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

February 5, 2019

Abstract

This paper performs the first systematic investigation of the effect of epidemic shocks for civil violence. The identification exploits exogenous within cell \times year variation in conditions that are suitable for malaria transmission using a panel database with month-by-month variation at a resolution of $1^\circ \times 1^\circ$ degrees 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 anti-malaria policies attenuate the effect. The results deliver insights for prevention and attenuation policies.

JEL-classification: D74, Q34, I1, J1

Keywords: Malaria Shocks; Epidemics, Civil Violence; Weather Shocks; Human Immunities; Productivity Shocks, Harvesting Months, Within Cell-level Variation, Africa.

1 Introduction

Civil violence is a major burden for the prospects of economic development, in particular in Africa. In recent years, the underlying reasons for outbreaks of civil violence have attracted considerable research efforts, and empirical work has shed light on the role of income shocks as important triggers of violence. The identification of these shocks has mainly relied on weather-related and commodity price-related income shocks. In contrast, very little is known about the role of health shocks in terms of epidemic outbreaks for triggering conflict. Such shocks represent existential threats that might lead to tensions and outbreaks of civil violence but have been neglected entirely in the existing literature. Understanding the role of epidemics for outbreaks of civil violence is of paramount importance from a positive and from a policy perspective, yet constitutes an open empirical question. In particular, the distinct nature of health shocks compared to income shocks related to variation in weather or commodity prices, and the peculiarities of their epidemiology, have crucial implications for the design, timing and implementation of appropriate prevention or accommodation policies that help avoiding outbreaks of civil violence.

This paper offers a first systematic empirical investigation of the role of epidemic shocks for civil violence. The analysis focuses on epidemic malaria outbreaks in Africa, which provide a particularly relevant case as they recurrently affect entire communities and are perceived as a major threat. Outbreaks are geographically and temporally confined, follow well-studied dynamics and generally last around three or four months. During this period they represent serious shocks affecting communities at large. Estimates suggest that between a third and a half of the local population, including adults, can be infected. Besides their direct implications for individual health, malaria outbreaks also represent considerable negative economic shocks for entire households, as a consequence of treatment costs and reduced labor productivity. The rapid increase in the number of cases and in the risk of infection within a short period of time, and the aggregate nature of these shocks, seriously limits the ability to implement mechanisms of inter-personal and inter-temporal labor substitution, particularly during sensitive production periods like short harvesting periods of subsistence crops. While the sudden distress of entire communities due to malaria has been associated repeatedly with an increasing incidence of civil violence, virtually no hard evidence on the effect of epidemic outbreaks on civil violence exists.

We use information on violence from the Armed Conflict Location and Event Data Project, ACLED at level of $1^\circ \times 1^\circ$ degrees grid cells for the last two decades in Africa. We commence the analysis by documenting that spikes in malaria infections are related to significant increases in the

incidence of civil violence. While suggestive, this correlation can be driven by violence-related factors such as population dynamics and reduced health coverage caused by conflicts. Furthermore, proxies of clinical malaria cases are constructed to be informative on medium-term trends in infections and are only available at yearly frequency. This prevents an analysis of the short-term dynamics and, accordingly, the causal identification of the effect of malaria epidemics on outbreaks of civil violence and of the respective mechanisms.

To face these challenges, we devise an empirical strategy to identify the impact of malaria epidemics on outbreaks of violence. The identification strategy builds closely on the well-studied epidemiological specificities of malaria outbreaks and allows us to isolate this impact from that of other shocks by exploring the mechanisms and their implications for the timing of outbreaks. In particular, the identification exploits the interaction between exogenous changes in the conditions for malaria transmission (which are mainly related to the occurrence of particular weather conditions) and different levels of susceptibility of the population to malaria outbreaks (as reflected by the prevalence of immunity among adults). This identification strategy thereby combines the central elements of malaria epidemics, since, according to MacDonald (1957) “an epidemic is an acute exacerbation of disease out of proportion to the normal to which the community is subject. [...] Epidemics are common only in zones of unstable malaria, where very slight modifications in any of the transmission factors may completely upset equilibrium, and where the restraining influence of immunity may be negligible or absent” (p. 45).

The identification makes use of monthly variation in the 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. In particular, identification relies on exogenous variation in weather conditions at a high level of temporal and spatial disaggregation, in terms of monthly data at the level of $1^\circ \times 1^\circ$ degree grid cells. The use of such highly disaggregated panel data in combination with exogenous variation in weather conditions at monthly frequencies allows us to identify the effect of malaria shocks and its timing within the year, and thereby to isolate the effect from that of other shocks, as well as the existence of attenuating factors. In particular, the analysis exploits month-by-month variation within cells, while conditioning on cell \times year and calendar month fixed effects and thereby implicitly accounting for seasonal effects and for other previously studied factors that have been shown to affect violence in a given grid cell using yearly variation, including weather-related and commodity price-related income shocks.

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

are characterized by a low resilience in terms of acquired immunities (and thus a high susceptibility) of the adult population. The baseline findings are shown to be robust to a very extensive set of sensitivity and falsification checks. Given the nature of the data, we devote particular attention to assess the sensitivity of the results with respect to the measurement of malaria suitable months and of the susceptibility of the population, with respect to short-term fluctuations of weather conditions per se, and with respect to location-specific confounding factors.

Following the conceptual considerations regarding the link between malaria epidemics and violence, we design several empirical strategies to study the mechanisms behind the baseline patterns. We find that an increase in the risk of epidemic malaria leads to an increase in unorganized violence, but not in violence driven by geo-political and military motives. A unique feature of the monthly panel data is that it allows studying the precise timing of the response to shocks and to perform falsification tests. The results show that violence is triggered upon impact and mostly during the acute phase of the epidemic, which is associated with the largest risk of infection. The likelihood of epidemic-related violence is highest during harvesting months of important crops, but only in epidemic areas and for crops that exhibit short harvesting seasons. In contrast, no differential effects of malaria outbreaks can be detected during growing season months. We find evidence of an attenuation of the effect of malaria shocks on violence in the presence of a higher prevalence of genetic immunities to malaria in the population. The results also show that the effect is attenuated by a more extensive coverage with anti-malaria policies. The quantitative implications of the results are illustrated by ways of a counterfactual simulation of the consequences of the introduction of a malaria vaccine. The results suggest that the eradication of malaria could bring sizable reductions in civil violence in epidemic areas.

The paper is organized as follows. Section 2 locates the contribution of this paper in the existing literature and discusses the policy implications of the findings. Section 3 presents the basics of malaria epidemiology, the conceptual framework and discusses anecdotal evidence. The data and empirical strategy are described in Section 4. Section 5 presents the baseline results. Section 6 explores the mechanisms. Section 7 concludes.

2 Contribution to the Literature and Policy Implications

The last decades have witnessed a large effort dedicated to the understanding of the determinants of civil violence. The literature has initially restricted attention to large scale civil conflicts and wars

at the country level and has identified several factors that increase the risk of civil wars.¹ The role of negative income shocks has been studied by exploiting two main sources of exogenous variation within countries over time, weather conditions like droughts that affect income production in rain-fed agriculture, see, e.g., Miguel, Satyanath, and Sergenti (2004), Ciccone (2011), Couttenier and Soubeyran (2014) and Berman and Couttenier (2015), and variation in international commodity prices, see, e.g., Bazzi and Blattman (2014), Caselli and Tesei (2016). While the literature has explored the economic implications of epidemics, to our knowledge, the only attempt to link the role of diseases to civil wars at the country level has been conducted by Cervellati, Sunde, and Valmori (2017).

The increasing availability of disaggregated panel data on civil violence has shifted attention to the investigation of the drivers of civil violence at the sub-national level using geo-localized information for Africa at yearly frequency. Compared to cross-country studies, the analysis at the disaggregated level faces limitations in terms of data availability, particularly for socio-economic covariates. On the other hand, disaggregated data provide an expanded set of strategies for identification of the drivers of local violence and the related mechanisms. Disaggregated violence data have initially been linked to time-invariant characteristics by exploiting variation across grid cells.²

This paper contributes more directly to the recent literature on the impact of short-term shocks of conflict that uses pixel-level panel data for Africa. Several contributions identify the effects of exogenous variation in (international) commodity prices. Berman and Couttenier (2015) and Berman, Couttenier and Soubeyran (2017) study the role of commodity price changes that act as threat multipliers affecting the value of production and the cost of production, respectively. McGuirk and Burke (2017) study the differential effect on violence of variation in agricultural prices that primarily hit producers or consumers. Berman, Couttenier, Rohner and Thoenig (2017) study the role of increasing prices of minerals and the control of mining areas for the scale-up in violence by rebel groups. In terms of source of exogenous variation our paper is conceptually most closely related to Harari and La Ferrara (2018) who isolate the role of unfavorable weather conditions for negative production shocks in rain-fed agriculture during growing seasons at the cell-year level.³

¹For a survey, see, e.g., Blattman and Miguel (2010).

