given location and year.

[3] The paper indirectly contributes to the studies of the role of life expectancy in general, and of malaria in particular, for long-term development (Cervellati & Sunde, 2011; Cervellati, Sunde, & Valmori, 2017; Cervellati, Chiovelli, & Esposito, 2020; and Depetris-Chauvin & Weil, 2018).

Section IV contains the baseline results, and section V explores the mechanisms. Section VI concludes.

## II. Conceptual Background

### A. *Epidemic Malaria in Africa*

Malaria comes in different variants. The most serious infections in Africa are due to the *tropica* variant that is caused by the *Plasmodium falciparum* parasite. *Plasmodium* parasites are heat sensitive and require a sufficiently warm environment. Transmission to humans occurs exclusively through female anopheles mosquitos. The disease has affected Africans for more than 10,000 years and remains a major source of morbidity and death on the continent. Exact figures on clinical cases are not available, but recent estimates of the death toll range from 400,000 to above 1 million per year.[4] The empirical analysis closely builds on well-established insights regarding the epidemiology of malaria. In a nutshell, epidemic outbreaks of malaria require two ingredients: a high disease susceptibility among the human population and the existence of suitable conditions for the outbreak and spread of the parasites. We briefly discuss some specificities of malaria outbreaks that are particularly relevant for the empirical strategy; the appendix (section 1 contains a more detailed account of the background).

Depending on location-specific factors that influence the stability of the transmission cycle of the pathogen, malaria can be classified as endemic or epidemic. In areas where the local geo-climatological conditions are generally favorable for the reproduction of both pathogens and vectors, the transmission cycle between humans and mosquitos is essentially uninterrupted and malaria is endemic. In areas with interrupted or more infrequent exposure to the pathogen, malaria is epidemic. In endemic areas, infection rates exhibit limited fluctuations, and infections mostly affect children, whereas in epidemic areas, infection rates are generally low or absent, but suitable weather conditions that facilitate malaria transmission over a sequence of months can materialize in sudden and intense outbreaks that affect individuals of all age groups. The reason is that the frequency of exposure to malaria infections determines the immune status and, accordingly, the susceptibility of the population in a region. In endemic areas, the repeated exposure to the pathogen of surviving individuals leads to the development of immunity, whereas in epidemic areas, the lack of frequent exposure prevents the acquisition of immunity among adults. As a result, in "areas with lower transmission . . . , infections are less frequent and a larger proportion of the older children and adults have no protective immunity. In such areas, malaria disease can be found in all age groups, and epidemics can occur."[5] Estimates suggest that the share of adults developing the disease upon inoculation of the pathogen is below 10% in endemic areas but 50% or above in epidemic areas (Griffin, Ferguson, & Ghani, 2013). This is also reflected in death rates, which are higher among adults than among children in epidemic areas (Kapesa et al., 2018).

Epidemic outbreaks require specific climatological conditions, in terms of temperature and humidity, that favor the reproduction and spread of both pathogen and vector. Epidemiologists devoted substantial effort to the development of early warning systems by identifying the specific combination of weather conditions that are suitable for malaria transmission. These conditions can be observed in endemic and epidemic areas, but their occurrence leads to epidemic outbreaks only in epidemic areas where the population has limited acquired immunities. Outbreaks tend to be geographically confined and relatively short-lived, typically lasting around three or four months, and they follow typical dynamics. The infection of few people at the onset is followed by a rapid scale-up in numbers with a spike during the acute phase, which typically corresponds to the second month of the epidemic, and a subsequent (often similarly sharp) reduction. Between one-third and above half of the total population of a community can be infected by the end of an outbreak. The risk of infection is largest during the acute phase of the epidemics, where the number of infected people increases the most. After that, during the normalization phase, infections sharply decline, though many individuals are still sick or in the process of recovery (WHO, 2016).

### B. *Malaria Epidemics and Civil Violence: Conceptual Framework*

Epidemic outbreaks of malaria put entire communities under sudden, intense stress with serious consequences for health and economic conditions. Health practitioners and international organizations have repeatedly warned that malaria epidemics can lead to outbreaks of violence as a consequence of the sudden economic and social disruption they entail for immunologically naive and socioeconomically unprepared populations.[6] These warnings also align with surveys that

[4]Malaria has killed more people than any other disease, which has led epidemiological historian Webb (2009) to label it "Humanity's Burden."

[5]Centers for Disease Control (https://www.cdc.gov/malaria/about/biology/human_factors.html). The development of acquired, or functional, immunities in endemic areas implies that infections of adults involve only mild symptoms or are even asymptomatic (see the online appendix for details on acquired immunities and the relationship between severity of infection and age in endemic areas). The decline in the share of adults developing infections upon inoculation with the level of malaria transmission stability has also become known as the "age peak-shift" phenomenon (MacDonald, 1957).

[6]For instance, WHO states that "malaria epidemics can occur when climate and other conditions suddenly favour transmission in areas where people have little or no immunity to malaria. . . . Usually regions or districts at risk are not sufficiently prepared to cope with the sudden increase of malaria transmission affecting numerous people in such a short period of time. From previous experiences and unofficial records, it is estimated that among the population at risk, 30% to 50% will develop the disease . . . depending on the rapidity and the effectiveness of the response"; as a consequence "a malaria epidemic may have disastrous consequences: disrupt the social, political and economic activity in a community [. . .] with severe political consequences." See WHO at http://www.who.int/malaria/publications/atoz/epidemicsmerajuly2004.pdf. The

illness (with malaria being the most frequently reported disease) is a main source of subjectively perceived stress in African communities.[7]

From an individual perspective, the effect of an epidemic malaria outbreak on the likelihood of violent behavior is ambiguous. Sick individuals have a reduced ability to engage in violent activities, but at the same time they may be more vulnerable to violence by others. Increased stress can also materialize in more aggressive behavioral responses to short-term threats. The potential relevance for civil violence is not straightforward to evaluate but cannot be ruled out a priori in the context of malaria, and it aligns with narratives from health practitioners.[8]

The most relevant implications of malaria outbreaks for civil violence unfold on the community level. During an outbreak, a sizable share of the community members is suddenly exposed to sickness or a large risk of sickness. As a result, healthy individuals also get under intense pressure. Sick household members and relatives need care, while health interventions and treatment impose substantial economic costs on families.[9]

From an economic perspective, malaria outbreaks also involve negative income shocks as a consequence of reduced labor productivity and labor supply. Although difficult to quantify, the consequences are considered substantial, particularly in terms of lost labor productivity. Estimates of lost workdays per year range between twenty and sixty and "bouts of malaria in agrarian households cause a decline in farm output and farm income, resulting in food insecurity and an increase in poverty" (Asenso et al., 2010, p. 7). Recent estimates by Dillon, Friedman, and Serneels (2021) based on data of piece-rate workers on a sugarcane plantation suggest that the effect of a malaria infection on worker earnings is on the order of 10% and works mainly through labor supply. In general, outbreaks are considered particularly damaging during periods of short and labor-intensive harvesting of crops that are important for subsistence. In these periods, the possibility of interpersonal substitution (e.g., mutual help with the harvest) and intertemporal labor substitution (e.g., a delayed harvest) is limited or impossible. These broader consequences have been noticed in several reports.[10] Taken together, they suggest that an epidemic outbreak likely alters the opportunity cost for conflict by imposing economic hardship and increasing difficulties in fulfilling the needs of families and the local community through work and other means. In particular, the intensity of epidemic shocks (and their health and economic consequences) has also been linked to the failure of an effective provision with health policies and insurance. Coverage with health insurance is generally limited in many developing countries (Gertler & Gruber, 2002). The fact that a large share of adults is infected or at risk of infection during a short period of time of an epidemic outbreak limits the ability of communities to accommodate the consequences of these shocks by means of formal or informal mechanisms of mutual insurance.

The remainder of this paper explores the empirical relevance of several implications that emerge from these conceptual considerations and can be summarized as follows. An elevated exposure to malaria, in terms of the occurrence of suitable conditions for malaria transmission, is conjectured to affect civil violence, but only in epidemic areas with susceptible adult populations, not in endemic areas with low susceptibility. The effect on civil violence is expected to unfold in quick reaction to the epidemic outbreaks, in light of the limited ability to smooth the consequences over time.[11] The negative economic consequences are expected to be largest during short harvesting seasons of important crops. Everything else equal, exposure to malaria risk should have smaller effects on civil violence in the presence of attenuating factors such as a greater prevalence of genetic immunity or more extensive coverage of antimalaria health policies.

### C. An Illustrative Case Study

Kenya is a useful case to illustrate the conceptual framework that underlies the empirical analysis. The country hosts a variety of different epidemiological environments, including endemic and epidemic malaria areas, as well as zones that exhibit negligible malaria risk for lack of suitability for malaria transmission. The lowlands and coastal areas feature a stable transmission environment of the pathogen, and the

UN Platform for Space-Based Information for Disaster Management and Emergency Response also warns about the social and political disruption and economic loss related to malaria epidemics (http://www.un-spider.org/disaster-management-guides/epidemic).

[7] See survey responses to CIFOR's Poverty and Environment Network (PEN) global data set collecting data on the subjectively perceived importance of environmental conditions in rural communities in sub-Saharan Africa. Figure A1 in the online appendix reports the shocks of different types. Illness (and death) and crop failure alone account for about 70% of reported shocks. Asset loss accounts for a further 20%. See also the Parima-Study: Doss, McPeak, and Barrett (2008) and McPeak, Little, and Doss (2012).

