Malaria infection and fetal growth during the war : evidence from Liberia

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Yuya KUDO*

February 2016

Abstract

This study investigates whether the Liberian civil war increased infant mortality by exposing pregnant women to a high risk of malaria infection, thus retarding fetal development. I find that the war-induced, one-percent increase in maternal infection risk resulted in a 0.44 percent increase in one-year mortality. This mortality effect gradually increased following childbirth as maternal passive immunity waned. The consequences were pronounced for infants conceived in rainy seasons by young mothers residing in rural, battle-intensive areas, with no gender difference detected. I also provide evidence suggesting the wartime culling of the weakest infants associated with maternal malaria infection.

Keywords: Armed conflict, fetal development, infant mortality, malaria in pregnancy
JEL classification: I15

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This study investigates whether the Liberian civil war increased infant mortality by exposing pregnant women to a high risk of malaria infection, thus retarding fetal development. I find that the war-induced, one-percent increase in maternal infection risk resulted in a 0.44 percent increase in one-year mortality. This mortality effect gradually increased following childbirth as maternal passive immunity waned. The consequences were pronounced for infants conceived in rainy seasons by young mothers residing in rural, battle-intensive areas, with no gender difference detected. I also provide evidence suggesting the wartime culling of the weakest infants associated with maternal malaria infection.

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1 Introduction

One legacy of civil war is the interruption of health accumulation. Following several pioneering studies (e.g., Akresh et al. (2011); Akresh et al. (2012a); Akresh et al. (2012b); Alderman et al. (2006); Bundervoet et al. (2009); Minoiu and Shemyakina (2012)), one recent leading question is in what ways an armed conflict harms the human capital stock rather than whether such harm occurs at all (Blattman and Miguel (2010), p. 42). This question is important to design effective assistance programs both during and after the conflict and to better understand wars' long-term influence on economic development. Accordingly, an emerging body of empirical research has only recently illuminated the significance of the limited effectiveness of consumption-smoothing strategies (Verpoorten (2009)), reduced dietary diversity (Dabalen and Paul (2014)), economic loss (Minoiu and Shemyakina (2014)), and prenatal maternal stress (Camacho (2008); Mansour and Rees (2012)) as mechanisms underlying health damage induced by conflict.

While malaria has historically been seen as one of the most important causes of violence-induced mortality and morbidity (Collier et al. (2003); Ross (1910), p. 577), few economic studies have assessed its influence in a rigorous manner. Armed conflict greatly increases the risk of malaria-related mortality because people seeking to escape from battle areas often hide in and/or travel through malaria-prone bushes and forests while being unable to maintain typical preventive actions (e.g., Foster et al. (2009)). Furthermore, despite this increase in infection risk, appropriate treatment is often unavailable due to health services impaired by the war.

By focusing on the Liberian civil war, this study explores whether the armed conflict increased infant mortality by exposing pregnant mothers to a higher risk of malaria infection, which would retard fetal development. This war provides a unique setting for the purpose of the current study. First, based on the 2011 Malaria Indicator Survey, this country is an ideal breeding ground for malaria-carrying mosquitoes. In almost all parts of this country, malaria transmission is possible throughout the year (see Appendix A.1 for a more detailed explanation of the geographic and climatic suitability).[1] In addition, due to the brutality of violence, approximately 60% of the pre-war populations was either internally or externally

[1]See National Malaria Control Program (NMCP) [Liberia], Ministry of Health and Social Welfare (MOHSW), Liberia Institute of Statistics and Geo-information Services (LISGIS) and ICF International (2012).

displaced during the 14-year conflict. Therefore, the Liberian people must have faced exposure to great infection risk when fleeing war-torn areas. Second, the large-scale armed conflict devastated Liberia's health system by destroying health facilities and inducing the deaths and flights of health professionals (Kruk et al. (2010)).

Infant mortality is worth exploring because among adults, mobile and pregnant populations are among the highest risk groups for malaria infection. In addition, it is well known that maternal malaria infection is highly associated with unfavorable birth outcomes (e.g., low birthweight), which in turn increase the risk of newborn deaths.

To address the question of interest, this study exploits child-level data based on the full birth history of the respondent females in two rounds (2007 and 2013) of the Demographic and Health Surveys (DHS) conducted in Liberia. The endemicity map of *Plasmodium falciparum* (*P.falciparum*), provided by the Malaria Atlas Project (Gething et al. (2011)), is also used to measure malaria infection risk. In Liberia, *P.falciparum* is a major plasmodium species (Patz and Olson (2006)) that produces the most deadly health consequences among human malaria parasites (WHO (2013), p. 72). Assuming that pregnant mothers hiding in and/or escaping from malaria endemic areas must have been exposed to greater infection risk than those doing so in and/or from less endemic areas, this study estimates an intent-to-treat effect (ITT) of malaria infection risk in pregnancy that increased in mothers' original places of residence at the time of the war.

By comparing changes in mortality for children conceived before and after the outbreak of the war between areas of high and low malaria endemicity (after controlling for fixed effects of the month- and year-of-conception as well as community-level fixed effects), this study will show that wartime pregnancy in areas of high infection risk exhibited increased infant mortality. This finding, provided by the differences-in-differences (DID) approach, is robust to the utilization of both the indicator and continuous measure of malaria infection risk, alternative controls (e.g., geographic and climate controls sourced from numerous geo-coded data sets, and mother-fixed effects that are expected to be the most powerful control), time-trends specific to each district, non-linearity of the empirical models (probit, hazard), alternative measures of malaria infection risk, and analyses exploiting separate sub-samples of the data.

This study will also show that war-induced infant mortality gradually increased and that it can be seen more clearly from six months after childbirth. It is expected that children born to a mother infected with malaria will have low birthweights and thus be vulnerable to several infectious diseases. While antibodies passed from mother to fetus protect such low-birthweight children, this maternal passive immunity wanes six to 12 months after the child is born. Therefore, this decrease in immunity might have made the vulnerability of low-birthweight children more evident while gradually increasing their mortality rate.

Moreover, the mortality effect was more pronounced for children conceived in rainy seasons by mothers residing in battle-intensive, rural areas, suggesting that these environmental factors increased the risk of malaria infection in wartime. Based on the knowledge of human biology, this study will also investigate whether parity- and (maternal) age-dependent immunity affects the mortality effect; it also provides evidence highlighting the importance of maternal age at birth. In contrast, the current research finds no significant gender differences in the adverse mortality impact.

This study will also argue that the DID estimate is a lower-bound effect of wartime malaria infection risk in pregnancy, and by taking three different approaches, it will provide some evidence supporting this view. These approaches include an assessment of the importance of unobservables (relative to the observed controls) required to explain the identified mortality effect (Oster (2015)) and an exploitation of an instrumental variable approach and of an insight obtained from a regression discontinuity design.

This study mainly contributes to three strands of the extant economic literature. First, the current study explores transmission channels driving human capital consequences of wars by focusing on the role of "malaria" infection in "pregnancy." By specifically investigating a country that experienced armed conflict, this research differs from the study conducted by Montalvo and Reynal-Querol (2007), which found an increase in malaria cases in countries that hosted refugees fleeing war. On the other hand, the current research reveals a similarity to Camacho (2008) and Mansour and Rees (2012), who examined the birthweights of children born to mothers exposed to violence during pregnancy from the perspective of maternal stress.

Second, within the field of economics, a rapidly growing body of research has demonstrated the negative influence of adverse prenatal conditions on human capital stocks. In contrast to some studies

investigating the role of maternal nutritional deficiencies (e.g., Almond and Mazumder (2011); van Ewijk (2011); Almond et al. (2015)), this research explores the influence of infectious diseases as others have done in the context of an influenza pandemic (e.g., Almond (2006); Almond and Mazumder (2005)).