²The work by Besley and Reynal-Querol (2014) documents the persistent role of historical conflicts on violence today. Michalopoulos and Papaioannou (2016) identify the legacy of the colonial scramble for Africa for ethnic rooted political violence. Cervellati et al. (2018) look at the effect of long-term exposure to malaria across grid cells. Their finding that cells with intermediate malaria stability are more violent can be interpreted as further background motivation for the study of the malaria shocks performed in this paper.

³In a broader perspective, the findings also complement the evidence for the role of weather fluctuations on economic performance by Dell, Jones, and Olken (2012) by documenting a further channel through which weather shocks can induce negative income shocks.

This paper contributes in several ways to this existing literature. First, the occurrence of local malaria shocks is conceptually orthogonal to income shocks related to weather during growing seasons or to variation in international commodity prices. Income and production shocks related to weather fluctuations during growing seasons have been documented to unfold their effects with a delay over several months and tend to affect violence from one year to the other. In contrast, epidemic shocks impose sudden, uninsurable stress on the affected communities and display an effect on unorganized civil violence, but not on violence related to geo-strategic or military conflicts. Differently from existing studies, the analysis below exploits panel data at monthly frequency and thereby disentangles the role of weather-driven epidemic shocks from other short-term shocks. In particular, conditioning on cell \times year (and calendar month) fixed effects allows isolating the role of spikes in malaria risk at monthly frequency, while accounting for factors that affect violence in a specific location and year, as the income shocks shown in earlier studies. In this respect, our results complement the existing literature by implicitly quantifying the overall role of the latent drivers of violence across years above and beyond short-term spikes in malaria risk. This is documented by the considerable increase in the explanatory power of the empirical specification when including cell \times year effects. At the same time, the main findings are essentially unaffected, which suggests that short-term variation in health-related to malaria risk tends to be orthogonal to observable or unobservable cell-year specific drivers of violence. The results are also robust to the consideration of spatial spillovers over time in both violence and malaria risk, which is a demanding exercise that has not been performed in the existing literature.

Second, while the timing of shocks and their effects are generally difficult to predict in the case of weather-related and price-related income shocks, the epidemiological dynamics allow us to isolate the dynamics of the effects and thereby disentangle the underlying mechanism from alternative drivers of conflict. In particular, we can track the response of localized violence to the impulse of malaria shocks within a year, explore the persistence of the effect, and analyze the lag and lead structure. The results suggest that not all months within a sequence of malaria suitable months are equally likely to lead to civil violence. The effect is detected mainly during the acute phases of epidemic outbreaks rather than at the onset of during the normalization phases.

Third, a large literature in development economics as well as a substantial body of anecdotal evidence from development practitioners points at conditions that are likely to amplify or attenuate the effects of epidemic shocks on violence. Malaria-induced income shocks are primarily related to shocks to labor productivity, which is considered a main source of income variation in developing countries. However, the role of shocks to labor productivity has not been explored in the context of

civil violence before. The evidence for the existence of a link between suitable conditions for malaria transmission and short harvesting seasons of important crops, but not with the growing season, provides reduced form evidence that the income effects of health shocks are mainly linked to labor productivity.

Fourth, the distinction between epidemic shocks on the one hand, and weather-related or commodity price-related income is important in view of the possibility of prevention and attenuation. Epidemic shocks require specific responses in terms of policies that differ from those applicable in the context of other shocks. In this respect, the results provide several new insights based on data for the entire continent of Africa. The findings document that the effect on violence takes place on impact and mostly during the acute phases of epidemic outbreaks. This supports the arguments often put forward by international organizations and development practitioners that an early detection of infections and a timely intervention are crucial for the prevention of the social consequences of epidemics. The evidence shows that a differential susceptibility in terms of the prevalence of genetic immunity in the population affects the impact of health shocks on violence. While policies cannot alter these innate immunities in the short-run, the findings also suggest that better coverage of anti-malaria policies can represent an effective substitute for containment and attenuation. Moreover, the results indicate that practices that have been implemented traditionally in endemic areas to limit the risk of harvest failures, such as avoiding labor-intensive crops with short harvesting periods that coincide with periods of high risk of malaria outbreaks, could be a useful strategy for epidemic areas, complementing suggestions by Arrow et al., (2004, Ch. 7). Given that the evidence is based on the use of algorithms developed by malaria epidemiologists for the purpose of developing early detection systems of immanent malaria outbreaks, the findings deliver relevant insights for early interventions by providing detailed information about regions and periods that exhibit particularly high risk of epidemic-driven civil violence.

3 Conceptual Background

3.1 Epidemic Malaria in Africa

Malaria comes in different variants but 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 since more than 10,000 years and is

still a major source of morbidity and death in the continent. Exact figures on clinical cases are not available, but recent estimates of the death toll range from 400,000 to above a 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. In the following, 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 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 then 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 Available recent estimates suggest that the share of adults developing the disease upon inoculation of the pathogen is below 10 percent in endemic areas but 50 percent or above in epidemic areas, see Griffin et. al. (2013).

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

⁴Malaria has killed more people than any other disease, which has led epidemiological historian Webb (2009) to label malaria “Humanity’s Burden”.

⁵*Center 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 only involve mild symptoms or are even asymptomatic, see the Supplementary Appendix for details and Figure A2 for a graphical illustration of the effect of acquired immunities, that explains the relationship severity of infection and age in endemic areas. This pattern of the share of adults developing infections upon inoculation being decreasing with the level of malaria transmission stability has also become known as the “age peak-shift” phenomenon (MacDonald, 1957).

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 both in endemic and epidemic areas but, as discussed above, 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 3 or 4 months and follow typical dynamics. The infection of few persons 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, even though many individuals are still sick or in the process of recovery, see also WHO (2016).

3.2 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 stated that malaria epidemics can lead to outbreaks of violence as consequence of the sudden economic and social disruption they engender on immunologically naive and socio-economically unprepared populations. For instance, the 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” and “a malaria epidemic may have disastrous consequences: disrupt the social, political and economic activity in a community [...] with severe political consequences”.⁶ The warnings also align with survey evidence according to which illness (with malaria being the most frequently reported disease) is a main source of subjectively perceived stress in African communities.⁷

⁶See WHO at (<http://www.who.int/malaria/publications/atoz/epidemicsmerajuly2004.pdf>). 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>).

⁷See Survey responses to CIFOR’s Poverty and Environment Network (PEN) global dataset collecting data on the subjectively perceived importance of environmental conditions in rural communities in Sub-Saharan Africa. Figure A1 in the Supplementary Appendix reports the shocks of different types. Illness (and death) and crop failure alone account

From an individual perspective, the effect of an epidemic malaria outbreak on the likelihood of violent behavior is a priori ambiguous. On the one hand, 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 of stress for civil violence is not straightforward to evaluate but cannot be ruled out a priori in the context of malaria, and aligns with narratives from health practitioners.⁸

The most relevant implications of malaria outbreaks for civil violence unfold on the community level, however. During an outbreak, a sizable share of the community members is suddenly exposed to sickness or a large risk of sickness. As a result, also healthy individuals are 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 shocks to income production as a consequence of reduced labor supply. Although not straightforward to quantify, the consequences are considered substantial, particularly in terms of lost labor productivity. Estimates of lost workdays per year range between 20 and 60 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”, report of the Food Policy Research Institute (Asenso et. al., 2010, p.7).¹⁰ 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 inter-personal substitution (e.g., mutual help with the harvest) and of inter-temporal labor substitution (e.g., a delayed harvest) is limited or just not possible. These broader consequences have been noticed in several reports. 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 et al., 2004, Ch. 7). Hence, a “brief period of illness [...] that coincides with the harvest may result in catastrophic effects” (UN Millenium Project, 2005).

Finally, the intensity of epidemic shocks (and their health and economic consequences) has been linked to the failure of an effective provision with health policies and insurance. Coverage with health

for about 70 percent of reported shocks. Asset loss accounts for a further 20 percent. See also the Parima-Study, Doss et al. (2008) and McPeak et al. (2012).

⁸The threat of infections has been documented to increase the release of “fight-or-flight” stress hormones such as cortisol, which can affect risk-taking. See also the Appendix in Section 1.

⁹See, e.g., Arrow et al. (2004, Ch. 7).