[8] The threat of infections can also trigger the release of fight-or-flight stress hormones such as cortisol, which can affect risk taking. See also section 1 of the online appendix.

[9] A large literature measures the economic costs of malaria, mostly focusing on quantifications of costs of health interventions and direct costs for treatment. The indirect costs of malaria in terms of loss of income production are more difficult to quantify. Estimates vary depending on estimated productivity of labor and across different methods (e.g., lost hours of work, estimates of value of lost production or willingness to pay to avoid infections), but the predicted losses are substantial, even in the most conservative scenarios. See Chima, Goodman, and Mills (2003) for a survey and the final report of the committee on the economics of antimalarial drugs (Arrow et al., 2004).

[10] For instance, "When people are too sick to work . . . there are economic consequences: wage earners are paid less; agriculturists may produce less (particularly if illness coincides with the harvest)" (Arrow at al., 2004). A "brief period of illness . . . that coincides with the harvest may result in catastrophic effects" (Teklehaimanot & Tozan, 2005).

[11] This is in contrast to the consequences of other negative income shocks studied in the literature, where it typically takes time for the negative consequences to unfold and become binding. For example, food reserves and consumption or production possibilities are not immediately depleted. Consistently, the literature has documented delayed effects of these shocks on conflict. The consequences of malaria-related economic distress and these other income shocks related should not be interpreted as mutually exclusive.

population is persistently exposed to infections. Malaria is endemic and mostly affects children. In the highlands, malaria is traditionally absent, but unusual suitable short-term conditions can spark the pathogen's transmission cycle. According to WHO's "Prevention and Control of Malaria Epidemics: Tutor's Guide" (2003), early monitoring and timely interventions (within the first two weeks) are crucial "since populations in these areas are immunologically naive towards malarial infections, changes that enable malaria transmission may cause explosive epidemics."[12]

Nyanza Province in Kenya, a semihighland area with low malaria transmission stability, has been subject to several epidemics and illustrates the typical patterns and consequences of outbreaks in epidemic areas. For instance, in 1999, particularly humid and warm weather during spring led to a significant increase in malaria risk. Based on the measures developed below, the risk of an epidemic outbreak in 1999 was 15% higher than in 1998 and 41% higher than in 2000. Unusually suitable conditions for malaria transmission indeed materialized in an epidemic outbreak during early summer, and the outbreak was over only in August. Epidemiologists repeatedly issued warnings about the high risk of such epidemic outbreaks and reiterated the usefulness of forecasts of such weather-related outbreaks (Hay et al., 2003).

A typical problem related to malaria outbreaks is that only a subset of cases are clinically detected as health symptoms are often not linked to malaria. As consequence and despite early warnings by epidemiologists, interventions during the 1999 epidemic in Nyanza occurred late, with the result that health facilities were overwhelmed quickly (e.g., with bed occupancy reaching 300% in the first three weeks). As often happens during outbreaks, systematic recording was not possible, and official estimates of the total number of cases for the whole region are not available. The available data suggest that it was largely adults who were affected. For instance, data for three hospitals and mobile health facilities run by Médecins Sans Frontières in the Kisii and Gucha districts reported 52,000 clinical cases of which about 80% referred to adults and around 9,000 were severe.[13]

The outbreak in Nyanza Province not only imposed health stress on entire communities and caused costs for treatments that households had to bear; it also coincided with the short harvesting season for important crops in the region.[14] Exact numbers of cases, reliable information about the number of affected agricultural workers, and estimates of the impact on labor hours or productivity of the epidemic are nonexistent. However, a suggestive piece of information comes from the cases recorded in private hospitals and health facilities in large tea plantations, like the Kericho Tea Company. In order to "maintain a healthy, productive workforce" (Shanks et al. 2005, p. 1426), these companies diagnose and treat malaria infections among their tea pickers on a continuous basis, making it a valuable, although spatially limited, source of information for epidemiologists. Records document hundreds of diagnosed malaria cases during the 1999 harvesting months.[15] As the Nyanza epidemic reached its peak, violent events soon unfolded. According to the ACLED database we used for the empirical analysis, these violent events included riots, cattle raids, and confrontations between groups of civilians. As a result of the events during the months of the epidemic, civil violence in 1999 was 55% (38%) more likely than in 1998 (2000) in the areas around Kisii and Gucha.

## III. Data and Empirical Approach

### A. Data

*Violent events.* Information about violent events comes from the Armed Conflict Location and Event Data (ACLED), which covers all African countries and contains geo-localized information about events at daily frequency. The data contain different types of events, including riots and protests, violence against civilians, and the type of actors involved (e.g., militaries, militias, civilians). The estimation analysis is conducted for the period 1998 to 2012.[16] The baseline measure of civil violence is a binary indicator variable taking the value 1 if at least one event of any type occurred in a given cell in a given period (year or month).[17] Several alternative measures of violence are considered to explore the channel and as robustness checks.

[12] See www.who.int/malaria/publications/atoz/epidemics_tg.pdf. Similarly, Chuma, Okungu, and Molyneux (2010) report that fever episodes among adults and children (over 5 years old) lasted significantly longer in districts with low transmission stability (low acute-transmission districts in the highlands of Kenya) than in high-transmission stability districts (Kenyan districts with high and intense perennial transmission).

[13] Nine districts have been severely affected—Buret, Gucha, Kisii, Mount Elgon, Narok, Nyamira, Trans Mara, Trans Nzoia, and West Pokot—but the epicenter of the epidemic took place in Kisii and Gucha. See Checci et al. (2006) and https://www.who.int/csr/don/1999_07_14a/en

[14] Information from FAO crop calendar (www.fao.org/agriculture/seed/cropcalendar/welcome.do).

[15] In Kenya, tea picking has two main peak seasons of high crop between April and June and between October and December. Figure 6 in Shanks et al. (2005) documents that during the peak of the epidemic, a plantation counted between 500 and 600 cases per month over four months during the first season. A computation of the incidence rate as a percentage of the population is difficult because of the lack of data about the permanent and seasonal workers present in the estate in 1999. Nevertheless, the numbers are substantial in view of the fact that the plantations usually employed around 1,000 workers. Data by Kapesa et al. (2018) for western Kenya suggest that the death rate due to malaria was highest among adults in epidemic areas, with 18.6 deaths per 1,000 hospital admissions (of which 62.8% were due to malaria infections).

[16] The choice of this period allows controlling for past realizations of conflict and using available weather data. Details on the construction of the gridded data are provided in section 2 in the online appendix.

[17] Alternative geocoded data for civil violence consider events that involve (more or less well identified and organized) groups and the government. Specifically, the UCDP Georeferenced Event data set (UCDP-GED) exclusively refers to violence by organized actors against other organized actors or civilians with at least 25 battle-related deaths per year. The Social Conflict in Africa database (SCAD) distinguishes between different types of events such as riots and demonstrations, but is restricted to violence directed at distinct groups or the government. In light of the conceptual framework discussed above, with its information on unorganized and small-scale violence, ACLED is the best available database for the research questions of this paper.

*Epidemic malaria areas.* Epidemiologists typically classify areas along a stable-unstable transmission gradient for malaria (see MacDonald, 1957). Due to the lack of acquired immunities, adults are exposed to a higher latent risk of malaria in areas with low to intermediate stability of malaria transmission than in areas with no or high stability (see section IIA and the online appendix). This implies that the latent risk of malaria epidemics is highest in these areas. To operationalize this information, we use data from the Malaria Stability Index by Kiszewski et al. (2004), a time-invariant spatial index of the stability and force of malaria transmission. This measure is based on the epidemiology of malaria transmission and uses ecological information about local geographic and climatological conditions to predict the long-term exposure to malaria.[18] In $1^\circ \times 1^\circ$ cells the index ranges from 0 to 34, with higher values indicating greater stability and force of malaria transmission. As a baseline, we construct a binary indicator for latent epidemic areas, *EA*, which takes the value 1 for cells with a Malaria Stability Index strictly larger than 0 and lower then or equal to 15. This baseline coding aligns with the evidence that levels of the malaria stability index above 15 to 20 are typically characterized by endemic malaria. This coding essentially implies assuming that areas at risk of epidemic malaria for adults are characterized by a low but positive index of malaria stability. Sensitivity checks regarding the measurement of latent epidemic areas are reported below.[19]

*Weather and malaria suitable months.* Information about precipitation (in mm per $m^2$) and temperatures (in degrees centigrade) at the month level is from the European Centre for Medium-Range Weather Forecasts (ECMWF) ERA-Interim data set.[20] In addition, we use the Standardized Precipitation and Evapotransipration Index (SPEI).[21]

The transmission of the *Plasmodium* pathogen depends on short-term weather conditions. The cycle of reproduction of both parasites and transmission vectors requires a sufficiently warm and humid environment for a sufficiently long period of time. To classify if a month is suitable for malaria transmission, we use the algorithm-based classification developed by Tanser, Sharp, and Le Sueur (2003) for the purpose of an early warning indicator of imminent malaria outbreaks.[22] We code a binary variable labeled Malaria Suitable Month (*MSM*) using data at monthly frequency based on the local weather conditions in a given grid cell during the proceding twelve months. The indicator of whether malaria transmission is possible takes the value 1 if conditions that are suitable for transmission of the pathogen are met in a given cell and month and 0 otherwise. In particular, the indicator takes the value 1 if all of the following conditions are satisfied: (a) the average monthly rainfall during the past three months exceeds a threshold of 60 mm/$m^2$; (b) rainfall exceeds 80 mm/$m^2$ in at least one of last three months; (c) no month in the past twelve months has an average temperature below 5°C; and (d) average temperature in the past three months exceeds 19.5°C plus the standard deviation of monthly temperature over the past twelve months.