Third, a socioeconomic consequence of infectious diseases is of general interest in health-related research. In contrast to several studies focusing on relatively unusual disorders such as HIV/AIDS (e.g., Fortson (2009); Kalemli-Ozcan and Turan (2011); Young (2005)), by exploiting "conflict" shocks, this study assesses the influence of a particular universal disease that possesses a major health problem in sub-Saharan Africa and elsewhere in the developing world (e.g., Kudamatsu et al. (2014)).

The rest of the paper is organized as follows. To facilitate an empirical analysis, Section 2 provides a review of the previous literature that links armed conflict with infant mortality, with particular emphasis placed on maternal malaria infection. The empirical strategy is presented in Section 3, followed by the data overview given in Section 4. Section 5 reports the empirical findings, with the concluding remarks summarized in Section 6.

2 Channels from war to infant mortality

The conceptual framework that underlies the subsequent empirical analysis can schematically be summarized as occurring through the following series of events: "fighting (subsection 2.1) ⇒ an increase in malaria infection risk (subsection 2.2) ⇒ pregnant mothers' infection with malaria (subsection 2.3) ⇒ fetal growth retardation (subsection 2.4) ⇒ an increase in mortality of weak infants associated with a ruined health system (subsection 2.5)." This section reviews the literature relevant to each of these stages.

2.1 Liberian civil war

While a detailed account is provided in Appendix A.2, Liberia's political unrest encompassed two civil wars between 1989 and 2003, with the first war taking place between December 1989 and July 1997 and the second war taking place between April 1999 and August 2003. During the 14-year state of chaos and fear, it is believed that more than 250,000 soldiers and civilians were killed, i.e., approximately more than

12% of the 2.1 million population in 1990 (Mama (2014), p. 55).[2] Due to the atrocious and indiscriminate nature of the fighting and human rights abuses (e.g., killings, looting, property destruction, rape, child recruitment), it is estimated that approximately 500,000 people were internally displaced and another 780,000 sought refuge abroad during the war.[3] Many factors (e.g., limited access to education and health services, severing of family and community links, concerns over gender-based violence, and absence of job opportunities) discouraged the displaced populations from returning to their homes after the war ended. Consequently, it took more than three years for the majority of the internally displaced persons (IDPs) to return or settle elsewhere since the Accra Peace Agreement was signed in 2003.[4] The UN refugee agency also completed a repatriation program in 2012 that facilitated the return of more than 155,000 refugees by the end of that year (Momodu (2013)).

2.2 Increase in malaria infection risk

Compared to combatant fatalities during a conflict, far more civilians tend to be killed, even after the conflict is over (Collier et al. (2003)).[5] Such high mortality rates are primarily attributed to infectious diseases among the IDPs and refugees, and their influence is highly persistent (e.g., Ghobarah et al. (2003); Ghobarah et al. (2004)). Of these diseases, malaria is seen as the most important cause of violence-induced mortality and morbidity for both displaced populations (e.g., Nafo-Traoré and Nabarro (2005); Rowland and Nosten (2001)) and host societies (e.g., Baez (2011); Montalvo and Reynal-Querol (2007)). While other diseases of acute epidemic potential (e.g., cholera, shigella dysentery, meningitis, and yellow fever) also play a role, their influence is usually short lived (Salama et al. (2004)).[6]

Conflict is expected to greatly raise the risk of malaria infection. To avoid areas of military operations,

[2]Information on the total population is sourced from "World Population Prospects: The 2012 Revision" (`http://esa.un.org/wpp/unpp/panel_population.htm`).

[3]Information pertaining to the externally and internally displaced persons is drawn from the "2005 UNHCR Statistical Year Book" (`http://www.unhcr.org/464478a72.html`) and "Liberia: Internal displacement in brief" as of December 2013, Internal Displacement Monitoring Center (IDMC) (`http://www.internal-displacement.org/sub-saharan-africa/liberia/summary`), respectively.

[4]The IDP camps were officially closed in April 2006, although this does not necessarily mean that all IDPs had returned to their place of origin by this time.

[5]Collier et al. (2003) provide a succinct summary of the adverse effects of civil war from several perspectives. In addition to the aspects of mortality and morbidity explained here, they also discuss the waste of productive resources, destruction of infrastructure, capital flight, loss of social capital, adverse political legacy, and psychological damage.

[6]Salama et al. (2004) also drew invaluable lessons from epidemiological studies on complex emergencies in the last decade of the 20th century. Based on their study, the major causes of mortality during the time of crises are fundamentally the same as those typical in developing countries (e.g., diarrhea, respiratory infection, and malaria), although such risks greatly increase due to the conflict. Malaria control is one of the most important health-related policy concerns in emergency settings (WHO (2005)).

for instance, people are forced to hide in and/or walk through unknown rural areas and forests widely inhabited by malaria-infected mosquitoes. These hiding and mobile populations also have difficulty exploiting typical risk-reducing strategies, such as staying inside a house at night, keeping doors and windows closed, cutting the grass, burning incense/firewood, avoiding unnecessary in-house water storage, and sleeping under bed nets.

In the Liberian war, fleeing populations often hid in swamps, bushes, and mountains for days, weeks, and even months in response to gunfire, government warnings or rumors of an assault, and surprise attacks (Foster et al. (2009)). This hiding was usually followed by internal displacement within Liberia and/or refuge in other countries. The cycle of fighting, hiding, and relocating was repeated during the long years of the conflict. In addition to these behavioral factors, deteriorating ecological conditions during the war (e.g., burned villages and inadequate sewage treatment) might also have enhanced the incidence of malaria.

2.3 Maternal malaria infection

Among adults, it is well acknowledged that pregnant women are at considerably high risk of contracting malaria.[7] Several insightful review and survey articles exist on this topic (e.g., Desai et al. (2007); Dorman and Shulman (2000); Lagerberg (2008); Steketee et al. (2001); Uneke (2007b)). Based on Desai et al. (2007), it is estimated that at least one in four women in malaria-endemic areas of Africa is infected with the disease at the time of childbirth. While adult females may be asymptomatic because of immunity acquired in childhood, their immune system still becomes weaker in pregnancy, particularly for primigravidae (Schantz-Dunn and Nour (2009)).

Maternal anemia is one of the major complications of this infectious disease in sub-Saharan Africa. Desai et al. (2007) estimated that approximately 26% of severe anemia among pregnant women can be attributed to malaria.[8] Pregnant women also suffer from a serious anemic burden due to the sequestration of the parasites in the placenta (placental malaria). As one recent estimate shows, in the absence of

[7] Other high-risk groups include infants, children under five years of age, HIV/AIDS patients, non-immune migrants, mobile populations, and travelers (`http://www.who.int/malaria/areas/high_risk_groups/en/`).

[8] This symptom is attributed to several factors, such as direct destruction of parasitized erythrocytes (i.e., hemolysis), immune responses shortening red cell survival, defective cell production, and hypersplenism.

pregnancy-specific interventions, approximately 41.2% of total pregnancies leading to live births would have contracted placental malaria at some stage during pregnancy in the high-transmission spectrum in Africa (Walker et al. (2014)). In Africa, co-infection with HIV could also strengthen the unfavorable association of malaria with maternal anemia (and possibly fetal development) because the virus impairs the mothers' immune system, which controls the malaria parasitemia (e.g., Brentlinger et al. (2006); Kuile et al. (2004); Steketee et al. (1996)); however, the prevalence of HIV/AIDS in Liberia was estimated at 1.5% and 1.9% based on the 2007 and 2013 DHS reports, respectively, which are substantially lower than the corresponding rates in eastern and southern African countries.

2.4 Influence of maternal malaria infection on fetal growth

It is widely accepted that pregnant women infected with malaria are prone to a variety of adverse perinatal outcomes, including miscarriage, stillbirth, intrauterine growth retardation, premature delivery, and low birthweight (e.g., Uneke (2007c)).