¹⁰A large literature has tried to quantify the economic costs of malaria. Most research concentrated attention to quantification of costs of health interventions and direct costs for treatment. The quantification of indirect costs of malaria in terms of loss of income production is more difficult to achieve. Estimates vary in magnitude 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, e.g., Chima et al. (2003) for a survey and the final report of the committee on the economics of antimalarial drugs, Arrow et al. (2004, Ch. 7).

insurance is generally limited in many developing countries, see e.g. Gertler and Gruber (2002). The fact that during epidemic outbreaks a large share of adults are infected or at risk of infection during a short period of time 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 the conceptual framework and that 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 anti-malaria health policies.

3.3 An Illustrative Case Study

Kenya is a useful case to illustrate the conceptual framework that underlies the empirical analysis that follows. 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 (lakes and sea) feature a stable transmission environment of the pathogen and the population is persistently exposed to infections. Malaria is endemic and mostly affects children. In the highlands, malaria is traditionally absent, but unusually suitable short term conditions can spark the pathogen's transmission cycle. According to the WHO Roll Back Malaria 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

The 2002 outbreak in the semi-highland areas at the border of the Nyanza Province in Kenya,

¹¹This 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 since, e.g., food reserves or 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.

¹²See www.who.int/malaria/publications/atoz/epidemics_tg.pdf. Similarly, Chuma et al. (2010) report that fever episodes among adults and children (over five years) 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).

a mixed agricultural area with low malaria transmission stability, followed the typical patterns of a malaria epidemic. Unusually humid and warm weather during spring increased malaria risk by more than 30 percent relative to the average for other years in the area. These conditions ultimately materialized in an epidemic outbreak in June and July that led to about 160,000 clinical cases (about 40 percent were adults) and 400 deaths within the first few weeks. Epidemiologists had issued warnings about the high risk of outbreaks and subsequently reiterated on the usefulness of forecasts based on weather conditions, see Hay et al. (2003) for a case study of the event. The epidemic coincided with the short harvesting season for several important crops in this region.¹³ As a consequence of the epidemic during this sensitive period, entire communities suddenly got under stress. This led to confrontations between groups of civilians, raids, riots and protests. The Health Permanent Secretary stated on the news that interventions were timely planned following early forecast in March but criticisms about delays in the implementation of the measures further sparked social tensions and violence. According to the ACLED database that is used for the empirical analysis below, civil violence spiked and episodes of violence eventually increased by more than 50 percent compared to the same period in other years in this region. The epidemic was over in August, when suitable conditions for transmission ceased, and violence also abated.

4 Data and Empirical Approach

4.1 Data

4.1.1 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 cover different types of events, including riots and protests, violence against civilians, and the type of actors involved (e.g., militaries, militias, civilians, among others). The estimation analysis is conducted for the period 1998-2012.¹⁴ The baseline measure of civil violence is a binary indicator variable taking value one if at least one event of any type occurred in a given cell in a given period (year or month).¹⁵ Several alternative measures of violence are considered to explore the channel and as robustness checks.

¹³Information from FAO crop Calendar (www.fao.org/agriculture/seed/cropcalendar/welcome.do).

¹⁴The 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 Supplementary Appendix.

¹⁵Alternative data from the UCDP Georeferenced Event Dataset are only available at annual frequency and therefore not suited for our main analysis at monthly frequency.

4.1.2 Epidemic Malaria Areas

Epidemiologists typically classify areas along a stable-unstable transmission gradient for malaria (see MacDonald, 1957). In areas with low to intermediate stability of malaria transmission, adults are exposed to a higher latent risk of malaria than in areas with no or high stability for the lack of acquired immunities (see Sections 3.1 above and 1 in the Appendix). This implies the highest latent risk of malaria epidemics in these areas. To operationalize this information, we use data from the Malaria Stability Index by Kiszewski et al. (2004), which is an ecology-based spatial index of the stability and force of malaria transmission.¹⁶ 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 baseline, we construct a binary indicator for latent epidemic areas, EA , which takes value 1 for cells with a Malaria Stability Index strictly larger than 0 and lower or equal to 15. This baseline coding aligns with the evidence that levels of the malaria stability index above 15-20 are typically characterized by endemic malaria. As discussed further below this coding essentially implies assuming that areas at risk of epidemic malaria for adults are characterized by a sufficiently low, but positive index of malaria stability. Several sensitivity checks regarding the measurement of latent epidemic areas are reported below.¹⁷

4.1.3 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 dataset.¹⁸ We also use information about the Standardized Precipitation and Evapotranspiration Index (SPEI).¹⁹

The transmission of the *plasmodium* pathogen depends on short term weather conditions. The cycle of reproduction of both parasites and transmission vectors requires, roughly speaking, a suffi-

¹⁶Transmission of the pathogen between humans and vectors depends on local bio-climatological conditions. See Section 1 in the Supplementary Appendix for more detailed discussions of the short-term conditions for malaria transmission and the construction of the index.

¹⁷The construction of the measure of malaria stability is discussed in more detail in Section 2 of the Supplementary Appendix. There we also present a validation using data for projected malaria infections and alternative semi-parametric empirical strategies that identify latent epidemic areas more flexibly. Figure A4 in the Supplementary Appendix shows that areas with malaria stability above 15-20 are characterized by high endemicity in the African population around year 1900.

¹⁸The ERA-Interim data set has the advantage of being based on data re-analysis 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 Appendix for details.

¹⁹The SPEI index is a combination of the Palmer Drought Severity Index (PDSI) which is based on supply and demand for water, and of the Standardized Precipitation Index (SPI); the SPEI measures drought severity, intensity and duration, but also allows for comparisons of drought severity through space and time, including different drought types, see Vicente-Serrano et al. (2010).

ciently 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 et al. (2003) for the purpose of an early warning indicator of immanent malaria outbreaks.²⁰ We code a variable labeled Malaria Suitable Month (*MSM*) as a binary indicator of whether malaria transmission is possible or not. The variable takes value 1 if conditions that are suitable for transmission of the pathogen are met in a given cell and month, and 0 otherwise. The index is constructed at monthly frequency based on the local weather conditions in a given grid cell during the proceeding twelve months. In particular, the indicator takes value one if all of the following conditions are satisfied:

1. the average monthly rainfall during the past 3 months exceeds a threshold of 60mm/m²;
2. rainfall exceed 80mm/m² in at least one of last three months;
3. no month in the past 12 months has an average temperature below 5°C;
4. average temperature in the past three months exceeds 19.5°C plus the standard deviation of monthly temperatures over the past 12 months.

Tanser et al. (2003) document that the index has a 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.

4.1.4 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 are interpolated across space to obtain a map of malaria incidence.²¹

²⁰The same index has been used by Kudamatsu and Persson, and Strömberg (2016) to study the effect of malaria shocks for child mortality at yearly frequencies. This index was constructed following the 20th Report of the WHO Expert Committee on Malaria, which called for the need of reliable indicators based on highly disaggregated meteorological information to be used for the prevention of malaria epidemics. The report states 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).

²¹Figure D1 in the Supplementary Appendix illustrates the cross-sectional variability of (average) projected malaria incidence across the respective grid cells in Africa for the year 2000, which is the first year for which the data are

4.1.5 Other Variables, Sources and Summary Statistics

The analysis also makes use of a rich set of time-invariant and time-varying variables as controls or for additional exercises, which are discussed along the way when needed. For reasons of space, further details on about the construction, coding and data sources for all variables used in the analysis is provided in the Supplementary Appendix in Section 2. A summary description of the main variables used in the analysis is reported in Tables D1, D2, and D3 in the Appendix. Summary statistics are reported in Tables S1 and S2 in the Appendix.

4.2 Malaria Infections and Violence

A natural starting point for an analysis of the effect of malaria shocks on violence is considering the relation between the incidence of malaria in terms of clinical cases and the incidence of 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 when controlling for lagged incidence of violence.

These findings should be interpreted as mostly suggestive, however. For the purpose of the investigation of the causal role of malaria for violence, 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 does not allow distinguishing the effect of endemic and epidemic infections. Most importantly, the malaria incidence data have been assembled with the goal of mapping the evolution of malaria incidence in the medium run. 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 socio-economic covariates. The projected clinical incidence for adults has been obtained on this basis of cases. Finally, the information about projected incidence is available only at a yearly frequency. This implies that measures of projected malaria incidence are not suited for a causal identification

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

Dep. Variable	Violent Events - ACLED Yearly Data			
	(1)	(2)	(3)	(4)
Clinical Infections of Malaria	0.189*** (0.046)	0.184*** (0.046)	0.183*** (0.046)	0.167*** (0.043)
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	0.702	0.702	0.705	0.706
Number of Cells	1,681	1,681	1,681	1,681

The dependent variable is a binary indicator variable taking value 1 if at least one conflict event (ACLED dataset) was observed in the given cell in the given year. “Clinical Incidence of Malaria” is a projection of clinical incidence of *Plasmodium falciparum* malaria (per 1000) 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 socio-demographic covariates, see Section 2.1 in the 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 x 1 degree cell. Panel data from 1998 to 2012 at yearly frequency. ***, **, * indicate significance at 1-, 5-, and 10-% level, respectively.