Tanser et al. (2003) document that the index has high predictive power for the absence of malaria-suitable conditions but less predictive power for actual outbreaks, thereby capturing necessary conditions for elevated malaria transmission rather than providing sufficient conditions. The interpretation of effects based on the variable *MSM* is therefore along the lines of an intention-to-treat analysis.

*Malaria infections: Projections of clinical cases.* Considerable effort by epidemiologists went into the construction of a comprehensive database of the dynamics of malaria infections among the adult populations in Africa. The best time-varying disaggregate data on malaria incidence in Africa available to date come from projections of clinical incidence of *Plasmodium falciparum* malaria assembled by Bhatt et al. (2015). These data are based on surveys from various sites for 35 sub-Saharan African countries over the years 2000 to 2015, which have been interpolated across space to obtain a map of malaria incidence.[23]

*Other variables, sources, and summary statistics.* The analysis also makes use of a rich set of time-invariant and

[18] The transmission of the pathogen between humans and vectors depends on local bio-climatological conditions. See section 1 in the online appendix for more detailed discussions of the short-term conditions for malaria transmission and the construction of the malaria ecology index.

[19] The construction of the measure of malaria stability is discussed in more detail in section 2 of the online appendix. There we also present a validation using data for projected malaria infections and alternative semiparametric empirical strategies that identify latent epidemic areas more flexibly. Figure A4 in the online appendix shows that areas with malaria stability above 15 to 20 are characterized by high endemicity in the African population around year 1900.

[20] The ERA-Interim data set has the advantage of being based on data reanalysis involving model simulations of past events that include the incorporation of historical observations taken from various sources, such as weather stations, satellites, and sensors, instead of relying on gauge data. To our knowledge, these are the highest quality weather data available. See also section 2 in the online appendix for details.

[21] The SPEI index is a combination of the Palmer Drought Severity Index (PDSI), which is based on supply and demand for water, and the Standardized Precipitation Index (SPI). The SPEI measures drought severity, intensity, and duration, but it also allows comparisons of drought severity through space and time, including different drought types. See Vicente-Serrano, Begueria, and Lopez-Moreno (2010).

[22] This index was constructed following the 20th Report of the WHO Expert Committee on Malaria (2000), which called for the need for reliable indicators based on highly disaggregated meteorological information to be used for the prevention of malaria epidemics, stating that "increasing numbers of malaria epidemics have been recently documented throughout the world, particularly in Africa. Areas become epidemic when conditions that normally limit transmission change radically as a result of abnormally heavy rains, long periods of increased humidity and temperature" (WHO, 2000, p. 6). The same index has been used by Kudamatsu, Persson, and Strömberg (2016) to study the effect of malaria shocks for child mortality at yearly frequencies.

[23] Figure D1 in the online appendix illustrates the cross-sectional variability of (average) projected malaria incidence across the respective grid cells in Africa for the year 2000, the first year for which the data are available. In the appendix we also report a validation of the specific role of malaria-suitable months for clinical incidence in epidemic and endemic areas; see figure V1 and table V1.

TABLE 1.—MALARIA INFECTIONS AND VIOLENCE

| | Violent Events: ACLED Yearly Data | | | |
|---|---|---|---|---|
| Dep. Variable | (1) | (2) | (3) | (4) |
| Clinical infections of malaria | 0.021*** (0.005) | 0.020*** (0.005) | 0.020*** (0.005) | 0.018*** (0.005) |
| Weather | No | Yes | Yes | Yes |
| Weather lag | No | No | Yes | Yes |
| Cell FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Violence lag | No | No | No | Yes |
| Observations | 21,853 | 21,853 | 21,853 | 21,853 |
| *R*-squared | 0.473 | 0.473 | 0.474 | 0.477 |
| Number of cells | 1,681 | 1,681 | 1,681 | 1,681 |

The dependent variable is a binary indicator variable taking the value 1 if at least one conflict event (ACLED data set) was observed in the given cell in the given year. "Clinical Incidence of Malaria" is a detrended and standardized projection of clinical incidence of *Plasmodium falciparum* malaria (per 1,000) obtained by interpolating across space and over time available malaria prevalence data retrieved from surveys using Bayesian geo-statistical models that employ a large number of environmental and sociodemographic covariates; see section 2 in the online appendix and Bhatt et al. (2015) for details. The "Weather" controls include average annual temperature, average annual precipitation; and average level of the Standard Precipitation and Evapotranspiration Index (SPEI); "Weather lags" include weather controls for the previous two years. OLS estimates (linear probability model). Standard errors clustered at the cell level are reported in parentheses. The unit of observation is a $1 \times 1$ degree cell. Panel data from 1998 to 2012 at yearly frequency. Significant at ***1%, **5%, and *10%.

time-varying variables as controls or for additional exercises, which are discussed along the way when needed. (Further details on the construction, coding, and data sources for all variables used in the analysis are provided in section 2 in the online appendix). A summary description of the main variables used in the analysis is reported in tables D1, D2, and D3 in the online appendix, and summary statistics of the main variables are reported in tables S1 and S2.

### B. Malaria Infections and Violence

Table 1 reports the results of regressing civil violence on the projection of malaria infections at yearly frequency. The estimation exploits within-cell variation over time by including cell and year fixed effects. The results document a positive and significant effect of within-cell variation in malaria infections on civil violence. The inclusion of cell fixed effects implies an effect of unusual infection rates in terms of deviations from the cell-specific mean over the observation period. The point estimate is unaffected when controlling for weather conditions, in terms of temperature, precipitation, and SPEI (and their lags) or for lagged incidence of violence.

These findings should be interpreted as suggestive since the data on projected clinical cases suffer from serious limitations that prevent their use for identification of a causal effect of malaria infections on violence and an exploration of the mechanisms. Furthermore, the data do not allow distinguishing the effect of endemic and epidemic infections. Most important, the malaria incidence data have been assembled with the goal of mapping the evolution of malaria incidence in the medium term. Consistent with this goal, the cell-level data have been constructed by epidemiologists using information from clinical surveys conducted at various locations, including information about clinical cases involving children. This information has then been interpolated across space and time using a large set of socioeconomic covariates to obtain an estimate of projected clinical incidence for adults. Finally, the information about projected incidence is available only at a yearly frequency. This implies that measures of projected malaria incidence are not suitable for a causal identification of the effect of epidemic outbreaks of malaria on civil violence, particularly in light of the short-run (within-year) dynamics of malaria shocks described in the conceptual discussion of section IIB.[24] Despite these shortcomings, the findings provide a first explicit exploration of the raw relationship, which has been prevented so far by the lack of highly disaggregated panel data for the whole of Africa.

### C. Identification Strategy

To make progress toward identifying the causal effect of malaria epidemics on civil violence, this section develops an approach that exploits exogenous variation in the short-term conditions for malaria transmission in latent epidemic areas. In particular, the identification strategy is based on the interaction between the differential susceptibility of adults to malaria infections, reflected in the coding of epidemic (as opposed to endemic) areas, *EA*, and the exogenous variations in the exposure to malaria outbreaks, reflected in monthly within-grid-cell variation in weather conditions particularly suitable for malaria transmission, malaria-suitable months *MSM*. We estimate the linear probability model:

$$Violence_{i,c,t} = \alpha MSM_{i,c,t} + \beta MSM_{i,c,t} \times EA_{i,c} + X'_{i,c,t}\Gamma + Z'_{i,c,t-1}\Delta + \Phi_{i,c,t} + u_{i,c,t}, \quad (1)$$

where $Violence_{i,c,t}$ is a binary indicator of civil violence in cell $i$ in country $c$ in period $t$. In the yearly data, $t$ reflects a year; in the monthly data, $t$ is a month. The latent risk of epidemic malaria is measured by the time-invariant binary indicator, $EA_{i,c}$, for cell $i$ (in country $c$). The variable $MSM_{i,c,t}$ denotes variation in malaria exposure in terms of months with suitable conditions for malaria transmission. In the analysis exploiting yearly variation, *MSM* represents the number of malaria-suitable months during the year of observation. In the monthly panel data, the variable *MSM* is an indicator that takes the value 1 if in a given month in a given cell, the conditions suitable for malaria are satisfied, and 0 otherwise. The coefficient of interest $\beta$ captures the effect of suitable short-term conditions for malaria in period $t$ in cell $i$ in country $c$ with latent epidemic malaria risk (as compared to areas with latent endemic malaria).

Reverse causality is ruled out by construction since short-run weather conditions in high and low malaria-risk areas are

[24] Survey data are not available every year, and measures of clinical incidence tend to record the medium-term effect of exposure over the past few years. This imposes serious limitations for an attempt at causal identification, because the possibility of ruling out that spikes in malaria infections are driven by previous spikes in conflicts within the year is ruled out by construction. See section 2 in the online appendix for a detailed description of data construction and quality.