Among these, low birthweight (i.e., a weight at birth less than 2,500 g) is one of the most important factors that affect neonatal and infant deaths.[9] Based on Guyatt and Snow (2001, 2004), in malaria-endemic areas in Africa, babies born to mothers having an infected placenta are twice as likely to exhibit low birthweights than those born to uninfected mothers, and these low birthweights may be responsible for approximately 5.7% of the infant deaths per year in this region.[10]

While the underlying biological pathways are still subject to debate, maternal malaria infection is thought to reduce the birthweight of newborns by affecting gestational length and/or causing fetal growth restriction (or a combination of these factors). For example, maternal active immune responses (carrying antibodies, cytokines, and macrophages) induced by malaria-parasitized placenta may stimulate early labor, although establishing this theory requires more careful research (e.g., Guyatt and Snow (2004); Menendez et al. (2000)).

[9] Another possible factor that links maternal malaria infection with infant mortality is congenital malaria (i.e., transplacental transmission of malaria to the fetus). This may result from the materno-fetal circulation or direct penetration through the chorionic villi. Previously, it was presumed that congenital transmission was rare, but more recently, cases have been increasingly reported (e.g., Menendez and Mayor (2007); Uneke (2007a)). Moreover, malaria infection in pregnancy during wartime may increase maternal stress, which would further reduce the birthweight of neonates (Camacho (2008); Mansour and Rees (2012)).

[10] It is also estimated that approximately 60—80% of newborn deaths are cases of low-birthweight neonates (Lawn et al. (2005)). See Appendix A.3 for a review of factors affecting infant mortality in general.

In contrast to the influence of malaria infection on prematurity, its relationships with intrauterine growth retardation are better understood. As reviewed in more detail in Appendix A.4, Umbers et al. (2011) provided a good summary on the underlying pathological factors that link placental malaria with fetal growth restriction.

Finally, whether due to malaria or not, it should also be noted that maternal anemia is also expected to reduce neonates' birthweight (e.g., Levy et al. (2005)).

2.5 Influence of health system impairment on weak infants

Low-birthweight children are vulnerable to a range of infectious diseases, such as pneumonia, diarrhea, and malaria, all of which raise the risk of infant mortality (e.g., Lawn et al. (2005); Lawn et al. (2010); Liu et al. (2015)). Compounding this, the wartime impairment of the health system is expected to increase this already-raised mortality risk.

Due to the near-total destruction of Liberia's infrastructure (e.g., road networks, water and power supplies) and the looting of clinics and medicines, more than half of Liberia's 550 pre-war health facilities were deemed non-functional as of the end of 2003 (Lee et al. (2011)). In addition, the number of public health sector employees also shrank from 3,526 in 1988 to 1,396 in 1998 because many doctors and nurses died or fled during the conflict (Varpilah et al. (2011)). It is also estimated that nine out of 10 doctors left the country (Downie (2012)), leaving only 30 physicians serving a population of 3 million at the end of the war (Kruk et al. (2010)). The brutal conflict also deprived training institutions of appropriate resources to train health care workers (Duale and Mataya (2007)). While some humanitarian aid agencies provided health services during the fighting, most of the population had little or no access to such services (Kruk et al. (2010)), particularly in rural areas (National Transitional Government of Liberia (2004)). While the available evidence is limited, the wartime chaos might have also made it difficult for the majority of Liberians to attempt self-treatment and/or traditional remedies that might have been common health practices before the conflict. In fact, based on the author's field interviews made with Liberians in Buduburam refugee settlement in Ghana, the pre-war access to formal health services was limited in remote areas, and people often used to take traditional medicines (e.g., herb) to

self-treat malaria infection.[11]

3 Empirical strategy

During the conflict, the fleeing populations might have contracted malaria either during their travels or while residing in a temporary settlement.[12] Thus, this scenario would be ideal to assess the significance of the disease risk undertaken by the war survivors throughout the war. However, the current study cannot take this approach because it is difficult to trace their escape routes.

Alternatively, this study therefore attempts to estimate the wars' influence on children born to mothers residing in malaria-endemic areas at the beginning of the conflicts. Compared to pregnant mothers hiding in and/or escaping from less-endemic areas, those doing so in and/or from disease-prone areas must have been exposed to greater infection risk. In addition, at the time of the war, infants must have suffered from the destruction of the country's health system, and this problem must have been more serious in areas of high malaria transmission than in areas of low transmission. This is because it is expected that children from the former area had low birthweights due to their mothers' infections in pregnancy, and therefore, they were more vulnerable to a number of deadly infectious diseases. These infectious diseases might have included malaria, which is also more prevalent in high endemic areas. This study jointly tests these assumptions and the mortality consequences while estimating an intent-to-treat (ITT) effect of malaria infection risk in pregnancy that increased in the mothers' original places of residence at the time of the war.

[11] After getting a research permit from the Ghana Refugee Board in Accra, the author conducted a semi-structured questionnaire-based survey in Buduburam in February 2016. While the respondents were not randomly selected, in this survey, two males and five females originating from five counties (Grand Bassa, Grand Gedeh, Maryland, Montserrado, Sinoe) were individually interviewed, and the duration of each of those interviews was approximately 30 minutes. The interviews collected information on the respondents' brief life-history since they left Liberia and their preventive and treatment strategies against malaria before, during, and after the war.

[12] Related to this point, once malaria-infected mosquitoes feed on human blood in refugee camps, the disease tends to become epidemic due to the concentration of the populations. Furthermore, the camps are also often located on marginal land prone to vector breeding (e.g., near water sources) (Rowland and Nosten (2001)).

3.1 Specification

3.1.1 Empirical model

To estimate the aforementioned effect, this study primarily uses data drawn from two rounds (2007 and 2013) of the Demographic and Health Surveys (DHS) in Liberia that aimed to collect representative data on the population, health, and nutrition of females of reproductive age (15—49). While the DHS data allow different units of analysis (e.g., household, women, children, etc.), the main empirical analysis focuses on data pertaining to children to whom respondents gave birth in the past, i.e., the mother's full history of births (up to 20 entries).[13] In the data set, the birth year of the investigated children ranges from 1969 to 2013.

More precisely, for a child i conceived in a calendar month s of a year t by a mother k currently residing in a community j, this study exploits a linear probability model (LPM) and estimates

$$y_{ikj}^{st} = \alpha_1 + \alpha_2 w_{ikj}^{st} \cdot m_j + \alpha_3 \mathbf{x_{ikj}^{st}} + \alpha_4 \mathbf{x_{kj}^{st}} + v_j + \delta_s + \rho_t + \epsilon_{ijk}^{st}, \tag{1}$$

whereby y_{ikj}^{st} takes a value of one if the child died within Z months after birth and zero otherwise; m_j measures the risk of malaria infection in a community j; w_{ikj}^{st} is an indicator equal to one if the month s in the year t of conception belongs to periods following the outbreak of the war (i.e., after December 1989); the vectors $\mathbf{x_{ikj}^{st}}$ and $\mathbf{x_{kj}^{st}}$ contain controls specific to the child (e.g., gender, birth order, single birth indicator, mothers' age at birth) and the mother (e.g., education, religion), respectively[14]; v_j is a dummy for each community (620 communities; see Figure 1 for the positions); δ_s and ρ_t are fixed effects of the month- and year-of-conception, respectively; and ϵ_{ijk}^{st} represents a stochastic error. The community-fixed effects are expected to control for all time-invariant geographic and climate characteristics specific to each community. The fixed effects of the year-of-conception control for global time-trends that affected mortality between areas of high and low malaria risks in a similar manner, such as economic sanctions (e.g., Cortright et al. (2000), pp. 189-193).

While the periods following the outbreak of the war can be broken down into four different categories

[13]The selection of 20 entries is due to a restriction imposed by the DHS. In the data set exploited in this study, no births more than 17 were recorded.