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 3.2.²² 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.

4.3 Identification Strategy

To make progress towards identifying the causal effect of malaria epidemics on civil violence, this section develops an identification strategy 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

²²Survey data are not even 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 of a causal identification, because the possibility to rule 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 Supplementary Appendix for a detailed description of data construction and quality.

exposure to malaria outbreaks, reflected in monthly within-grid-cell variation in weather conditions that are particularly suitable for malaria transmission, malaria-suitable months MSM . We estimate the linear probability model:

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

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 for which conflicts are observed. In the monthly panel data, the variable MSM is an indicator that takes value one if in a given month in a given cell the conditions suitable conditions for malaria discussed above are satisfied, and 0 otherwise. The coefficient of interest is β , which captures the effect of the suitable short term conditions for malaria in i in country c in period t in cells with latent epidemic malaria risk (as compared to areas with latent endemic malaria).

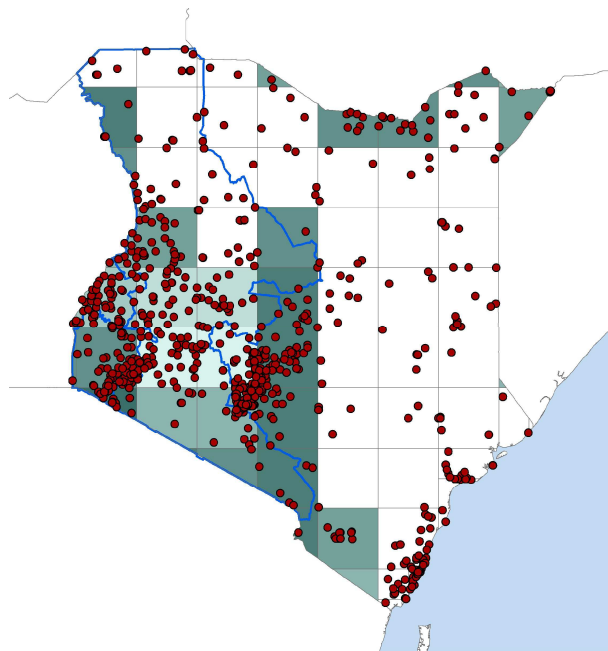
Reverse causality is ruled out by construction since short-run weather conditions in high and low malaria risk areas are exogenous to civil violence. The vectors X and Z contain additional contemporaneous or lagged covariates. Depending on the particular specification and the panel data frequency, the 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 \times year and calendar month fixed effects. The monthly panel data also allows us to study the within-year dynamics of malaria risk and violence and counterfactual exercises and placebo tests (using, e.g., 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, and spatial correlations with neighboring cells (Bester et al., 2011). For robustness we also explore the existence

of dynamic spatial spill-overs, we estimate both Spatial Autoregressive (SAR) and Spatial Durbin (SDM) models estimated by maximum likelihood.

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 semi-desert 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 2002 epidemic mentioned in Section 3.3 above 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 *vs.* non-epidemic malaria areas (*EA*) (in terms of the differential effects across shaded and white cells).

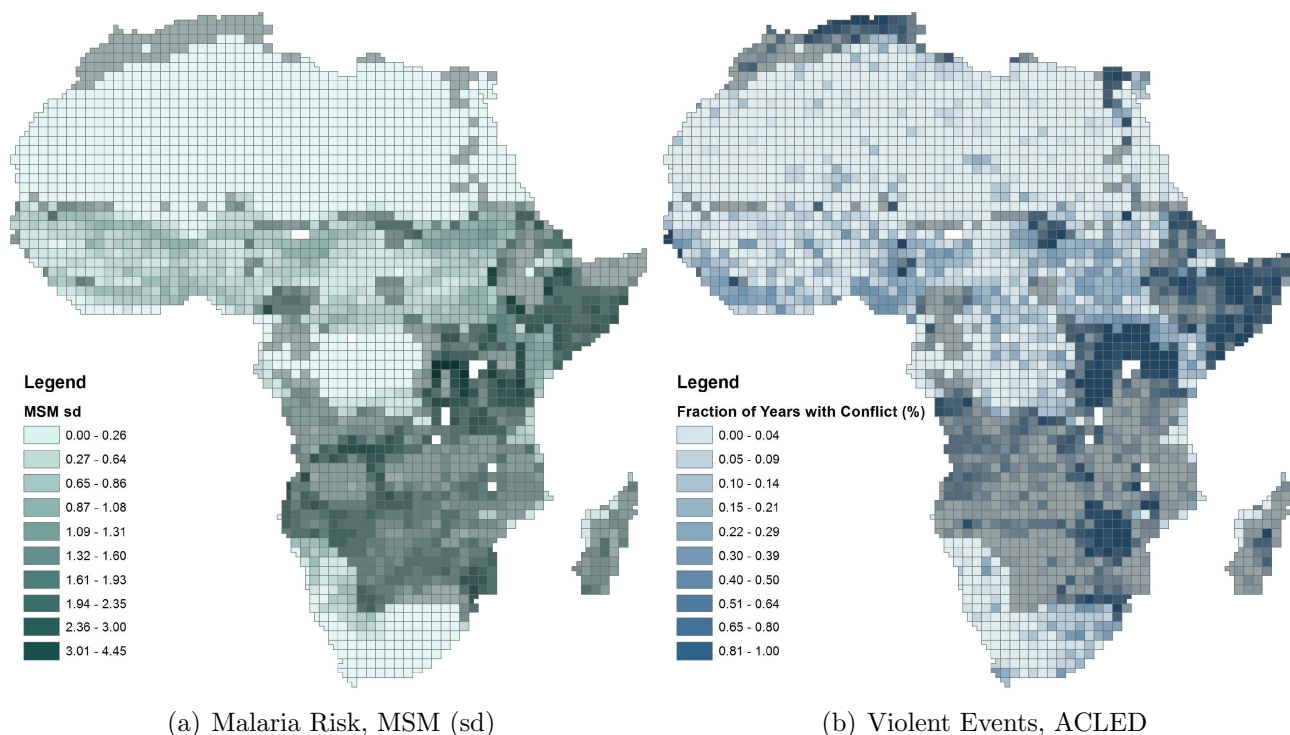
FIGURE 1: MALARIA RISK AND VIOLENT EVENTS IN KENYA



Note: 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).

To maximize external validity, the empirical analysis is performed for the entire African continent. Figure 2(a) 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. The latent epidemic areas with high susceptibility of adults for malaria infections under suitable conditions ($EA = 1$) are depicted with a light shadow.²³ Figure 2(b) 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.

FIGURE 2: MALARIA RISK AND VIOLENCE: AFRICA



Note: Panel (a) depicts 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, ($EA = 0$). Panel (b) depicts 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, ($EA = 0$).

²³The 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 Appendix.

5 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 robustness exercises that have been performed to evaluate their potential role in driving the results.

5.1 Estimation Results

The baseline results are reported in Table 2.²⁴ Columns (1)-(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 areas in a two-way fixed effects specification.²⁵ 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 (i.e., in non-epidemic areas). However, the occurrence of an additional month with suitable conditions for malaria increases the risk of violence by 1.3-1.5 percentage points in latent epidemic areas, which corresponds to 7-8 percent compared to the unconditional probability of 18.6 percent. The estimated model accounts for about 50 percent 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 3.2. 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 \times year fixed effects and thereby for all time-varying factors that affect average level of violence in a given cell across years (above and beyond monthly changes in malaria risk). These include, for instance, factors like the effect of income shocks triggered by bad weather during growing seasons, or fluctuations in commodity prices whose effect on civil violence have been identified at the cell-year level. In other words, the

²⁴The coefficients of the indicator *EA* is not identified in specifications with cell fixed effects since it is time-invariant by construction.

²⁵For space reasons we report the specification that directly looks at the differential effect of malaria risk in epidemic areas, conditional on weather conditions and their lags. Alternative specifications are reported in the Supplementary Appendix as discussed below.