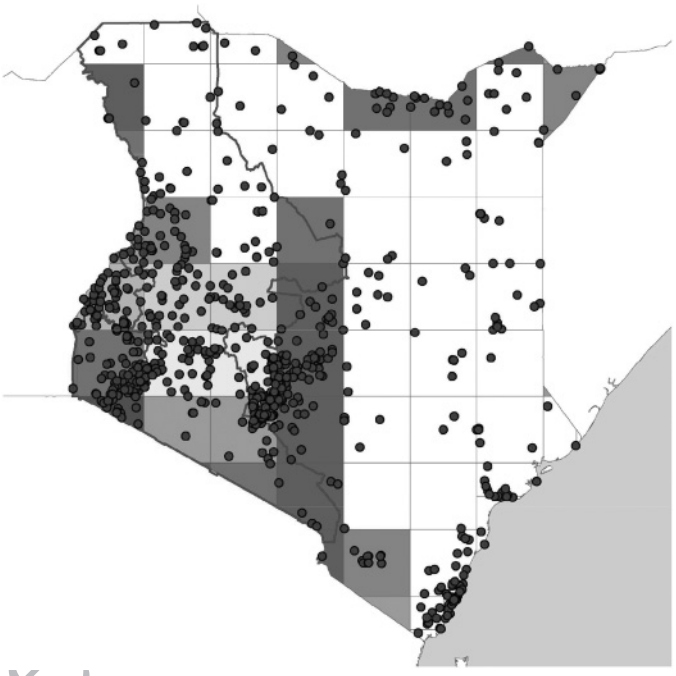
exogenous to civil violence. The vectors $X$ and $Z$ contain additional contemporaneous or lagged covariates. Depending on the particular specification and the panel data frequency, the covariates include the main effect of $EA_{i,c}$, weather conditions (and their lags), and the lagged dependent variable, among others. The vector $\Phi_{i,c,t}$ generically indicates the inclusion of different types of fixed effects at the level of cell $i$, country $c$, or period $t$, and possibly their interactions, which are included in isolation or jointly, depending on the specification. For instance, the baseline specification at yearly frequency exploits within-cell variation over time with cell and year fixed effects (so that $\Phi_{i,c,t} = \phi_i + \phi_t$). When using data at monthly frequency, we estimate specifications equivalent to a two-way (cell and month) fixed-effects model. Alternatively, we consider more flexible specifications, including cell×year and calendar month fixed effects. The monthly panel data also allow us to study the within-year dynamics of malaria risk and violence and counterfactual exercises and placebo tests (e.g., using, for example, the occurrence of *MSM* in future months).

As baseline, we estimate linear probability models with robust standard errors that allow for arbitrary heteroskedasticity and autocorrelation of the error term within a given cell. As a robustness check, we also allow for spatial correlations with neighboring cells (in particular, Conley standard errors for a range of 400 km).

*Graphical illustration.* The logic of the identification strategy can be illustrated considering again the case of Kenya. Figure 1 depicts the map of Kenya overlaid with $1^\circ \times 1^\circ$ cells used as unit of observation in the empirical analysis. The picture discriminates between latent epidemic areas that are characterized by intermediate levels of malaria stability and areas that are at low risk for adults, including endemic areas as well as arid or semidesert areas with zero malaria stability where the pathogen cannot be transmitted. Grid cells with a shadow indicate epidemic areas. The intensity of the shadow relates to the standard deviation of malaria-suitable months across the years. Adults are more at risk in darker cells that are characterized by lower stability of malaria transmission. The figure also depicts the spatial distribution of violent events over the observation period, reflected by dots. The area involving the 1999 epidemic mentioned in section IIIC is located in the intermediate malaria-stability area in the South west corner of the map and shows a spatial cluster of violent events. The identification strategy captures the differential impact of the occurrence of suitable conditions for malaria transmission (*MSM*) in epidemic versus nonepidemic malaria areas (*EA*) (in terms of the differential effects across shaded and white cells).

To maximize external validity, the empirical analysis is performed for the entire African continent. Figure 2a illustrates the variability of suitable conditions for malaria transmission within cells by depicting the standard deviation of malaria-suitable months (*MSM*) in each cell for the entire continent.

FIGURE 1.—MALARIA RISK AND VIOLENT EVENTS IN KENYA



Epidemic areas, $EA = 1$ are identified by a shadow. The white cells have no or high (endemic) malaria transmission. Darker cells imply higher standard deviations of suitable transmission conditions (*MSM*). The dots represent single episodes of civil violence (ACLED database).

The latent epidemic areas with high susceptibility of adults for malaria infections under suitable conditions ($EA = 1$) are depicted with a light shadow.[25] Figure 2b depicts the share of years with at least one violent event over the observation period. Again, light shadowing depicts latent epidemic areas ($EA = 1$). Although the empirical analysis exploits variation over time in the risk of outbreaks of malaria (reflected by *MSM*) and violence rather than cross-sectional variation the figure offers a first visual impression of the unconditional correlation between malaria risk and violence in areas with latent epidemic malaria.

## IV. Malaria Risk and Civil Violence

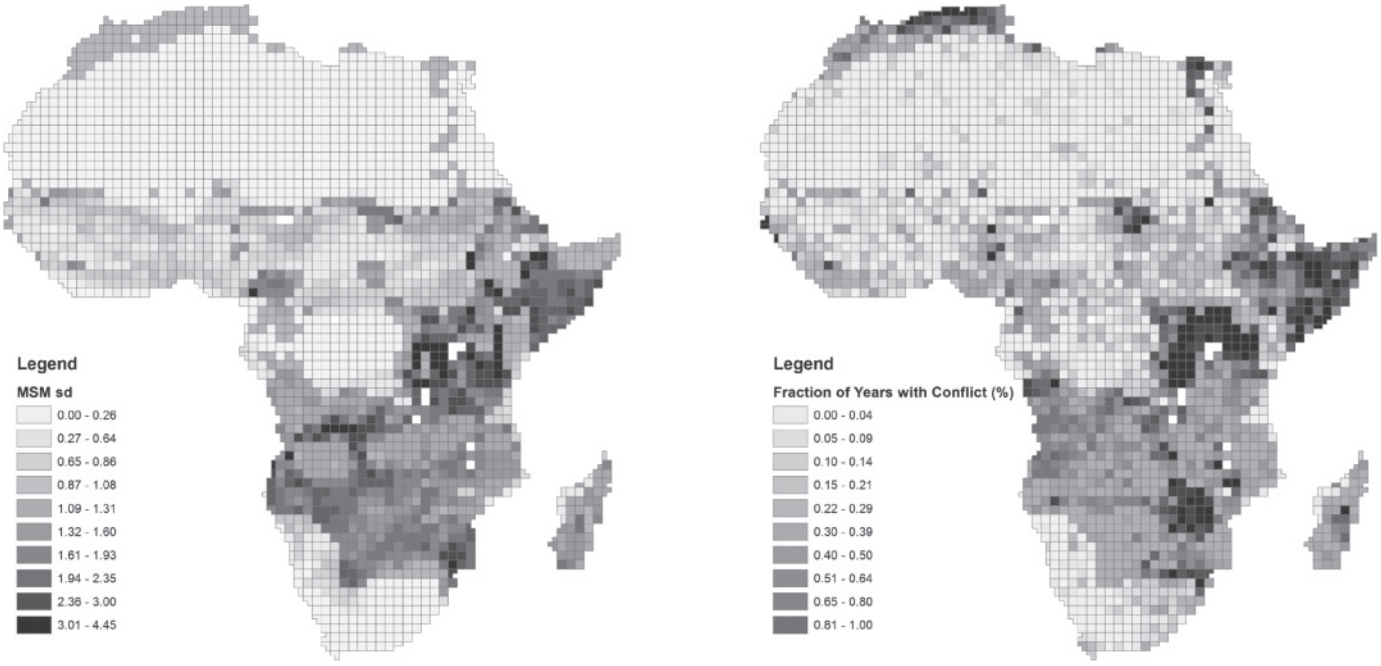
This section presents the baseline results followed by a brief discussion of the results of several sensitivity checks. We also discuss the main possible confounders and the results of several corresponding robustness exercises.

The baseline results are reported in table 2.[26] Columns 1 to 3 report estimates of the empirical specification reported in equation (1) at yearly frequencies. Column 1 contains the results of a difference-in-difference specification that isolates the differential effect of malaria risk in latent epidemic

[25] The spatial distribution of the original malaria stability index that is used as information to construct the indicator latent epidemic areas is reported in figure D2 in the online appendix.

[26] The coefficient of the time-invariant indicator *EA* is not identified in specifications with cell fixed effects.

FIGURE 2.—MALARIA RISK AND VIOLENCE: AFRICA




(a) Malaria Risk, MSM (sd) (b) Violent Events, ACLED

(a) The standard deviation of malaria-suitable months (*MSM*) built following Tanser et al. (2003) (see text for details) in latent epidemic areas ($EA = 1$, light shadow), and low malaria risk cells ($EA = 0$). (b) The spatial distribution of violent events (fractions of years with at least one violent event over observation period) in latent epidemic areas ($EA = 1$, light shadow) and low-risk cells, ($EA = 0$).

areas in a two-way, fixed-effects specification.[27] Columns 2 and 3 present corresponding results when allowing for time-varying country-specific effects and when accounting for lagged conflict incidence, respectively. The findings document throughout that latent epidemic outbreaks of malaria (in terms of *MSM*) do not have a significant effect on violent events per se in nonepidemic areas, but the occurrence of an additional month with suitable conditions for malaria increases the risk of violence by 1.3 to 1.5 percentage points in epidemic areas. This corresponds to 7% to 8% compared to the unconditional probability of 18.6% and the model accounts for about 50% of the variation in violent events at the yearly level.