[14]Information on respondents' ethnicity is not available in the Liberian DHS data.

(first war, ceasefire, second war, and post-war), the benchmark analysis avoided this disaggregation for two reasons. First, the ceasefire and end of the war do not necessarily mean the economic and relevant health situation immediately recovered (Kruk et al. (2010); Lee et al. (2011); Varpilah et al. (2011)). Second, the disaggregation did not reveal a noticeable difference in the estimated coefficients across those periods, as will be discussed later.

Regarding the malaria infection risk, this study uses an index sourced from the Malaria Atlas Project (Gething et al. (2011)) that ranges from zero to one. This index estimates the 2010 endemicity levels of *P.falciparum*, which is a major plasmodium species in Liberia (WHO (2013), p. 149) as the proportion of the infected populations aged 2—10 years.[15] Figure 1 provides a graphical representation of the index at each location (approximately 1 km^2) and the position of the DHS communities. In the empirical analysis, this study assigned the value of a raster point to each DHS community in its closest proximity in this endemicity map.

In contrast to standard nonlinear models such as logit and probit, the LPM enables coefficients on the interaction term to have a straightforward interpretation (Ai and Norton (2003)). To avoid a censoring problem, the analysis also exploits data on children born more than Z months before the month of the DHS interview. The standard errors are robust to heteroscedasticity and are adjusted for clustering on a community.

[Here, Figure 1]

3.1.2 Measurement issues

By exploiting the surrounding malaria endemicity of each DHS community as a measure of the infection risk that affected respondents during the war, the empirical strategy implicitly assumes that all the displaced populations returned to their original places of residence after the war. However, the assumption made here allows for the possibility that displaced populations resettled elsewhere in Liberia but might not have been widespread enough to invalidate the empirical analysis. Based on the 2007 DHS, at

[15] More precisely, this index averages a monthly proportion of children aged 2—10 years in the general population who are infected with *P.falciparum* at any one time over the 12 months in 2010. The infected population is estimated by exploiting data drawn from *Plasmodium falciparum* parasite surveys and Bayesian model-based geostatistics. The data can be downloaded from the Malaria Atlas Project (`http://www.map.ox.ac.uk/browse-resources/endemicity/Pf_mean/world/`).

least 55% of the respondent females were identified as permanent residents of the surveyed community (corresponding information was not available in the 2013 DHS). As shown later, the main findings of this study are robust to utilizing this sub-sample. Moreover, it is also noted that the remaining females include two groups: (1) those who were not permanent residents but who settled in the DHS community before the war and (2) young respondents who must have recently relocated due to patrilocal marriage into the surveyed community in the vicinity of their birthplaces.

Relatedly, this study also needs to assume that malaria endemicity has not noticeably changed over the last two decades. However, the situation relevant to malaria control in sub-Saharan Africa has remarkably improved since the turn of the 21st century, as reflected in the Roll Back Malaria initiative launched in 1998 and the Millennium Development Goals established in 2000. In addition, the Liberian Government established a National Health Policy in 2007 that planned to deliver a basic package of health services to citizens without charge, including services intended to control communicable diseases such as HIV/AIDS and malaria, thus making the above assumption somewhat doubtful.[16]

However, based on Noor et al. (2014) (Figure 6), the mean *Plasmodium falciparum* parasite rate (PfPR) was almost identical between 2000 and 2010 in Liberia. In addition, this global concerted campaign of malaria control only reached peak intensity after 2010 (Bhatt et al. (2015)). Moreover, the corresponding report of the 2007 DHS (p. 158) also refers to a huge gap between a national target within the national malaria policy and the present coverage of relevant interventions. It appears to take more time for those initiatives to achieve a significant improvement in this country (Kruk et al. (2010); Lee et al. (2011)).

For every child born to a female respondent, the DHS data contain information on year and month of birth. Given the presumption that a mother became impregnated nine months before delivery (e.g., January for children born in October), this study utilizes the estimated month of conception. In areas of high malaria transmission in Africa, the immunity that women acquire in childhood prevents the febrile episodes that often cause premature deliveries (Desai et al. (2007), p. 97). This medical knowledge may somewhat alleviate concerns over measurement noise pertaining to the timing of conception.

[16]The package also included emergency care, maternal and newborn health, and mental health care (Downie (2012)).

3.2 Identification

3.2.1 Parallel trend assumption

As seen from the specification (1), this study compares changes in infant mortality before and after the outbreak of the war between areas of high and low malaria transmission. The key identification assumption of this differences-in-differences (DID) estimation is that in the absence of the conflict, infant mortality would have followed parallel trends for children conceived in both disease-prone and remaining areas.

Exploiting the median value of the malaria endemicity index as the criteria of sample separation, Figure 2 plots the fraction of children who died within one year after birth by year of conception. Vertical lines indicate the timings of (1) the first war's outbreak (December 1989), (2) start of the ceasefire (August 1997), (3) beginning of the second war (April 1999), and (4) end of the second war (September 2003), respectively. The post-1977 fraction was presented in the figure because the yearly number of children conceived before 1977 was small (less than 100); thus, the estimated mortality rate is likely to be imprecise.

First, a remarkably similar trend of infant mortality between malaria-endemic and remaining areas was observed both before and after the outbreak of the war. Based on a more formal test described in Appendix B, the mortality trend was not significantly different between the high and low endemic areas of malaria parasites before the fighting took place. This finding lends great credit to the key identification assumption of the DID approach.

Second, while the pre-war mortality rate of children conceived in malaria-prone areas was lower than the corresponding rate in less endemic areas, after the war's outbreak, the mortality rate in the former areas reached the levels seen in the latter areas. This finding suggests unfavorable health consequences of wartime pregnancy in high-infection-risk areas, which the present study aims to identify.

In addition to these main findings, it may deserve repeating that before the war, the one-year mortality rate was higher in areas of low malaria endemicity than in areas of high endemicity. This is possible because the immunity women acquire by the time of pregnancy tends to be weak if they grew up in areas of low malaria transmission. In such areas, it is perceived that malaria infection during pregnancy results

in more serious symptoms, including deaths of the mothers and fetuses, compared to infections occurring in endemic areas (Desai et al. (2007), p. 96).

Finally, in areas of both high and low malaria endemicity, mortality levels have declined over the last two decades. As the corresponding report of the 2007 DHS indicates, this decline possibly stems from several organizational initiatives that made at least some progress in improving maternal and child health in this country, particularly in post-conflict periods (e.g., immunization programs, malaria prevention initiatives, suspension of user fees in all health facilities) (Kruk et al. (2010); Lee et al. (2011)). On the other hand, as noted in the 2007 and 2013 DHS reports, selective omission of childhood deaths from the data set may also explain the downward trend. Two major reasons account for this selection. First, respondents often fail to report pregnancies that did not result in normal delivery (e.g., miscarriage, stillbirth) and/or resulted in deaths early in infancy. Second, in the DHS data, only female respondents who survived the civil war (and returned to Liberia if the war had displaced them to other countries) were interviewed. If the violence increased the likelihood of unsuccessful pregnancy and/or females in good health in wartime predominantly constituted such survivors, these selection mechanisms lead to underestimation of the mortality rate during the war.[17]

[Here, Figure 2]

3.2.2 Potential underestimation

Despite the considerable support provided for the parallel trend assumption, however, it is still expected that this study underestimates the adverse influence of malaria infection risk for many reasons. First, women originally residing in areas of low malaria endemicity have less immunity to malaria. Therefore, whether by escaping to malaria-endemic forests or by coming into contact with people fleeing disease-prone areas, they tend to show higher propensity to contract the disease and undergo more serious birth outcomes. As a result, it appears that the mortality of children born to such women becomes high. On the other hand, the mortality of children born to mothers originating from areas of high endemicity would

[17]The gradual decline in mortality levels is also possible provided that such wartime deaths not recorded in the DHS started with the weakest mothers and infants and slowly moved to stronger ones.

tend to be low for the same immunological reason.[18] Somewhat relatedly, people living in malaria-endemic areas also tend to have sickle cell genes that make them malaria resistant (e.g., Livingstone (1958); Piel et al. (2010)). This may also result in underestimation of the mortality consequence in endemic areas.