TABLE 2: MALARIA RISK AND VIOLENCE: BASELINE RESULTS

Dependent Variable	Violent Events, ACLED					
Panel Data Frequencies:	Yearly			Monthly		
	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Suitable Month(s)	-0.008 (0.004) [0.005]	-0.005 (0.005) [0.005]	-0.005 (0.004) [0.005]	-0.003 (0.002) [0.003]	-0.002 (0.002) [0.002]	-0.002 (0.002) [0.002]
Mal. Suit. Month(s)×Epidemic Area	0.015** (0.005) [0.007]	0.013** (0.005) [0.006]	0.013** (0.005) [0.006]	0.008*** (0.003) [0.003]	0.007*** (0.003) [0.003]	0.007*** (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
<u>Fixed Effects:</u>						
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	38,340	38,340	38,340	457,560	457,560	457,560
R-squared	0.467	0.521	0.523	0.242	0.413	0.413
Number of Cells	2,556	2,556	2,556	2,556	2,556	2,556

The dependent variable is a binary indicator variable taking value 1 if at least one conflict event (ACLED dataset) 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 characterised 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 12 lags in the monthly panel. Panel data from 1998 to 2012 at yearly and monthly frequencies. The unit of observation is a 1 x 1 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 the threshold of 400 km are reported in square brackets, [·]. ***, **, * indicate significance at 1-, 5-, and 10-% level computed using the largest standard errors (cell clusters or Conley) of each specification.

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)-(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.²⁶

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 percent, the coefficient estimates of 0.007-0.008 in Columns (4)-(6) imply a sizable increase in the risk of violence of 16-18 percent 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. Second, the diff-in-diff specification at monthly frequencies of Column (4) explains about 20 percent of the variation in the data, while accounting for cell \times year specific effects almost doubles the share of variation explained by the empirical model. This suggests that cell \times year specific shocks like those emphasized in the existing 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.

5.2 Robustness and Confounders

To explore the robustness of the baseline findings, we conducted a large set of additional analyses. In the following, we provide a brief summary of sensitivity checks, potential confounders and alternative codings of epidemic areas and weather shocks. The corresponding details are reported in the Supplementary Appendix.

5.2.1 Empirical Specification: Robustness and Sensitivity

Accounting for spatial spill-overs. In order to explore the potential role of spatial dynamics for the main result, we estimated extensions of the baseline framework that explicitly account for dynamic spill-overs across space following the approach by Bester et al. (2011). In particular, we estimated spatial autoregressive models (SAR) that explicitly account for spatial contiguities in the diffusion of violent events across space; alternatively, we estimated a more demanding spatial Durbin

²⁶Note 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. See also Table A7 in the Appendix for results for extended dynamic panel specifications.

model (SDM) to further check for spatial dependencies in the occurrence of suitable conditions for malaria transmission or in weather shocks.²⁷ The results obtained for these models, which account for spatial autocorrelation in the dependent and independent variables, respectively, provide evidence for spatial-temporal spill-overs, particularly in terms of lagged violence in neighboring cells, but at the same time confirm the baseline findings (see Tables A1 and A2). Since the results confirm the baseline patterns, we continue the analysis by accounting for spatial clustering as in the baseline specifications.²⁸

Econometric specification. The results consistently emerge with alternative econometric specifications, including pooled OLS specifications, alternative specifications at yearly and monthly frequencies, unconditional regressions without weather controls, alternative dynamic panels at monthly frequencies and non-linear estimators (see Tables A3, A4, A5, A6, A7 and A8).²⁹

Alternative coding of violent events. Similar patterns as for the baseline specification consistently emerge when excluding episodes of violence with more uncertain location and of neuralgic conflict regions like Rwanda and Burundi and North Africa (Tables A9, A10); the incidence of suitable conditions for malaria transmission in epidemic areas has a positive effect on the onset and, to

²⁷In particular, we estimated a spatial autoregressive model, SAR, of the form

$$Violence_{i,c,t} = \rho \sum_j w_{ij} Violence_{j,c,t} + \alpha MSM_{i,c,t} + \beta EA_{i,c} \times MSM_{i,c,t} + \Gamma X'_{i,c,t} + \Delta Z'_{i,c,t-1} + \Phi_{i,c,t} + u_{i,c,t}$$

where w_{ij} represents the elements of the i th row of a (row-normalized) spatial contiguity matrix W_i that contains information on the cells j with a direct common border to the cell under consideration, i . Diagonal elements equal to zero. The model is estimated using maximum likelihood. See, e.g., Elhorst (2009) for details on spatial models and their estimation. Alternatively, we estimated a Spatial Durbin Model, SDM, of the form

$$Violence_{i,c,t} = \rho \sum_j w_{ij} Violence_{j,c,t} + \alpha MSM_{i,c,t} + \beta EA_{i,c} \times MSM_{i,c,t} + \zeta \sum_j w_{ij} MSM_{j,c,t} + \xi \sum_j w_{ij} EA_{j,c} \times MSM_{j,c,t} + \Gamma X'_{i,c,t} + \Delta Z'_{i,c,t-1} + \Phi_{i,c,t} + u_{i,c,t}$$

by maximum likelihood, where w_{ij} represents the elements of the i th row of (row-normalized) spatial contiguity matrix W_i that contains information on the cells with a direct common border to the cell under consideration, i .

²⁸The baseline results are reported in Table A1. The estimates at monthly frequencies with two-way fixed effects for the entire continent of Africa involve dealing with almost half a million observations for 150 time series observations in 2600 cells connected by a contiguity matrix. The specifications allow for two lags over time and in space in both violent events, malaria risk and the weather co-variates. These spatial models are extremely demanding specifications, both in terms of the residual variation left in the data for identification, and in terms of computational effort. We are not aware of previous attempts to estimate spatial models exploiting within-cell variation at monthly frequencies for the whole of Africa. We also estimated the models using alternative specifications involving more flexible weighting of latent epidemic areas, see Table A2.

²⁹The pooled OLS specifications allow estimating the coefficient for latent epidemic malaria areas (EA), which is robustly positive, even when including a large set of covariates. In terms of quantitative importance, the effects are very similar to those obtained with within-cell estimates, indicating that the effect of malaria risk tends to be relatively insensitive to cell-specific characteristics other than latent epidemic or endemic areas.

a less significant degree, the termination of violence as well as on the intensive margin of violence in terms of number of casualties (Tables A11 and A12).

5.2.2 Epidemic Areas: Alternative Coding and Potential Confounders

To explore the sensitivity of the baseline findings, and rule out spurious results, we replicated the results with an extensive set of time-invariant, geographic characteristics that might represent confounders, and with alternative codings for epidemic areas.

Potential Confounders: Geography, Population and Development and Placebo Diseases.

Epidemic areas differ from endemic areas (and areas that are not suitable for malaria) not only in terms of frequency of exposure to the pathogen.³⁰ We conduct various robustness checks that explore the potential role of interactions of other time-invariant features with the occurrence of malaria suitable months. In particular, we run a battery of specification checks that explicitly allow for interactions between malaria suitable conditions and a set of potential confounders in terms of:

- *Geography*: elevation, ruggedness, land suitability for agriculture (Tables A13, A14, A15, A16);
- *Agricultural Land and Forest Cover* (Tables A17 and A18);
- *Population and Development (proxied by light intensity at night)*: (Tables A19, A20, and A21).
- *Accessibility (measures of access to roads)*: (Tables A22, A23, and A24).
- *Placebo Diseases*: Risk of Dengue (Table A25), risk of Trypanosomiasis measured by TseTse suitability (Table A26), or prevalence of HIV in the population (Table A27).³¹

The results document that the baseline findings are very stable and robust while no systematic evidence of significant interactions between the listed cell-specific features and malaria suitable conditions can be detected.

Alternative coding of Epidemic Areas. The baseline results are obtained with the binary, time-invariant index of epidemic malaria areas, which is constructed based on values of the predicted malaria transmission stability by Kiszewski et al. (2004) below or above a threshold of 15. Estimates

³⁰Table S3 in the Appendix shows a balancing table.

³¹The measure of malaria risk, *MSM*, isolates specific suitable conditions for the transmission of plasmodium parasites that are informative on temporary spikes in malaria risk but should not interact systematically with the prevalence of pathogens transmitted from human to human or other vector-borne diseases involving different transmission vectors and cycles.

based on alternative measures deliver similar results. In particular, similar results are obtained when eliminating cells that are not suitable for malaria (see Table A28 and Figure A5 for a histogram of the distribution of the stability index across grid cells).³² Similar patterns as for the baseline specification are obtained when coding latent epidemic areas at a threshold of the transmission stability index of 10 or 20 (Table A29) or when using any threshold between 5 and 25 (Figure V2).