Spikes in malaria risk are closely confined in terms of time and space and follow well-studied dynamics, as discussed in section IIB. The index of suitable conditions for malaria transmission (*MSM*) has been designed by epidemiologists specifically for the purpose of identifying temporary increases in transmission risk at a monthly frequency and a high level of geographic precision. The estimation of the empirical model at the monthly level makes use of this information and allows us to exploit variation over time within cells and years and to account for seasonal patterns in the likelihood of conflicts. The specification at the month level also allows controlling for cell×year fixed effects and thereby for all time-varying factors that affect the average level of violence in a given cell across years (above and beyond monthly changes in malaria risk). Thus, the effect of elevated malaria risk (in terms of *MSM*) in a given month on the incidence of violence in the same month is identified by comparing months with suitable conditions for malaria transmission with months that do not exhibit suitable conditions, in the same cell within the same year.

Columns 4 to 6 show the respective results. The results in column 4 correspond again to standard difference-in-difference estimates and confirm that the occurrence of suitable conditions for malaria transmission (*MSM*) in a given cell and month leads to a significant increase in the incidence of civil violence in latent epidemic areas. Column 5 reports the results for a specification that also controls for cell×year effects and calendar month fixed effects. The coefficient estimates are effectively unchanged. Column 6 confirms these patterns by extending the specification to a dynamic panel model that also controls for the incidence of violent events in the previous month.[28]

[27] For space reasons we report results for the differential effect of malaria risk in epidemic areas, conditional on weather conditions and their lags. Alternative specifications are reported in the online appendix, as discussed below.

[28] Note that the dynamic panel estimates of column 6 are based on a high number of time series observations at monthly frequencies (around 150), which limits the problems from dynamic panel (Nickell-)bias. The results are also confirmed when estimating extended dynamic panel specifications that include cell×month fixed effects to control, for instance, for recurrent events in a cell-month, such as ethnic or religious festivities or religious celebrations. Table A5 in the online appendix documents the robustness of the baseline result even in very demanding specifications.

TABLE 2.—MALARIA RISK AND VIOLENCE: BASELINE RESULTS

| Dependent Variable | Violent Events, ACLED | | | | | |
|---|---|---|---|---|---|---|
| | Yearly | | | Monthly | | |
| Panel Data Frequencies: | (1) | (2) | (3) | (4) | (5) | (6) |
| Malaria-suitable month(s) | −0.008* | −0.005 | −0.005 | −0.003 | −0.002 | −0.002 |
| | (0.004) | (0.005) | (0.005) | (0.002) | (0.002) | (0.002) |
| Mal. Suit. Month(s) × Epidemic Area | 0.015*** | 0.014** | 0.013** | 0.008*** | 0.007*** | 0.007*** |
| | (0.005) | (0.005) | (0.005) | (0.003) | (0.003) | (0.002) |
| | [0.007] | [0.006] | [0.006] | [0.003] | [0.003] | [0.003] |
| Weather | Yes | Yes | Yes | Yes | Yes | Yes |
| Weather lags | Yes | Yes | Yes | Yes | Yes | Yes |
| Conflict lag | No | No | Yes | No | No | Yes |
| Fixed effects | | | | | | |
| Cell | Yes | Yes | Yes | Yes | No | No |
| Year | Yes | No | No | No | No | No |
| Country × Year | No | Yes | Yes | No | No | No |
| Cell × Year | n.a. | n.a. | n.a. | No | Yes | Yes |
| Month × Year | n.a. | n.a. | n.a. | Yes | No | No |
| Month FE | n.a. | n.a. | n.a. | No | Yes | Yes |
| Observations | 38,130 | 38,130 | 38,130 | 457,560 | 457,560 | 457,560 |
| *R*-squared | 0.467 | 0.521 | 0.522 | 0.242 | 0.413 | 0.436 |
| Number of cells | 2,542 | 2,542 | 2,542 | 2,542 | 2,542 | 2,542 |

The dependent variable is a binary indicator variable taking the value 1 if at least one conflict event (ACLED data set) was observed in the given cell in the given period. "Malaria-suitable month" is an indicator variable for conditions that are suitable for malaria transmission in a given cell and month (in the yearly panel is the total number of MSM in a year). The "Epidemic Area" variable is a binary indicator for epidemic areas characterized by small to intermediate malaria stability of transmission (see text for details). "Weather" controls include the average temperature, the average precipitation, and the effective rainfall (the Standard Precipitation and Evapotranspiration Index (SPEI)) in the respective year or month. The "Weather lags" variables include the first two lags in the yearly panel and the twelve lags in the monthly panel. Panel data from 1998 to 2012 at yearly and monthly frequencies. The unit of observation is a $1^\circ \times 1^\circ$ degree cell. OLS estimates (linear probability model). Standard errors clustered at the cell level are reported in parentheses, (·), and Conley standard errors allowing for spatial and serial autocorrelation up to a threshold of 400 km are reported in brackets. Significant at ***1%, **5%, and *10%, computed using the largest standard errors (cell clusters or Conley) of each specification.

The estimates deliver two important findings. First, the point estimate of the coefficient of interest turns out to be essentially unaffected by the specification of the empirical model. Compared to an unconditional probability of violence around 4.4%, the coefficient estimates of 0.007 to 0.008 in columns 4 to 6 imply a sizable increase in the risk of violence of 16% to 18% when suitable weather conditions for malaria outbreaks occur in epidemic areas. The magnitude of this effect is larger in the estimates at monthly frequency compared to those at yearly frequency, which essentially involve averaging variation in malaria risk and violence across different months of the same year. Relating the size of this effect to existing estimates of the effect of variation in income or productivity on conflict incidence is not straightforward and is complicated by the fact that the data on malaria incidence suffer from serious limitations. Assuming that the occurrence of suitable conditions for an epidemic outbreak of malaria leads to a monthly incidence of malaria cases among adults of 20% (in line with numbers reported by Shanks et al., 2005, figures 5 and 6), and combining this with a decrease in earnings due to a malaria infection of around 10% (as in Dillon et al., 2020) delivers an estimated decline in average income of 2%. Thus, combining this with the estimates of columns 4 to 6, the implied effect of a 1% decrease in income on the probability of conflict would be on the order of 8% to 9%, which is consistent with estimates in the literature.[29]

[29]For instance, Berman et al. (2021) report an elasticity in the probability of conflict for a 1% increase in agricultural productivity of −4.4% to −11.3% in comparison to a decrease in the probability of conflict in response to a 1% increase in rainfall-induced GDP growth of 9.5% in the estimates of Miguel et al. (2004).

Second, the diff-in-diff specification at monthly frequencies of column 4 explains about 20% of the variation in the data, while accounting for cell×year specific effects almost doubles the share of variation explained by the empirical model. This suggests that cell×year specific shocks like those emphasized in the literature indeed explain a considerable amount of variation in civil violence. In this respect, the results confirm the relevance of mechanisms at this level of variation that have been identified in earlier studies. At the same time, the stability of the effect of the occurrence of suitable conditions for malaria transmission in epidemic areas suggests that the effect of malaria risk is unaffected by, or orthogonal to, all cell-year specific shocks.

*Robustness.* To explore the robustness of the baseline findings, we conducted a large set of additional analyses (the details are in the online appendix). In particular, the baseline findings are robust to alternative econometric specifications (tables A1–A6), different thresholds of malaria stability or using weighting functions (figure A7), a coarser grid with cells of $2^\circ \times 2^\circ$ (table A7), alternative codings of violent events or sample composition (tables A8–A10 and figure A8), and the use of alternative data for violent events (table A11). We also replicated the analysis accounting for interactions of malaria shocks with an extensive set of time-invariant, geographic characteristics that might represent confounders, including elevation, ruggedness, land suitability for agriculture (tables A12–A15), land cover (tables A16–A17), population and economic development (tables A18–A20) and infrastructure (tables A21–A23), other (placebo) diseases such

as dengue (table A24), risk of trypanosomiasis measured by tsetse suitability (table A25), or prevalence of HIV in the population (table A26), as well as for alternative codings for epidemic areas (figure A9 and tables A27–A28). Finally, the results are robust to alternative codings of suitable conditions for malaria transmission (malaria-suitable months), including specifications with interactions with weather conditions (tables A29–A30), nonlinear controls for weather conditions and floods (tables A31–A32), placebo measures of malaria-suitable months (tables A33–A34), and when controlling for other confounders like soil moisture, vegetation, or pests (tables A35–A38).

## V. Exploring the Mechanisms

This section studies the mechanism behind the baseline results by exploring the testable implications, discussed in section IIB, in terms of the types of violent events, the timing of the effect in response to malaria shocks, interactions with the crop cycle, and the existence of attenuating factors.