Second, the timing of conception may not be seen as strictly exogenous. The estimated timing based on month of birth unavoidably includes noise. In addition, wartime childbearing (particularly in malaria-endemic areas) might have been possible only when parents could guarantee the safety of nursing the neonates, which reduced the risk of mortality. In the aforementioned field survey in Bududuram, the author identified one elderly Liberian refugee that midwifed another refugee during the wartime travel, and asked for the neighboring villagers' support provided for the mother after the childbirth.

Third, as noted in subsection 3.1.2, the measured malaria endemicity also suffers from a measurement concern due to the post-war resettlement issue of the displaced populations, which may attenuate the true effects of interest (as long as the measurement errors are classical).

Moreover, the aforementioned omission of childhood deaths from the DHS data may also play a role. If unsuccessful pregnancies were more pronounced in malaria-endemic areas than in low endemic areas during the conflict or if the wartime health condition of the female survivors was better in malaria-prone areas compared to that in low malarial transmission areas, the DID estimation is likely to underestimate unfavorable consequences for child survival.

Finally, the war might have increased malaria infection risk in pregnancy even in low endemic areas of malaria parasites. For any areas having malaria infection risk, the relevant counter-factual should have been those having no such risk, which are not exploited in the current study.

After showing the main estimation results, this study will provide evidence suggesting the potential underestimation of the DID estimate in subsection 5.6.

[18]Related to this point, as the war became protracted, malaria parasites might have been transmitted from high to low endemic areas (e.g., from rural immune to urban non-immune populations) due to great migration while reducing the mortality gap between the two areas. This is an important empirical concern because most IDPs sought refuge in Monrovia, the capital (Nilsson (2003)), whereby the mean value of the malaria endemicity index takes a value of 0.28 compared to the overall average of 0.42 in the data set. One possible way to investigate the presence of such spillover effects is to compare changes in mortality for children conceived before and after the war's outbreak between low malaria-endemic areas in Liberia and similarly malaria-endemic areas unaffected by the war. However, it is difficult to find such comparison areas because all the neighboring countries (i.e., Côte d'Ivoire, Guinea, and Sierra Leone) were influenced by their own conflicts and/or an influx of refugees fleeing from their neighbors during the periods of investigation.

4 Data

This study primarily uses the repeated cross-sectional data drawn from the Liberia Demographic and Health Surveys in 2007 and 2013, which were designed to provide nationally representative information in the fields of population, health, and nutrition (e.g., marriage, fertility, and child health).[19] The interviews were conducted from December 2006 to April 2007 for the 2007 survey and from March to July 2013 for the latter round, respectively.

In both rounds, a two-stage sample design was exploited. The first stage of the respective round selected communities (clusters) from enumeration areas of the 1984 and 2008 Population Census, followed by the second stage, which involved a systematic sampling of 25 (2007 round) or 30 (2013 round) households in each community. The survey team aimed at interviewing all women aged 15—49 years in each selected household, enabling the DHS to eventually contact 7,092 and 9,239 female respondents residing in 298 and 322 communities in the respective rounds. Exploiting the history of children born to all the female respondents (up to 20 births), the current research uses the children as the main analytical unit.

4.1 Immunity, infection risk, and mortality

As Figure 2 demonstrated, before the war, the one-year mortality rate was higher in less endemic areas than in high endemic areas while highlighting the weak (strong) immunity acquired by women who grew up in the former (latter) areas. Before providing summary statistics, this section describes in more detail the relationships between immunity, infection risk (measured by malaria endemicity), and mortality because it may facilitate an understanding of the effects this study aims at identifying.

After separating the sample children into 10 quantile groups based on the level of malaria endemicity in their mothers' residential areas, Figure 3 plots the one-year mortality rate of children conceived before and after the outbreak of the first war, respectively. Two findings are notable. First, the relationship between the endemicity and mortality is non-monotonic for both sampled periods. This is expected because at

[19]Data and relevant documents were publicly available from `http://dhsprogram.com/what-we-do/survey/survey-display-271.cfm` and `http://dhsprogram.com/what-we-do/survey/survey-display-435.cfm` for the 2007 and 2013 rounds, respectively.

very low levels of endemicity, the infection risk is too low to raise the infant mortality level even though pregnant mothers' immunity is weak. On the other hand, at the high end of the malaria transmission spectrum, the mothers' strong immunity can prevent infant mortality even if the infection risk is high. Consequently, the mortality rate is the highest for intermediate levels of endemicity. Notably, while the relationship between *levels* of infection risk and mortality is non-monotonic, this does not necessarily mean that *changes* in risk and mortality are also non-monotonic. As seen in the subsequent empirical analysis, the increase in infection risk induced by the war raised the rate of infant mortality in a monotonic manner.

Second, at all endemicity levels, the mortality rate declined after the first war began. This corresponds to the picture in Figure 2 showing that mortality levels have declined over the last two decades in both high and low endemic areas of malaria parasites. The DID approach attempts to show that this decline in mortality levels is smaller in high endemic areas than in low endemic areas, i.e., the war-induced infection risk increased the mortality rate in high endemic areas more evidently compared to that in low endemic areas.

[Here, Figure 3]

4.2 Summary statistics

For children conceived both before and after the outbreak of the war, Table 1 provides a description of several selected variables with checks on the equality of the mean between two groups characterized by the endemicity of $P.falciparum$. The sample separation was made based on the median value of the endemicity index corresponding to their mothers' (i.e., the DHS respondents') residential community. Information on malaria endemicity was provided by the Malaria Atlas Project (Gething et al. (2011)).

A few observations are worth noting here before proceeding with the empirical analysis. First, before the war, the one-year mortality rate was significantly higher in areas of low malaria transmission compared to the corresponding rate in malaria-endemic regions, but this difference disappeared after the war began. Second, in both high and low endemic areas, mortality rates have declined over time. In addition to these observations, which are consistent with the findings provided in Figure 2, malaria parasites were more

prevalent in rural (and likely forested and mountainous) areas of high altitude, which are intuitive findings because such environmental characteristics make the parasite's development easier.

While this information will be explained in more detail and used in the analysis presented in subsection 5.3.3, this study also exploited a map of monthly mean temperatures (multiplied by 10 °C) and precipitation amounts (mm) between 1950 and 2000 provided by the "WorldClim - Global Climate Data" (Hijmans et al. (2005)) and assigned each DHS community the value of a raster point at its closest proximity. Based on these data, malaria-endemic areas show lower temperatures (probably due to high altitudes) with lower amounts of rainfall than less malaria-endemic areas.

This study also calculated the number of battle events that occurred within a 25-km radius from each DHS community based on information sourced from the Armed Conflict Location and Event Database (ACLED).[20] ACLED is a well-known data set used in the scientific analysis of conflict (e.g., Adhvaryu and Fenske (2014); Dabalen and Paul (2014)), and it contains information on specific dates and locations of political violence from 1960 to 2004, types of events, and groups involved (Raleigh et al. (2012)). In Liberia, 265 battle events that occurred between April 1980 and July 2003 were recorded in this data set (see Figure A.1 for battle locations). As the summary statistics reveal, the fighting occurred more intensively in less malaria-endemic areas of lower-altitude (more urban) land. This information will be exploited in the empirical analysis conducted in subsection 5.4.1.