Effect of Acquired Immunities: Malaria risk within Epidemic Areas. The epidemic nature of malaria infections is associated with a lack of acquired immunities in the population. The prevalence of such immunities depends on the frequency of exposure to malaria in the past, which can be proxied by the level of malaria stability in each location.³³ To explore this specific prediction in more detail, we investigate the heterogeneity of effects *within* epidemic areas. We do so by replicating the analysis looking at sub-samples with different ranges of malaria stability and using weighting functions. The results are reported in Figure 3. Panel (a) reports the coefficient estimates of the effect of occurrence of suitable conditions for malaria transmission in a month, *MSM*, in sub-samples of cells at different ranges of the malaria stability index with increasing thresholds.³⁴ Alternatively, Panel (b) reports the results from applying a weighting function that assigns full weight to cells within a range around each threshold of the stability index, and then values that are gradually decreasing to zero as the index approaches its maximum value. The weighted regressions allow estimating the effect of malaria risk on the full sample, while progressively shifting the weight from low to high levels of malaria stability.³⁵

The findings document that the occurrence of a month with suitable malaria conditions increases the incidence of civil violence only within latent epidemic areas, with the magnitude of the coefficient spiking strictly within these areas. Furthermore, in line with evidence on the acquired immunities, the effect of latent malaria outbreaks on violence gradually washes out as the level of malaria stability

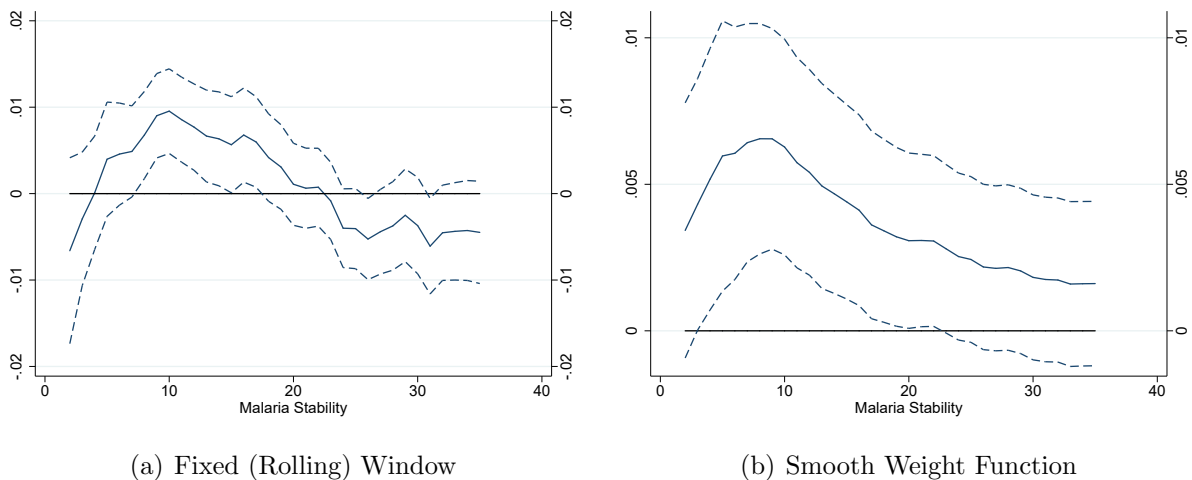
³²Moreover, the estimates are quantitatively almost identical to the main results, which rules out purely mechanical effects due to systematic differences in variation malaria stability.

³³As mentioned in Section 4.2 no information exists that would allow tracking the past exposure to malaria at high frequency. No consistent information is available, a fortiori, on the history of past exposure to malaria and survival rates across-locations and over time, which could serve as direct proxies of acquired immunities.

³⁴The sub-samples are selected as follows. We first construct a binary indicator for cells that fall within multiple (10-unit values) ranges of the index constructed by Kiszewski et al. (2004). For instance, we start estimating the effects of malaria suitable months for cells with malaria stability in range 1-10, then for cells in the range 2-11, and so on. This exercise essentially involves estimating the effect of malaria risk for the respective sub-samples of cells around different levels of malaria stability.

³⁵The results are insensitive to the particular width of the window or the particular weighting function used; the figure shows results for a weighting function that gives weight 0.1 to malaria stability index 0, weight 1 to malaria stability index up to the respective threshold depicted on the horizontal axis; above this threshold the weight is hyperbolically decreasing with a weight function of the form $\frac{1}{(index - threshold) + 1}$. Alternatively, we experimented with a weight function $\frac{(1-0.1)}{36.41181 - threshold}(index - 12)$ with very similar results.

FIGURE 3: EFFECT WITHIN EPIDEMIC AREAS



Panel (a) shows coefficient estimates obtained in sub-samples composed by grid cells within 10-unit ranges of malaria stability (e.g. 1-11, 2-12) with the x-axis indicating the highest level of the respective interval. Panel (b) shows alternative estimates obtained when applying parametric weights on the full sample. See text for details.

increases, with an effect that is effectively zero in endemic areas (with malaria stability above the conventional thresholds of 15 or 20). These findings are further reassuring that the baseline effects are not spurious as alternative potential unobserved confounders (in addition to those studied above) would have to account not only for the documented differential effect in epidemic and non-epidemic areas, but also for the specific patterns within epidemic areas.

5.2.3 Malaria Suitable Months: Non-linear Variation in Weather Conditions

Epidemiologists have documented that predicting conditions that are suitable for malaria transmission crucially requires tracking the specific non-linear combinations of weather conditions over several months as described for the construction of the *MSM*-index (see Section 4.1 and the Supplementary Appendix). One potential concern is whether the algorithms predicting malaria suitable conditions might pick-up short-term variation in weather that for some reason affect violence above and beyond their effect on malaria risk. The large set of robustness checks discussed above already partially address this issue by directly conditioning on weather conditions and their lags (up to 12 months). The possible confounding effect of short term weather conditions may not be limited to linear effects, however, or it may relate to interactions with epidemic areas. This concern is hard to explore systematically in the absence of a directed hypothesis. We nonetheless perform a series of additional checks.

Interactions with Weather Conditions in Epidemic Areas. In line with available epidemiological evidence that variation in weather conditions per se does not influence malaria risk, we find no evidence for any significant differential effect of weather conditions (in terms of precipitation, temperature, or the SPEI index) in epidemic and non-epidemic areas (see Tables A31, A30 and A32).

Non-Linear Effects of Weather and Floods. The results are confirmed by extending the specification to controlling non-linearly for weather conditions (and their 12 months lags) in specifications that also allow for month-specific effects in epidemic areas, and for different seasonality above and below the equator (see Table A33). To explore the role of threshold effects, particularly in terms of extreme increases in precipitation, we also confirm that the results hold also when further accounting for the direct and interacted effect of floods (see Table A34).

Malaria Suitable Months and Weather Conditions: Recoding and Placebos. Epidemiologists have emphasized repeatedly the need to check the fulfilment of *all* conditions that need to hold *during an extended period* (i.e., a sequence of months) in order to enable the reproduction of both parasites and vectors. To explicitly explore the specific role of malaria suitable months and to study the existence of potentially non-linear effects of weather conditions *per se*, we conducted two placebo exercises that involve a minor recoding of the measure of malaria risk. According to epidemiologists, this should imply that the ability to predict malaria risk with this recoded measure is lost.

First, we estimate the effect of the fulfilment of *each* of the necessary conditions in isolation, rather than their combination. The results show that the effect of malaria risk emerges when all conditions that are deemed necessary for malaria transmission jointly hold, while the realization of each specific condition in isolation (e.g. enough humidity regardless of the average temperature in the previous months) is not associated with any significant effect on civil violence (Table A35).

Second, we rebuild the index of malaria suitable months using information on the fulfilment of all weather conditions, but only during the current month. The results show that the effect of malaria risk indeed requires suitable conditions being met for an extended period in order to activate the malaria transmission cycle (Table A36).

The results of these exercises closely align with the epidemiological findings that the predictability of latent malaria outbreaks is highly sensitive to even small perturbations in the specific weather conditions that have been isolated as suitable for malaria transmission.

6 Exploring the Mechanisms

This section studies the mechanism behind the baseline results by exploring the testable implications, discussed in Section 3.2, 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.

6.1 Type of Events: Civil Violence *vs.* Geo-Strategic Conflicts

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. Exploiting information about the actors involved in each violent event in the ACLED data, we look at 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. The former two categories code events involving civilians and potentially confrontations between civilians and unorganized militias, while the latter categories refer to violence that is more related to geo-political aims and involves militarized rebel groups and confrontations between them and governmental forces.

The results are reported in Table 3. Spikes in malaria risk (in terms of *MSM*) increase the likelihood of violent events involving rioters or protesters, or confrontations between militias and civilians. The effect is statistically marginally significant even when accounting for cell \times year and calendar month fixed effects.³⁶ There is no evidence for an effect on political violence involving rebel groups and the governments. 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, between militaries and rebels and events involving only militaries - including non-violent events that involve changes in the control over territories and changes of headquarters (see also Table A37 for extended specifications).