### A. *Types of Violent Events*

The effect of a malaria shock is expected to be related to civil violence that reflects the sudden stress imposed by the shock rather than geo-strategic motives for violent confrontations. This hypothesis can be investigated by considering different types of violent events (in terms of riots and protests, violent confrontations involving militias and civilians, violence involving actions by militarized rebel groups, and struggles between military forces and rebels) based on information about the actors involved in a violent event. The results for these different types of violence show that spikes in malaria risk (in terms of *MSM*) increase the likelihood of unorganized violent events involving rioters or protesters or confrontations between militias and civilians, while there is no evidence for an effect on violence involving organized groups, such as rebel groups or the government (table A39). This suggests that epidemic outbreaks do not relate to the opportunity costs for participating in conflicts that are more likely related to political motives. In additional analysis, we also find no evidence of a significant effect of malaria risk on geo-strategic events in terms of confrontations involving rebel groups and between militaries and rebels and events involving only militaries, including nonviolent events that involve changes in the control over territories and changes of headquarters.[30] These findings provide a slightly different picture from results found by earlier contributions that consider other types of income shocks.[31]

### B. *Timing of Violent Responses during Phases of Epidemic Outbreaks*

The rapid scale-up in the level of health-related and economic stress in the population and the limited ability to smooth the consequences over time are conjectured to lead to immediate responses in terms of the incidence of civil violence. In contrast to economic shocks, such as weather-related crop failures or income shocks related to commodity prices, whose negative effects have been documented to take some time to unfold, the anecdotal evidence points to the risk of civil violence outbreaks being closely related to the dynamics of malaria epidemics. By isolating the effect of malaria risk within a year, the following analysis explores the evolution of the dynamic response of violence to malaria shocks. In this respect, the analysis of high-frequency data helps to disentangle different mechanisms behind the reduced-form effects already documented.

*Timing: Lags and leads.* In line with warnings issued by development practitioners, estimates obtained with an extended empirical specification that includes the effects of malaria-suitable months during the past two months provide evidence that spikes in malaria risk affect the incidence of violence on impact. At the same time, falsification tests with controls for malaria-suitable months during the following two months deliver no evidence for the existence of anticipation effects related to future malaria shocks. The results also hold when considering an extended specification with cell×year and calendar month fixed effects or when including both lags and leads (figure A10 and tables A43–A44).

*Sequence of malaria suitable months.* The epidemiological literature suggests that the occurrence of suitable conditions for malaria transmission for a number of months in a row increases the risk of malaria transmission due to an amplification of the pathogenic pressure. The empirical results align with this effect and reveal that compared to baseline, the response in terms of violence is larger during months characterized by extended sequences of malaria-suitable conditions, although the number of months fulfilling this condition is lower (table A45).

*Risk of infection during different phases of epidemic outbreaks.* As discussed in section IIB, the risk of infection is largest during the acute phase of the epidemic when the number of infected people increases the most. The risk of

[30] Table A40 reports results for extended specifications. Table A41 reports similar results for data from the SCAD database. Table A42 reports results for the UCDP-GED data. These data only contain events involving organized actors and leading to at least 25 battle-related deaths, and are therefore not as suited to investigating the research question as data that also incorporate smaller events. In fact, the unconditional probability of these events is very low at monthly frequencies, and we find no significant effect with this data set.

[31] For instance, Berman et al. (2017) find that an increase in mineral prices leads to more riots and protests and more violence against civilians, but also to more battles. Similarly, Harari and la Ferrara (2018) find that negative weather shocks during the growing season lead to an increased incidence of conflicts related to battles, rebels, and violence against civilians.

TABLE 3.—VIOLENCE DURING DIFFERENT PHASES OF EPIDEMIC OUTBREAKS

| | Violent Events: ACLED Monthly Panel | | | | | |
|---|---|---|---|---|---|---|
| Dependent Variable | (1) | (2) | (3) | (4) | (5) | (6) |
| *First Month* (Onset Phase) | | | | | | |
| MSM | −0.000 | −0.000 | −0.000 | −0.001 | −0.001 | −0.001 |
| | (0.003) | (0.003) | (0.003) | (0.003) | (0.003) | (0.003) |
| MSM × Epidemic Area | 0.000 | 0.001 | 0.001 | 0.001 | 0.002 | 0.002 |
| | (0.003) | (0.003) | (0.003) | (0.003) | (0.003) | (0.003) |
| *Second Month* (Acute Phase) | | | | | | |
| MSM | | −0.002 | −0.002 | | −0.002 | −0.003 |
| | | (0.003) | (0.003) | | (0.003) | (0.003) |
| MSM × Epidemic Area | | 0.009*** | 0.009** | | 0.009*** | 0.009** |
| | | (0.004) | (0.004) | | (0.004) | (0.004) |
| *Third Month* (Normalization Phase) | | | | | | |
| MSM | | | −0.002 | | | −0.002 |
| | | | (0.003) | | | (0.003) |
| MSM × Epidemic Area | | | −0.000 | | | −0.001 |
| | | | (0.004) | | | (0.004) |
| Weather | Yes | Yes | Yes | Yes | Yes | Yes |
| Weather lag | Yes | Yes | Yes | Yes | Yes | Yes |
| Cell FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Month FE | Yes | Yes | Yes | No | No | No |
| Month × Year FE | No | No | No | Yes | Yes | Yes |
| Observations | 457,560 | 457,560 | 457,560 | 457,560 | 457,560 | 457,560 |
| *R*-squared | 0.236 | 0.236 | 0.236 | 0.242 | 0.242 | 0.242 |
| Number of cells | 2,542 | 2,542 | 2,542 | 2,542 | 2,542 | 2,542 |

Replication of analysis of table 2 by discriminating between different types of malaria-suitable months depending on whether they identify the onset of risk, the acute phase during the second month of the sequence, or the normalization phase in the third month. The onset of malaria risk is coded as sequences of months such that $MSM_t = 1$ and $MSM_{t-1} = 0$. Incidence is coded as $MSM_t = 1$, $MSM_{t-1} = 1$, and $MSM_{t-2} = 0$. Prolonged incidence is coded as $MSM_t = 1$, $MSM_{t-1} = 1$, $MSM_{t-2} = 1$, and $MSM_{t-3} = 0$. See text for details. Significant at ***1%, **5%, and *10%.

infection is lower at the onset (when the risk of infection is still low) and during the normalization phase. Since epidemics typically unfold over the course of three or four months, we investigate the existence of heterogeneous effects on the first, second, or third consecutive month of a sequence of malaria-suitable months, which can conceptually be interpreted as onset, acute, and normalization phases.

The results in table 3 are consistent with the conjecture that the response of violence follows the time-varying intensity of the risk of infection in the population. The results show that the effect of malaria risk on violence tends to be concentrated in the acute phase (where the effect is about 20% larger than in the baseline specification). The effect on violence is not significant during the onset and during the normalization phase. These findings document that not all months with suitable conditions for malaria transmission affect the incidence of civil violence equally and provide further insights into the nature of these short-term shocks related to the discussion above.

### C. *Labor Productivity Shocks during Agricultural Cycles: Harvesting and Growing Seasons*

The literature has studied the role of adverse income shocks as either triggered by low precipitation during growing seasons or by external commodity price shocks that alter production, the value of harvests, or the cost of food, respectively. In contrast, the consequences of malaria are related to health problems, costs for treatment, and reductions in labor productivity. However, the role of health shocks for triggering violence has not been explored before. In particular, the consequences for labor productivity and the ability to work are expected to have the biggest negative economic impact when they coincide with short harvesting seasons of important crops, up to the dramatic outcome of a failure of harvesting. In spite of the host of anecdotal evidence, an empirical test of these predictions is still lacking. We exploit again the monthly frequency of the data and explore the interactions between malaria risk and the agricultural cycle in a given cell.

*Harvesting months.* Data on harvesting months for different crops are not readily available at the grid cell level. To identify harvesting months during crop cycles in each grid cell, we extracted information on the harvesting seasons for all the crops from the FAO crop calendar, which lists information at the level of agro-ecological regions for the whole of Africa.[32] We use several alternative definitions for harvesting periods. Harvest months are coded as a binary indicator that takes the value 1 during the harvesting months for each specific crop in each cell. To explore the role of their importance for the subsistence of the local population, we bundle crops in terms of energy content. To proxy for the possibility of

[32]Notice that different geo-climatological conditions imply that the same crop may have different harvesting seasons in different geographical areas. The data report crop cycles at the level of different administrative units (ranging from regions to groups of municipalities) for different countries or even agri-ecological areas that do not coincide with administrative regions. This makes the aggregation into grid cells an extremely time-consuming process. The measures are constructed based on satellite images; see online appendix table D2 for details.