[Here, Table 1]

5 Estimation results

5.1 Main results

The estimation results of one-year mortality ($Z = 12$) of the specification (1) are reported in Table 2. The analysis in column (a) separated the sample into 10 categories based on the value of malaria endemicity, and the corresponding indicators for each category are included (the reference group is the lowest percentile). Based on the result, wartime pregnancies in the groups of 60—100 percentiles of

[20]The data and relevant documents are freely available from `https://www.prio.org/Data/Armed-Conflict/Armed-Conflict-Location-and-Event-Data/`.

malaria endemicity experienced significantly increased one-year mortality rates by approximately 4—5 percentage points. The estimation in column (b), which alternatively exploited an indicator for the highest 50 percentiles of malaria endemicity, also revealed a 3% increase in the mortality rate in the corresponding areas.

As the one-year mortality rate of children conceived before the war was approximately 17% based on the exploited data, the 3-percent increase in infant mortality accounts for about 17.6% of the pre-war mortality. In addition, the estimated mean proportion of the infected population aged 2—10 years (i.e., values of malaria endemicity index) is about 0.34 and 0.49 in areas belonging to the lowest and highest 50 percentiles of malaria endemicity, respectively. By combining these numbers with the 17.6% change in the mortality rate, therefore, it can be determined that the war might have caused a 0.44-percent increase in the infant mortality rate in response to a one-percent increase in the infection risk measured by the infected population.

It is difficult to speculate on this magnitude in terms of the number of infants killed by maternal malaria infection during the war and to find estimates comparable to this elasticity. However, the impact size is non-negligible, considering the presumption that the estimated coefficient does not necessarily capture the full effect of maternal malaria infection. This is because not all women residing in malaria-endemic areas may contract the disease. Since the infection probability is typically less than unity, the treatment effect of malaria infection is still likely to be larger than the ITT estimate (i.e., treatment effect of malaria infection risk) shown in the present analysis.

The exercise in column (c) replaced the dummy for the upper 50% quantile of malaria endemicity with the continuous value of the index. Exploiting the continuous measure also confirmed the view that after the war, children conceived by mothers residing in malaria-endemic areas more clearly lost their life within one year after birth compared to those conceived by mothers in low endemic areas.

By including the fixed effects of year-of-conception, the previous estimations controlled for all time-varying factors that affected areas of high and low malaria endemicity in a similar manner. However, it is possible that the war noticeably changed the health-related infrastructure across areas having different levels of malaria endemicity while biasing the estimated α_2. To mitigate this concern to some extent, this

study grouped the sample data into 66 categories based on administration areas of "districts" (see Figure A.1 for the boundaries) and in the analysis of column (d) in Table 2, controlled for a time trend (over 40 years) specific to each district.[21] The exercise exploiting the continuous measure of malaria endemicity yielded similar estimates to those obtained in column (c).

To alleviate the potential bias attributed to possible pregnancy cases that were not reported to the DHS team, the analysis in column (e) included an indicator equal to one if a mother had ever experienced a pregnancy that terminated in a miscarriage, abortion, or stillbirth. This additional control left the implications obtained from the previous estimations almost unchanged.

As discussed in subsection 3.2.2, wartime childbearing might have been possible only for those with particular parental characteristics (e.g., health safety). To control for such (only) time-invariant characteristics, this study replaced the previously exploited covariates relevant to mothers and community-fixed effects with mother-fixed effects and then re-estimated the specification (1). Column (f) reports the estimation result, which again highlights the adverse mortality effect of wartime pregnancy associated with high malaria infection risk.

Another empirical challenge noted in subsections 3.1.2 and 3.2.2 is that the respondents might have resided in different locations from the current DHS communities because the crisis induced massive population displacement. In this case, the measured malaria endemicity includes some noise. To determine whether previous findings were sensitive to this concern, two exercises were performed. First, this study exploited data pertaining only to children born to mothers who were identified as permanent residents of the surveyed community in the 2007 DHS and re-estimated the specification (1) in column (g). Data for the 2013 DHS were not exploited in this estimation because the relevant information was not available in that round. Second, during the conflict, most IDPs headed towards the capital, Monrovia (Nilsson (2003)). In addition, Liberian refugees were repatriated to the capital under a program run by the UN refugee agency after the war. Because some of those people preferred to stay there instead of returning to their original homes in the post-war periods, the current residents of the capital area must show a

[21] It was not possible to identify a district corresponding to each community from the DHS data alone. To achieve this purpose, therefore, this study matched a community's GPS latitude/longitude coordinates with a map of Liberia sourced from DIVA-GIS (`http://www.diva-gis.org/datadown`). Consequently, the communities were categorized into 65 districts plus one group for which the ArcGIS failed to identify the corresponding district. The analysis in column (d) in Table 2 included the unidentified group as one district because it constituted only 3% of the sample.

great tendency to have resided in different places during the years of the conflict. This is particularly true if they no longer had any relatives living in their original communities (Jesuit Refugee Service (2007); Omata (2012)). Given this likelihood, the other exercise conducted in column (h) excluded from the estimated sample those currently living in the Greater Monrovia District. As is evident from the estimation results, the additional analyses based on these sub-samples of the data yielded similar implications to that obtained before.[22]

[Here, Table 2]

5.2 Robustness checks

5.2.1 Survival lengths

The specification (1) was estimated for different lengths of child survival by varying the value of Z; Figure 4 reports the estimated α_2 with 95% confidence intervals. The left-hand panel uses the indicator for the upper 50% quantile of malaria endemicity, in contrast to the right-hand panel, which exploits the continuous value of the index. Two interesting findings emerged. First, the unfavorable effects of wartime pregnancy in malaria-endemic areas gradually increased as this study examined longer survival periods, as can particularly be seen in the estimations based on the indicator of malaria endemicity (left-hand panel) as well as for analyses of survival periods equal to or less than 12 months (i.e., $Z \leq 12$). Second, the mortality effects became more statistically significant as the examined survival periods grew longer than six months.

Presumably, children born to a mother infected with malaria have low birthweights and thus are vulnerable to several infectious diseases. Although antibodies passed from a mother to the fetus through the placenta still protect newborns, in humans, such maternal passive immunity wanes over six to 12 months after the child's birth (e.g., Niewiesk (2014), p. 2). Therefore, the infant mortality rate might have gradually increased as the vulnerability of such low-birthweight children became more evident.

[22] Admittedly, limiting analytical attention to sub-samples possibly introduces selection bias because the choice of residential places is endogenous. In the present context, the permanent residents might have resided in less conflict-affected areas. However, regressing a dummy for children conceived by mothers identified as permanent residents on the number of battle events that occurred within a 25-km radius from DHS communities as well as fixed effects of year-of-conception and of districts yielded a coefficient that was insignificantly different from zero. Replacing the number of battle events with malaria endemicity or simultaneously including these variables in similar regressions also identified insignificant relationships between these variables and the outcome dummy.

[Here, Figure 4]

5.2.2 Non-linear empirical models

By exploiting probit and Cox proportional hazard models, this study also checked whether the aforementioned findings were robust to non-linear empirical models (and controls), as detailed in Appendix C.1. The relevant exercises provided support for the findings provided by Table 2 and Figure 4.

5.2.3 Trend of the mortality effects

In column (a) in Table 3, this study interacted the previously exploited indicator for areas of high malaria endemicity (50—100 percentiles) with four different periods of conception, namely, first war, ceasefire, second war, and after the end of the war, and estimated a version of specification (1). Compared to the inter-war periods, it is expected that people have lower infection risk in the post-war periods. However, the results show significant mortality effects even after the war ended. Exploiting a continuous measure of malaria endemicity in column (b) also did not affect this finding.