6.2 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

³⁶Notice that this is a very demanding exercise in the context of our identification strategy, given the low unconditional probability of violent events of different types at the monthly level.

TABLE 3: TYPES OF VIOLENCE

Actors	Civil Violence				Geo-Political Motives			
	Rioters/ Protesters		Civilian <i>vs</i> Militias		Rebels		Military <i>vs</i> Rebels	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mal. Suit. Month	-0.000 (0.001)	0.000 (0.001)	-0.001 (0.001)	-0.001 (0.001)	0.002 (0.001)	0.001 (0.001)	0.001 (0.001)	-0.000 (0.001)
<i>MSM</i> × Epidemic Area	0.003*** (0.001)	0.002* (0.001)	0.004*** (0.001)	0.003* (0.002)	0.000 (0.001)	0.001 (0.002)	0.000 (0.001)	0.002 (0.001)
Weather	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weather L. 1-12	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Cell FE	No	No	No	No	No	No	No	No
Month × Year FE	No	No	No	No	No	No	No	No
Cell × Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month FE	No	Yes	No	Yes	No	Yes	No	Yes
Observations	457,560	457,560	457,560	457,560	457,560	457,560	457,560	457,560
R-squared	0.302	0.302	0.331	0.331	0.398	0.399	0.355	0.356
Number of Cells	2,556	2,556	2,556	2,556	2,556	2,556	2,556	2,556

The dependent variable is a binary indicator variable taking value 1 if at least one conflict event (ACLED dataset) was observed in the given cell in the given month that involved the respective actors listed. The results replicate the analysis reporting for each type of conflicts the results of the specification of Table 2 columns (4) and (5). See also text for details.

some time to unfold, the anecdotal evidence points at the risk of civil violence outbreaks being closely related with 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 disentangling different mechanisms behind reduced form effects documented above.

Timing: Lags and Leads. Table 4 presents results from an extended empirical specification that includes estimates of the effects of malaria suitable months during the past two months, or, as a falsification test, during the following two months. In line with warnings issued by development practitioners, the results provide evidence that spikes in malaria risk affect the incidence of violence on impact, and with an effect of a spike in malaria risk during the past month that is close to significant at conventional levels. At the same time, there is no evidence for the existence of anticipation effects related to future malaria shocks. The results also hold when considering both lags and leads, see Figure A7.

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

TABLE 4: TIMING OF THE EFFECT: LAGS AND LEADS (PLACEBO)

Dependent Variable	Violent Events - ACLED Monthly Panel			
	(1)	(2)	(3)	(4)
MSM	-0.003 (0.002)	-0.004* (0.002)	-0.003 (0.002)	-0.003 (0.002)
MSM \times Epidemic Area	0.006** (0.003)	0.008*** (0.003)	0.006** (0.003)	0.007** (0.003)
LAGS:				
MSM(t-1)	-0.001 (0.002)		-0.001 (0.002)	
MSM(t-1) \times Epidemic Area	0.005 (0.003)		0.005 (0.003)	
MSM(t-2)	-0.001 (0.002)		0.001 (0.002)	
MSM(t-2) \times Epidemic Area	-0.003 (0.003)		-0.003 (0.003)	
LEADS:				
MSM(t+1)		-0.000 (0.002)		-0.000 (0.002)
MSM(t+1) \times Epidemic Area		0.001 (0.003)		0.001 (0.003)
MSM(t+2)		-0.002 (0.002)		-0.001 (0.002)
MSM(t+2) \times Epidemic Area		-0.002 (0.003)		0.002 (0.003)
Weather	Yes	Yes	Yes	Yes
Weather Lag	Yes	Yes	Yes	Yes
Cell FE	Yes	Yes	Yes	Yes
Month FE	Yes	Yes	No	No
Month \times Year FE	No	No	Yes	Yes
Observations	457,560	452,476	457,560	452,476
R-squared	0.236	0.237	0.242	0.241
Number of Cells	2,556	2,556	2,556	2,556

The Table extends the analysis of Table 2 to the inclusion of the two lags of malaria risk and their interaction with epidemic areas (Columns 1 and 3) and studies the counterfactual placebo effect of two leads (and their interaction with epidemic areas).

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 A38).

Risk of Infection During Different Phases of Epidemic Outbreaks. As discussed in Section 3.2 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 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.

TABLE 5: VIOLENCE DURING DIFFERENT PHASES OF EPIDEMIC OUTBREAKS

Dependent Variable	Violent Events - ACLED Monthly Panel					
	(1)	(2)	(3)	(4)	(5)	(6)
<i>First Month (Onset Phase)</i>						
MSM	0.001 [0.003]	0.001 [0.003]	0.001 [0.003]	-0.001 [0.003]	-0.001 [0.003]	-0.001 [0.003]
MSM×Epidemic Area	0.001 [0.003]	0.001 [0.003]	0.001 [0.003]	-0.001 [0.003]	-0.001 [0.003]	-0.001 [0.003]
<i>Second Month (Acute Phase)</i>						
MSM		-0.002 [0.003]	-0.002 [0.003]		-0.002 [0.003]	-0.003 [0.003]
MSM ×Epidemic Area		0.009*** [0.004]	0.009** [0.004]		0.009*** [0.004]	0.009** [0.004]
<i>Third Month (Normalization Phase)</i>						
MSM			-0.002 [0.003]			-0.002 [0.003]
MSM×Epidemic Area			0.001 [0.004]			-0.001 [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	452,476	457,560	452,476	457,560	452,476
R-squared	0.236	0.236	0.236	0.242	0.242	0.242
Number of Cells	2,556	2,556	2,556	2,556	2,556	2,556

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 are 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.

The results in Table 5 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 percent 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 about the nature of these short-term shocks related to the discussion above.

6.3 Labor Productivity Shocks during Agricultural Cycles: Harvesting and Growing Seasons

The existing 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 economic consequences of malaria are related to shocks to labor productivity and labor participation, whose role for triggering violence has not been explored before. These shocks 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 FAO crop calendar, which lists information at the level of agro-ecological regions for the whole of Africa.³⁷ We use several alternative definitions for harvesting periods. Harvest months are coded as a binary indicator that takes 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 inter-temporal labor substitution, we discriminate crop bundles in terms of the duration of the harvesting seasons.

Table 6 presents the corresponding results.³⁸ 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)-(4) show that the effect of malaria shocks on conflict is, however, largest during short harvesting seasons (two months or less) with an effect

³⁷Notice that different geo-climatological conditions imply that the same crop may have different harvesting seasons in different geographical areas. The data reports 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 Table D2 for details.

³⁸The role of harvesting months in the Table is studied restricting attention to the subset of epidemic areas for which harvesting data are available in Panel A, and using the weighted regression approach as shown in Figure 3 in Panel B which allows for different levels of latent malaria risk by exploiting the full sample. These formulations avoid having to estimate additional main effects and triple interaction terms.

that tends to increase with the importance of the respective crop for subsistence (measured in terms of caloric yield).³⁹ These results are confirmed with alternative specifications on the full sample (Table A39).

Combining the analysis for different conflict types in Table 3 with the evidence on the role of harvesting months in Table 6 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 A40). 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 A41 and A42).⁴⁰ The baseline findings are also confirmed when accounting for interactions between growing seasons and weather conditions (Table A43).⁴¹

6.4 Attenuating Factors: Genetic Immunities and Anti-Malarial 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 in epidemic areas: genetic immunities and the extent of coverage with anti-malaria policies.

Genetic Immunity. To explore the potential role of genetic immunity as 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.⁴²

³⁹For 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, per se, periods with high opportunity cost for violent activities.

⁴⁰The growing season is constructed as a binary indicator variable that takes 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 Supplementary Appendix for details.

⁴¹This exercise effectively replicates and confirms the analysis of Harari and La Ferrara (2018) but at monthly frequencies and exploiting within cell×year variation. Accounting for interactions with weather conditions leaves the main effect of malaria shocks unaffected.