TABLE 4.—MALARIA SHOCKS DURING SHORT HARVESTING MONTHS

| Dependent Variable | Violent Events: ACLED Monthly Panel | | | |
|---|---|---|---|---|
| | Only Epidemic Areas ($EA = 1$) | | | |
| Sample | (1) | (2) | (3) | (4) |
| Malaria-suitable month | 0.006** | 0.005** | 0.005* | 0.005** |
| | (0.003) | (0.003) | (0.002) | (0.002) |
| Harvest month | −0.000 | −0.000 | −0.008*** | −0.009*** |
| | (0.003) | (0.002) | (0.003) | (0.003) |
| MSM × Harvest Month | −0.000 | 0.005 | 0.014*** | 0.015*** |
| | (0.003) | (0.004) | (0.004) | (0.005) |
| Observations | 141,300 | 141,300 | 141,300 | 141,300 |
| *R*-squared | 0.450 | 0.450 | 0.450 | 0.450 |
| Number of cells | 785 | 785 | 785 | 785 |
| Specification (both panels) | | | | |
| Importance of bundle (energy) | >1,000 KJ | >500 KJ | >1,000 KJ | >1,500 KJ |
| Duration of harvest season | ≥4 months | ≤2 months | ≤2 months | ≤2 months |
| Weather | Yes | Yes | Yes | Yes |
| Weather lags 1–12 | Yes | Yes | Yes | Yes |
| Cell × Year FE | Yes | Yes | Yes | Yes |
| Month FE | Yes | Yes | Yes | Yes |

The analysis replicates the baseline results by extending the empirical specification to the consideration of harvesting months and their interactions with malaria-suitable months. Panel A restricts attention to the subset of Latent Epidemic cells. Panel B replicates the analysis for all cells for which harvesting data are available with "Malaria-Suitable Months$_{EA}$" is an indicator that takes a value 1 if the conditions in the given month in the given cell were suitable for malaria to be transmitted (monthly data) with differential weights that take the value 1 for latent epidemic areas (the same indicator used in the baseline analysis and lower weights that are gradually decreasing to 0 as the index approaches its maximum value. Harvest period is a binary indicator for the month being a harvest month for the respective crop. Weighted OLS estimates (linear probability model). The covariates and specification follow table 2, column 5 for the subsample for which data on harvesting months are available. Significant at ***1%, **5%, and *10%.

intertemporal labor substitution, we discriminate crop bundles in terms of the duration of the harvesting seasons.[33]

Table 4 presents the corresponding results.[34] We find no evidence for a differential effect of malaria shocks striking during comparatively long harvesting seasons (with a duration of at least four months; see column 1). The results in columns 2 to 4 show that the effect of malaria shocks on conflict is, however, largest during short harvesting seasons (two months or less) with an effect that tends to increase with the importance of the respective crop for subsistence (measured in terms of caloric yield).[35] These results are confirmed when considering weighted estimates on the full sample (tables A46–A47).

Combining the analysis for different conflict types in online appendix table A40 with the evidence on the role of harvesting months in table 4 shows that malaria shocks during short harvesting seasons are associated with increases in unorganized violence (riots and protests and for confrontations involving militias and civilians; see table A48). These additional analyses also confirm that the effect is monotonically decreasing the longer the harvest season and the lower the caloric importance of the crops.

*Malaria shocks during growing seasons.* In contrast, latent epidemic outbreaks of malaria during the growing season do not lead to increased incidence of civil violence (tables A49–A50).[36] The baseline findings are also confirmed when accounting for interactions between growing seasons and weather conditions (table A51) or when accounting for agriculture-relevant weather shocks during growing seasons (table A52). These exercises effectively replicate and confirm the analysis of Harari and Ferrara (2018) but at monthly frequencies and exploiting within cell×year variation, but leave the main effect of malaria shocks unaffected. In sum, the findings suggest that the documented effect of malaria shocks on conflict is elevated during harvesting seasons, particularly when harvesting seasons are short and relate to important bundles of crops. We find no such elevation in the effect during growing seasons. This is further evidence pointing to economic hardship related to shocks to labor supply as a potentially important mechanism for conflict outbreaks, complementing the price-related or weather-related income shocks that have been studied in the literature.

### D. *Attenuating Factors: Genetic Immunities and Health Interventions*

The last implication of the conceptual framework relates to the role of attenuating factors. Two main factors are expected to attenuate the susceptibility of the population conditional on the realization of suitable conditions for malaria outbreaks

[33]The frequency of malaria-suitable conditions is approximately balanced across harvesting and nonharvesting months; see figure A11 in the online appendix.

[34]The role of harvesting months in the table is studied by restricting attention to the subset of epidemic areas for which harvesting data are available. This specification avoids having to estimate additional main effects and triple interaction terms.

[35]For these more essential crops, the occurrence of harvest periods without malaria shocks tends to be associated with a reduction in the incidence of civil violence, which is consistent with the view that harvesting months are periods with high opportunity cost for violent activities.

[36]The growing season is constructed as a binary indicator variable that takes the value 1 if a grid cell exhibits temperature and moisture conditions that are suitable for crop growth in the specific month. The measure is also constructed based on satellite images, taken from http://harvestchoice.org/labs/measuring-growing-seasons. See the online appendix for details.

TABLE 5.—GENETIC IMMUNITIES AND ANTIMALARIAL POLICIES

| | Violent Events: ACLED Monthly Panel | | | |
|---|---|---|---|---|
| | Genetic Immunities | | Antimalarial Policies | |
| Moderating Factor: | Sickle Cell % (1) | Sickle Cell DV (2) | Average Coverage (3) | Maximum Coverage (4) |
| Malaria-suitable month | 0.009*** | 0.009*** | 0.016*** | 0.015*** |
| | (0.003) | (0.003) | (0.004) | (0.004) |
| MSM × Genetic Immunities | −0.072** | −0.006* | | |
| | (0.035) | (0.003) | | |
| Antimalarial Policies | | | −0.178*** | −0.054*** |
| | | | (0.030) | (0.016) |
| MSM × A. M. Policies | | | −0.096*** | −0.045*** |
| | | | (0.032) | (0.017) |
| Weather | Yes | Yes | Yes | Yes |
| Weather lags | Yes | Yes | Yes | Yes |
| Cell fixed effects | Yes | Yes | Yes | Yes |
| Month-year fixed effects | Yes | Yes | Yes | Yes |
| Observations | 172,260 | 172,260 | 131,976 | 131,976 |
| *R*-squared | 0.279 | 0.279 | 0.308 | 0.307 |
| Number of cells | 957 | 957 | 846 | 846 |

The results replicate the analysis of table 2, column 5 at monthly frequencies. "Genetic Immunities" is the average prevalence (% of the population) of the sickle cell trait in the cell, or measured by a time-invariant binary indicator relative to the sample mean. Policies are the coverage of artemisinin-based combination therapy, insecticide-treated bed net, and indoor residual spraying; they are measured by the average of the three coverage rates, or by the maximum coverage of any one policy at yearly frequencies. See text for details. For reasons of data availability, the analysis is restricted to cells in which antimalaria policy data are available. Significant at *** 1%, ** 5%, and * 10%.

in epidemic areas: genetic immunities and the extent of coverage with antimalaria policies.

*Genetic immunity.* To explore the potential role of genetic immunity as an attenuating factor of malaria shocks, we use information on the spatial distribution of the so-called sickle cell trait in the population. Like several other monogenetic diseases, an abnormal hemoglobin gene (HbS), the so-called sickle cell trait, provides highly effective protection against *Plasmodium falciparum* (Ferreira et al., 2011). Information about this trait is available for a relatively large subset of locations in Africa.[37] The results in columns 1 and 2 of table 5 show that a higher prevalence of genetic immunity tends to attenuate the effects of malaria risk on violence in epidemic areas. The patterns hold when discriminating between epidemic and endemic areas in the full sample (table A53). Further explorations also show that the effect of suitable conditions for malaria transmission is larger in cells with a low prevalence of genetic immunities and that the effect of the malaria shock occurring during harvesting months documented above is more pronounced in these cells (table A54).[38]

*Antimalaria policies.* To explore the role of antimalaria policies as a potentially attenuating factor, we use information about the coverage of antimalaria policies that is available for a subset of cells in Africa. In particular, the data contain information about the coverage in terms of artemisinin-based therapy, insecticide-treated bed nets, and indoor spraying and coating of walls and other surfaces with residual insecticides.[39] The results in columns 3 and 4 of table 5 reveal a negative effect of antimalaria policies (regardless of whether measured in terms of average or maximum coverage) on civil violence, as well as a negative interaction between *MSM* and policy coverage (see also table A56).

These results should be interpreted with caution, given the nature of the data on policies, which record actual coverage that could itself be affected by violence. Nevertheless, the patterns, and in particular the evidence about the interactions between time-varying exposure to malaria outbreaks and policy coverage, are suggestive of a potentially relevant but so far neglected indirect effect of health interventions in terms of reducing the negative effects of spikes in malaria risk on civil violence.[40]

*A vaccine thought experiment.* The potential role of health policies in this context can be illustrated by way of a simple thought experiment. Considering the estimation results of the baseline model with cell × year fixed effects, the hypothetical eradication of malaria, resulting from, for example, the introduction of an effective vaccine, would be associated with a reduction in the incidence of violent events by around