This finding is possible because the ITT interpretation of mortality consequences includes the influence of limited access to pre-natal and post-natal care. While the 2007 National Health Policy has contributed to progress in providing basic health services, Kruk et al. (2010) noted that the ambitious target set by the government was unlikely to be achieved by 2010 based on their study exploring the availability of essential health services in 2008. They attempted to survey a "typical" rural population and showed that just one-quarter of their respondents had access to basic emergency obstetric care, while even fewer obtained the integrated management service of childhood illness.[23] The overall service quality is also low, with long travel times required at the nearest health facilities (Lee et al. (2011)), long waits at clinics, few available medicines, impassable dirt roads (particularly in the rainy season), enormous rural—urban disparities, and so on (Downie (2012)).

On the other hand, the Ministry of Health and Social Welfare has also attempted to increase the size and capacity of human resources for health, and the number of health workers greatly increased from 2006 to 2010 (Varpilah et al. (2011)). Whether due to this increase or not, it may deserve mentioning

[23]The integrated management of childhood illness seeks to prevent deaths from pneumonia, malaria, and diarrhea.

that the mortality effect has shown a declining tendency starting with conceptions occurring in 2009, as seen in Figure 5. This figure further disaggregated periods following the outbreak of the first war and reported the estimated α_2 (with 95% confidence intervals) specific to each year of conception.[24]

[Here, Table 3 and Figure 5]

5.2.4 Alternative measures of malaria infection risk

In Appendix C.2, this study also checked whether the main findings were robust to the exploitation of alternative estimates of malaria endemicity provided by Bhatt et al. (2015). The key implications remained unchanged.

5.3 Threats to interpretation

5.3.1 Conception or birth?

One concern regarding the previous findings is that the unfavorable mortality effect may be attributed to malaria infection occurring in infants themselves in malaria-endemic areas because the current research estimated the timing of conception based on the month of birth.

To mitigate this concern, this study replaced w_{ikj}^{st} in the specification (1) with an indicator equal to one if the child was born after the outbreak of the war and then re-estimated the equation. If the present concern is true, this alternative indicator will serve as a more accurate measure of factors driving the unfavorable health consequence; therefore, it is likely to identify greater influences on the infant mortality than the w_{ikj}^{st} did.

The estimation results exploiting a dummy variable for the upper 50% quantile and continuous measure of malaria endemicity were reported in columns (c) and (d) in Table 3, respectively. Compared to the estimation results presented in Table 2, the adverse mortality effect is attenuated and its statistical significance is weak, suggesting that the timing of conception plays a more important role in explaining child survival than that of birth. This study also simultaneously included both the w_{ikj}^{st} and alternative indicator (interacted by the measured malaria endemicity) in regressors and reported the estimation

[24] In these estimations, children conceived in December 1989 (i.e., the beginning of the first war) were included in a group of those conceived in 1990 for the sake of tractability.

results in columns (e) and (f). Admittedly, exploiting two highly positively correlated variables in the same estimation tends to result in overestimating one parameter and underestimating the other based on the informal statistical guidelines (Williams (2013)). Nevertheless, the results still highlighted the significance of the timing of conception.

It is also important to note that maternal malaria infection could reinforce an infant's own infection because children born to mothers infected with malaria are expected to have low birthweights and are therefore vulnerable to several infectious diseases, including malaria. In this case, the influence of an infant's own infection (i.e., timing of birth) would include effects of maternal infection (i.e., timing of conception).

5.3.2 Sexual violence

During the war, acts of sexual and gender-based violence against women and girls were exploited as weapons of warfare. This raises the issue that children born of these unwanted events might have been abandoned or killed shortly after birth (e.g., Denov (2015)). If such violence was more evident in malaria-endemic areas, the previous findings may be consistent with this possibility.[25]

However, the current study sees this possibility as less likely for several reasons. First, to the best of the author's knowledge, such cases of infanticide are not supported by the relevant qualitative research in Liberian contexts (e.g., Foster et al. (2009); UNFPA (2011)). Second, as described in Section 4, the fighting was *less* intense in malaria-endemic areas. Third, if this alternative interpretation is true, it is likely that newborn deaths will frequently occur during the immediate months following these births. The gradual increase in mortality rate (based on the examined periods of child survival) shown in Figure 4 is not compatible with this prediction. Fourth, this scenario cannot explain the long-term positive selection effect on the health of female survivors, as discussed in subsection 5.5.

[25]Recent scientific research has also revealed systematic relationships between climate (e.g., high temperature, high precipitation) and frequency of violence (e.g., Cane et al. (2014); Hsiang et al. (2013)), and subsequent analyses made in the fifth and sixth columns in Table 5 showed that the significant mortality effect was more pronounced for children conceived in rainy seasons. By exploiting data sets of war crimes recorded by the Truth and Reconciliation Commission (TRC), which amount to 163,615 cases sourced from 17,160 testimonies (see Table A.12 for the breakdown), and data sets of battle events drawn from the aforementioned ACLED, this study found some (albeit marginal) evidence suggesting that human rights violations more frequently occurred in rainy seasons during the time of conflict (see Appendix E for details of the analysis).

5.3.3 Maternal undernutrition

It is acknowledged that poor nutritional status in pregnancy positively correlates with adverse birth outcomes, such as low birthweight and preterm delivery (for example, see Abu-Saad and Fraser (2010) and Villar et al. (2003) for an overview of the relevant observational and experimental studies, respectively). Accordingly, if the malaria-endemic areas were food insecure in wartime, maternal nutritional deprivation may also explain the previous findings.[26] Needless to say, maternal undernutrition increases the risk of morbidity and mortality resulting from malaria infection; therefore, these two factors may not mutually be exclusive. Nevertheless, this concern still deserves attention.

This study also included in the specification (1) interaction terms between w_{ikj}^{st} and proxies for community-level food availability, which is measured by climatology, land scape, and soil quality in the vicinity of each DHS community (see also Appendix F for details) as well as a community's GPS-based coordinates. These estimation results are reported in Table 4.[27] In this table, only the relevant coefficients are presented. For each type of these geographic and climatic conditions as well as for the GPS latitude and longitude position, the results exploiting an indicator for malaria-endemic areas and a continuous measure of malaria endemicity are reported in Table 4. In the presence of the above concern, the malaria-related mortality effect may disappear due to the inclusion of these additional controls.

The "WorldClim - Global Climate Data" provided information on mean monthly precipitation amounts (mm) and temperatures (multiplied by 10 °C) over 1950 and 2000 with a spatial resolution of 30 seconds (≈ 1 km^2) longitude/latitude in a raster format (Hijmans et al. (2005)). In addition to information on the elevation (m) of each community, which was based on the Shuttle Radar Topography Mission (SRTM) Digital Elevation Model (DEM) and directly available in the DHS data, this study also took information on slope (percent) and terrain ruggedness index (100 m) from data provided by Nunn and Puga (2012) at the cell levels on a 30 arc-second grid. The information on soil quality was sourced from a 30 arc-second raster data set provided by the "Harmonized World Soil Database" (Fisher et al. (2008)). For each of

[26] The pronounced mortality effect revealed in Table 5 for children conceived in the rainy season may also be consistent with the fact that in Liberia, this season almost overlaps with hunger periods given heavy agricultural workloads (Owadi et al. (2010), Figure 4.7) due to the frequent heavy rains, inaccessible roads, limited access to markets, depletion of food stocks, food price hikes, and so on; however, the relationship between mothers' dietary intake and positive or negative birth outcomes may differ by pregnancy stages, types of nutrients (e.g., energy, protein, iron, zinc, vitamin A, folate, vitamin D), and a combination of these factors.

[27] The GPS position may also represent any confounding factors inherent to each community (e.g., sanitary conditions).

six soil quality variables (nutrient availability, nutrient retention capacity, rooting conditions, oxygen availability to roots, excess salts, and field-management constraint), this study created an indicator for a community characterized as having "moderate, severe, or very severe constraint" (reference group is "no or slight constraint").[28]

As the results in Table 4 show, the inclusion of variables relevant to climatology [columns (a)—(b)], landscape [columns (c)—(d)], soil quality [columns (e)—(f)], and GPS coordinates [columns (g)—(h)] almost did not affect the previously identified effects of wartime malaria infection risk in pregnancy. While the results are not reported to save space, simultaneously including all those geographic and climate controls also left the obtained implications unchanged.