⁴²See 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

TABLE 6: MALARIA SHOCKS DURING SHORT HARVESTING MONTHS

Dependent Variable	Violent Events - ACLED Monthly Data			
Sample	Panel A: Only Epidemic Areas ($EA = 1$)			
	(1)	(2)	(3)	(4)
Malaria Suitable Month	0.006** (0.003)	0.005** (0.003)	0.005* (0.002)	0.005** (0.002)
Harvest Month	0.000 (0.003)	0.000 (0.002)	-0.008*** (0.003)	-0.009*** (0.003)
MSM×Harvest Month	0.000 (0.003)	0.005 (0.004)	0.014*** (0.004)	0.015*** (0.005)
Observations	141,300	141,300	141,300	141,300
R-squared	0.45	0.45	0.45	0.45
Number of Cells	785	785	785	785
Sample	Panel B: Full - Weighted Estimates			
	(1)	(2)	(3)	(4)
Malaria Suitable Month _{EA}	0.005** (0.002)	0.004* (0.002)	0.003 (0.002)	0.004* (0.002)
Harvest Month	-0.001 (0.002)	-0.001 (0.002)	-0.008*** (0.002)	-0.009*** (0.003)
MSM _{EA} ×Harvest Month	-0.000 (0.003)	0.007** (0.003)	0.015*** (0.004)	0.016*** (0.005)
Observations	360,730	360,730	360,730	360,730
R-squared	0.442	0.442	0.442	0.442
Number of Cells	1,993	1,993	1,993	1,993
Specification (both Panels):				
Importance of Bundle (Energy):	>1000 KJ	>500 KJ	>1000 KJ	>1500KJ
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 is available with “Malaria Suitable Months_{EA}” is an indicator that takes 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 value one for latent epidemic areas (the same indicator used in the baseline analysis and lower weights that are gradually decreasing to zero 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 is available.

The results in Columns (1) and (2) of Table 7 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 A44). Further epidemic.

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 (Tables A45 and A46).⁴³

Anti-Malaria Policies. To explore the role of anti-malaria policies as a potentially attenuating factor, we use information about the coverage of anti-malaria 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.⁴⁴ The results in Columns (3) and (4) of Table 7 reveal a negative effect of anti-malaria 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 A48).

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.⁴⁵

A Vaccine Thought Experiment. The potential role of health policies in this context can be illustrated by ways of a simple thought experiment. Considering the estimation results of the baseline model with cell \times year fixed effects, the hypothetical eradication of malaria, resulting from, e.g., the introduction of an effective vaccine, would be associated with a reduction in the incidence of violent events by around 14 percent (compared to a standard deviation of 27 percent). Figure 4 illustrates the spatial distribution of the benefits from this counterfactual exercise.⁴⁶ The results illustrate that

⁴³Additional results document that the effect of malaria risk is largest in areas with highest genetic and ethnic diversity (Table A47). 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, see, e.g., Esteban, Mayoral, and Ray (2012).

⁴⁴These data are only available for cells with frequent malaria exposure. See the Appendix for details on data sources and construction.

⁴⁵The occurrence of a month with conditions of elevated transmission risk for cells below the 25th percentile of the distribution of anti-malaria 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.

⁴⁶Formally, the incidence of violence is predicted for each cell and year using the baseline specification (corresponding to Table 2 Column (1)) while setting to zero the effect of malaria suitable months in cells in epidemic malaria areas. We use the difference between the two predicted models to get the average predicted reduction in incidence of violence (in terms of a share that lies in the interval between 0 and 1) for each cell. Finally, we use information about the

TABLE 7: GENETIC IMMUNITIES AND ANTI-MALARIAL POLICIES

Violent Events - ACLED Monthly Panel				
Moderating Factor	Genetic Immunities;		Anti-Malarial Policies	
	Sickle Cell %	Sickle Cell DV	Average Coverage	Maximum Coverage
	(1)	(2)	(3)	(4)
Malaria Suitable Month	0.009*** (0.003)	0.009*** (0.003)	0.016*** (0.004)	0.015*** (0.004)
MSM×Genetic Immunities	-0.072** (0.035)	-0.006* (0.003)		
Anti Malarial Policies			-0.178*** (0.014)	-0.054*** (0.016)
MSM×A.M. Policies			-0.096*** (0.032)	-0.045*** (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 Immunity” is the average prevalence (% of the population) of 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 bednet, and indoor residual spraying; policies 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 anti-malaria policy data are available.

eliminating malaria-related violence would affect particularly central Africa, the Great Lakes region and Eastern DRC, but also some Western parts of DRC or the deltas of the rivers Congo and Niger.

7 Concluding Discussion

This paper has contributed a first systematic investigation of the so far neglected role of epidemic shocks for civil violence. 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. However, no hard empirical evidence was available until now regarding the implications for the incidence of civil violence. 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 actual frequency of conflicts to compute the counterfactual predicted incidence for each cell over the full period.

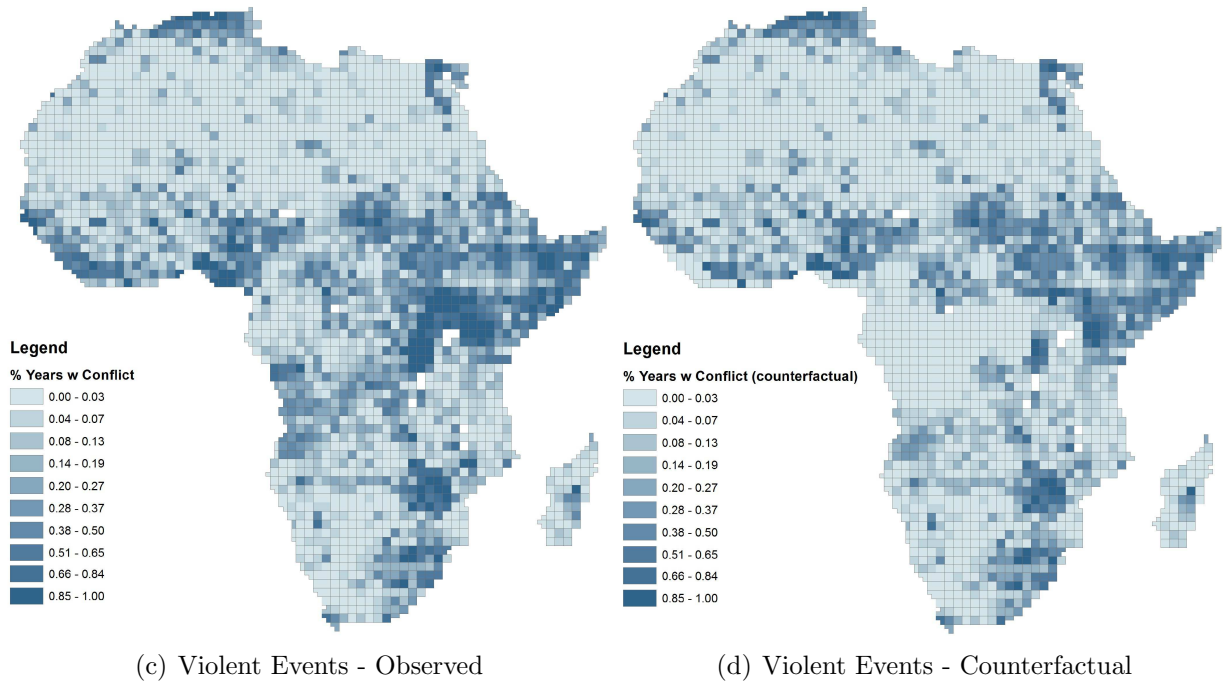


Figure 4: Comparing Actual and Predicted (Counterfactual) Conflict Incidence

Figure 4(c) depicts the actual incidence of violent events (fraction of years with at least one conflict over the period 1997-2012). Figure 4(d) depicts the predicted incidence of violent events obtained under a counterfactual “Malaria-Vaccine” scenario that switches off the estimated effect of malaria suitable months.

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 last 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, is concentrated in the month of the acute phase of the epidemic and spikes during sensitive production periods that are 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 anti-malaria 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. The results document important side effects of coverage with anti-malaria 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 the prioritisation of containment policies. While the development of a tool for directing measures of prevention of outbreaks of malaria and of malaria-related conflicts is clearly beyond the scope of the current 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 timely, i.e., within the first months of the epidemic. Finally, the results suggest important interactions between epidemiological dynamics and agricultural cycles, with important insights for agricultural practices and for the organization of labor.

Taken together, the results provide the first shred of systematic evidence regarding the potential 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 socio-economic 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 anti-malaria campaigns after the recent intensification, the results suggest that further research should be directed towards a better understanding of the potential role of climate change in the different areas at risk. The possible implications of increasing temperatures for the expansion of the spatial distribution of malaria to areas where it had not been present, or was not stable, in the past have been pointed out repeatedly. In addition, climate change could also lead to increasing weather variability and alter the frequency of occurrence of malaria suitable conditions.⁴⁷ Understanding the role of climate change and identifying the areas mostly at risk requires a dedicated, and potentially not straightforward, investment, but appears an important avenue for further research in the literature.

⁴⁷See, e.g. IPCC (2012) and Siraj et al. (2014).

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