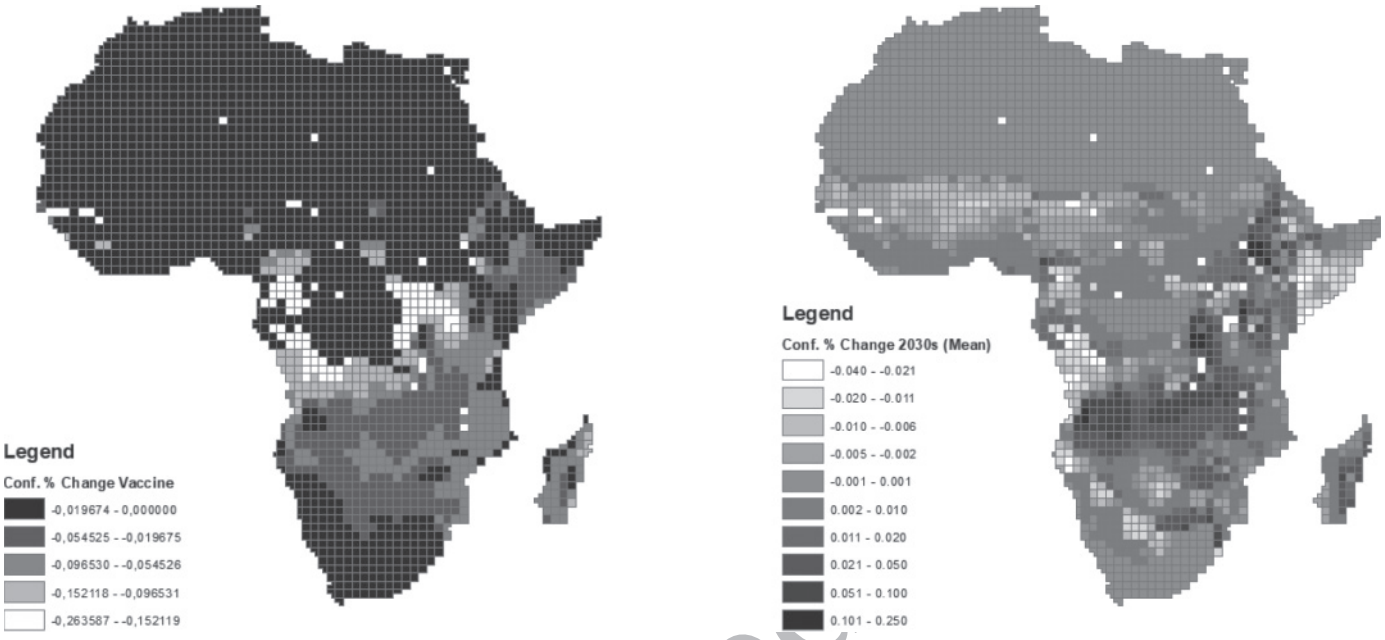
[37] See the appendix for a discussion of genetic immunities, data sources, and results, respectively. For reasons of data availability and comparability with the other results in the table, the analysis is confined to areas in which malaria is epidemic.

[38] Additional results document that the effect of malaria risk is largest in areas with the highest genetic and ethnic diversity (table A55). This suggests that epidemic-related stress may interact with latent ethnic tensions, providing novel evidence for the possibility that ethnic divisions work as amplifiers of short-term shocks (Esteban, Mayoral, & Ray, 2012).

[39] These data are only available for cells with frequent malaria exposure. See the online appendix for details on data sources and construction.

[40] The occurrence of a month with conditions of elevated transmission risk for cells below the 25th percentile of the distribution of antimalaria policies (where coverage is 0.0015) is essentially not mitigated by policies. In contrast, the occurrence of a malaria-suitable month in cells at the 75th percentile of the distribution (where coverage is 0.14) has essentially no effect on civil violence.

FIGURE 3.—COUNTERFACTUALS: IMMUNIZATION AND CLIMATE CHANGE




(a) Comparing Actual and Predicted (Counterfactual) Conflict Incidence

(b) Climate Change and the Projected Increase in Civil Violence

(a) The difference between the predicted incidence of violent events obtained under a counterfactual "Malaria-Vaccine" scenario that switches off the estimated effect of malaria-suitable months (fraction of years with at least one conflict over the period 1997 to 2012) and the actual incidence of violent events (fraction of years with at least one conflict over the period 1997 to 2012). (b) The predicted difference between projected incidence and observed incidence of violent events. The projection is based on a combination of the baseline estimates and the projected frequency of malaria suitable conditions based on projections of monthly averages of precipitation and temperature for the period 2020 to 2030. Climate projections are based on an average of difference scenarios of the Coupled Model Intercomparison Project Phase 5 (CIMP5) of the World Climate Research Programme.



14% (compared to a standard deviation of 27%). Figure 3a illustrates the spatial distribution of the benefits from this counterfactual exercise.[41] The results illustrate that eliminating malaria-related violence would affect particularly central Africa, the Great Lakes region, and eastern Democratic Republic of the Congo, but also some western parts of it, and the deltas of the rivers Congo and Niger. Also regions in eastern Kenya would benefit from the elimination of malaria-related violence, suggesting that effective treatment of malaria would have helped prevent a substantial fraction (10%–15%) of the observed violence in some of the cells that exhibited most violence.

### *E. Exacerbating Factors: Climate Change*

The results obtained so far imply the relevance of a largely overlooked link between geography and climate (reflected by the long-term conditions for malaria transmission stability) and weather shocks (that cause a temporarily elevated risk of malaria outbreaks) for socioeconomic distress and the risk of civil violence. The evidence on the peculiar interactions between weather-related malaria risk and the susceptibility of the population aligns with arguments made by the WHO (2016) on the fact that "a community that has not been exposed to malaria for a number of years will have little or no immunity to malaria; reinvasion can therefore result in sudden, explosive, catastrophic epidemics." Besides supporting warnings about the risks of relaxing the efforts devoted to antimalaria campaigns after the recent intensification, the results suggest that further research should be directed toward a better understanding of the potential role of climate change in the different areas at risk.

In fact, the results raise a warning regarding the potential implications of climate change for the spread of epidemic malaria to areas that so far exhibited no or very little exposure in light of indications that climate change could also lead to increasing weather variability and alter the frequency of occurrence of malaria-suitable conditions.[42] Combining our baseline estimates with projections of temperature and precipitation for the decade 2020 to 2030, it is possible to project the incidence of civil violence and compare it with the average incidence observed in the past. Figure 3b plots a map of the differences between so-projected incidence of civil violence during the next decade and the actual violence during the observation period of this study. The figure illustrates the cells for which the predicted increase is particularly pronounced. These cells partly coincide with the cells for which the reduction in violence that could be achieved by

[41] Formally, the incidence of violence is predicted for each cell and year using the baseline specification (corresponding to column 1 in table 2) while setting to 0 the effect of malaria-suitable months in cells in epidemic malaria areas. We then construct the difference between this predicted model and the actualy observed incidence of violence to get the average predicted reduction in incidence of violence (in terms of a share that lies in the interval between 0 and 1) over the full observation period for each cell.

[42] See IPCC (2012) and Siraj et al. (2014).

effective malaria prevention according to the results in figure 3a. This also includes, for instance, cells in eastern Kenya for which an increase in epidemic malaria as consequence of climate change has already been observed (see the discussion in section IIC).

## VI. Conclusion

This paper has contributed a first systematic investigation of the neglected role of epidemic shocks for civil violence. Whereas a wealth of arguments and narratives by development practitioners warned about the serious social, economic, and political consequences of these shocks, which put entire communities in distress, empirical evidence regarding the implications for the incidence of civil violence was missing. The analysis builds on the insights of a large literature in epidemiology that studied the drivers and dynamics of malaria outbreaks. The econometric identification exploits exogenous variation in weather conditions that are suitable for the outbreak of malaria in interaction with variation in the susceptibility of the adult population reflected by the stability of malaria transmission in a location. The analysis is based on a newly constructed data set for the entire African continent over the past two decades that features extraordinarily high spatial and temporal resolution. The results of this paper document that malaria outbreaks in epidemic areas lead to a significant increase in civil violence. A specific novelty of the analysis rests on the identification of the timing of the effects. The results document that the effect of malaria shocks on violence is on impact, concentrated in the month of the acute phase of the epidemic and spikes during sensitive production periods associated with short harvesting seasons of important crops. The results provide evidence for an attenuation of the effects in areas where the prevalence of genetic immunities in the population is higher or in areas that exhibit a higher coverage with antimalaria policies. The findings and the identification of the mechanisms consistently emerge in an extensive set of sensitivity and robustness checks.

The analysis offers several policy-relevant insights that are specific to these health shocks. In particular, the results document new evidence for so far overlooked side effects of coverage with antimalaria policies beyond the health domain. The identification of an effect of the occurrence of particular weather conditions in malaria-epidemic-prone areas on civil violence at a high spatial and temporal resolution provides insights that are relevant for prioritization of containment policies. While the development of a tool for directing measures of prevention of outbreaks of malaria and malaria-related conflicts is clearly beyond the scope of this paper, the empirical results are helpful for predicting latent spikes in violence. In this respect, the findings suggest that early warning systems that have been considered key for prevention of malaria outbreaks could also be useful to prevent spikes in civil violence. However, the results also indicate that it is crucial that policies are implemented within the first months of the epidemic. The more general message of this research is not restricted to malaria, as it implies that avoiding, or effectively curbing, epidemics may be valuable above and beyond health by also helping to curb social tensions. While in the context of malaria, our results suggest that early monitoring using weather data and the use of mosquito nets appears a promising and potentially important strategy for avoiding malaria-related civil violence, other interventions, such as various forms of quarantine or lockdown that might be effective in the context of epidemics related to diseases that are directly transmitted from human to human, appear useless. This does not limit the internal and external validity of our results. Malaria constitutes a disease that affects millions of people and creates recurrent threats for entire communities. We thus believe that the results are of general interest, even if the specific policy implications cannot be mechanically transferred to other types of epidemics. The results also suggest important interactions between epidemiological dynamics and agricultural cycles, with important insights for agricultural practices and for the organization of labor. Finally, the results point toward a nexus between climate change and civil violence that works through health and has been largely overlooked in the literature. Understanding the role of climate change and identifying the areas that will be affected the most appears to be an important avenue for future research.



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