[Here, Table 4]

5.3.4 Other tropical diseases

If malaria-endemic areas were prone to other tropical diseases, the unfavorable consequences for infant mortality may also be consistent with this possibility. Among "neglected tropical diseases" including hookworm, ascariasis, trichuriasis, schistosomiasis, lymphatic filariasis, onchocerciasis, and dengue, (only) the estimated number of people infected with schistosomiasis and onchocerciasis is comparable to that of those infected with malaria in Liberia (e.g., Hotez (2015)).[29] However, morbidity of pregnant women and their children attributable to the first is largely unknown (Friedman et al. (2007)), and a major symptom of the second is blindness, both of which have not been highlighted by the relevant qualitative research in the context of the Liberian war. The vector-mediated transmission of dengue is also much less common in this country (e.g., Amarasinghe et al. (2011)). The aforementioned geographic and climate controls may also help in mitigating the present concern.

[28] Soil quality data also included information on toxicity. All DHS communities were identified as having "no or slight constraint"; however, this information was not exploited in this study. In addition, analyses utilizing these soil quality variables excluded from the regressions children born to mothers residing in 30 communities (out of 620 communities) whose nearest raster point was not located on land, which corresponded to approximately 4% of all the recorded childbirths.

[29] It is thought that hookworm infection in pregnancy can also result in adverse birth outcomes (e.g,. premature delivery, low birthweight, and intrauterine growth retardation).

5.4 Heterogeneity

5.4.1 Environmental risk

Several environmental factors might have raised malaria infection risk in wartime pregnancy while further discouraging child survival. Three relevant perspectives were examined in Table 5: battle intensity, types of residential areas, and seasons of conception. Analyses made in panels (A)—(C) in that table exploited an indicator for the upper 50% quantile of malaria endemicity, compared to the remaining panels (D)—(F), which used the continuous value of the index. Given the significance of maternal passive immunity suggested by the exercises performed in Figure 4, the estimations in this table (and Table 6) investigated periods of child survival equal to or longer than 12 months (more precisely, $Z = 12$, 36, and 60 months). For brevity's sake, this study also suppressed coefficients on controls, which are available upon request.

First, this study explored the possibility that the unfavorable mortality effect was more pronounced in areas more frequently affected by the armed conflict. For this purpose, exercises conducted in the first two columns in Table 5 split the sample based on the number of battle events that occurred within a 25-km radius from each DHS community and independently estimated the specification (1) for both the sub-samples. The sample median number of fighting incidents was exploited as the criterion of sample separation. As explained in Section 4, the information on the battle events was sourced from the ACLED. As the estimation results in the first two columns show, particularly in panels (D)—(F) based on the continuous measure of malaria endemicity, a significant mortality effect was more evident for infants born to mothers (currently) residing in war-torn areas.

Given the presumption that the mortality effect is more significant in rural than in urban areas due to a likely high infection risk and/or limited access to health services (e.g., few health facilities, dirt roads difficult to traverse in rainy seasons), the specification (1) was estimated for both the sub-samples of (the current) rural and urban residents in the third and fourth columns in Table 5. The results supported the expectation.

Typically, malaria transmission positively correlates with relatively predictable patterns of precipitation (Stanley C. Oaks et al. (1991), pp. 217-218). While malaria infection is possible throughout the year in Liberia due to its climatically suitable nature for the vector-mediated transmission, this picture

still seems true even in this country. *Anopheles gambiae*, a principal anopheles species in Liberia (WHO (2013), p. 149), is most abundant during the rainy season in this country (e.g., Fahmy et al. (2015); Gelfand (1955); Hogh et al. (1993); Somah (2005)). Based on these arguments, this study estimated the specification (1) for both the sub-samples of children conceived in rainy (May to October) and dry seasons (November to April) in the fifth and sixth columns in Table 5, respectively. Strictly speaking, interpretation of the estimates is not straightforward because the seasonal influence of maternal malaria infection varies by three factors, i.e., gestational timing of the highest infection risk, overlap between gestation periods and malaria-prone seasons, and gestational timing, yielding the worst birth outcomes attributed to malaria in pregnancy (see Appendix A.5 for details). Acknowledging this limitation, the results showed that the negative health consequences of wartime pregnancy in malaria-endemic areas were more clearly identified for children conceived in the rainy reason than for those conceived in the remaining periods.

[Here, Table 5]

5.4.2 Human biology

As shown in Table 6, this study also investigated whether the influence of wartime pregnancy in malaria-endemic areas varied by biological factors from two standpoints: maternal immunity and child genetics. Similar to Table 5, both the indicator and continuous measure of malaria endemicity were exploited in the estimations in panels (A)—(C) and (D)—(F), respectively, with each panel corresponding to analyses of different survival periods (i.e., Z = 12, 36, and 60 months). As before, coefficients on controls are suppressed but are available upon request.

First, women acquire parity-dependent immunity; therefore, it is viewed that first- and second-time mothers are at greater risk of malaria infection compared to multigravidae (e.g., Desai et al. (2007); Schantz-Dunn and Nour (2009); Uneke (2007b)). Similarly, young maternal age also independently increases the risk of infection due to acquisition of age-associated immunity.[30] Accordingly, this study separated the sample into children born to paucigravidae and the others in the first two columns in Table

[30]In general, it is possible that HIV/AIDS eliminates this gravidity- or age-specific patterns of malaria risk by shifting the burden to all pregnant women (Desai et al. (2007)), although the prevalence rate of this disease is low in Liberia as described in subsection 2.3.

6 and into those born to young (lower 50% quantile of mothers' age at birth) and elderly mothers (upper 50% quantile) in the next two columns.

The results confirm only the age-dependent heterogeneity of the mortality effect. This study might have failed to identify the presence of gravidity-dependent heterogeneity because those identified as the first and second children in the data set might have had elder siblings who did not experience normal deliveries and/or who died early in infancy and thus were not reported to the DHS team. Alternatively, the diagnosis of malaria in pregnancy is sometimes sensitive to exploited methods (e.g., Othoro et al. (2006)); therefore, the parity-dependent heterogeneity may not be uncontroversial.[31]

The level effects of a male dummy reported in Table 2 are consistent with the oft-cited biological "weakness" of male infants relative to females (e.g., Waldron (1983)). To explore whether this male vulnerability made a significant difference in the malaria-related mortality effect across gender, this study separately estimated the specification (1) for boys and girls, and the results are reported in the final two columns in Table 6. As seen from the results, such a gender difference in mortality was not clearly observed.

Two remarks can be made regarding this finding. First, the finding may be consistent with that provided by Akresh et al. (2011). They showed that in Rwanda, the armed conflict equally reduced the height-for-age of boys and girls while highlighting the indiscriminate nature of the violence (compared to a peacetime crop failure, which made a significant gender difference). Second, if boys are more vulnerable than girls, the "no gender-difference" result suggests that the identified mortality consequences may be underestimates of the total population effect. This is because this study might have failed to consider the mortality effect on boys who did not result in normal childbearing and/or who died shortly after birth and therefore were not recorded in the DHS data.[32]

[Here, Table 6]

[31] Based on Rogerson et al. (2003) (p. 1372), for example, the parity-specific pattern was more evident for microscopically detectable infection with *P.falciparum*, compared to that detected by histology.

[32] Related to the second point, this study assessed whether changes in the mean values of several variables reported in Table 1 relevant to children conceived before and after the conflict were statistically equal between areas of high and low malaria transmission; the DID estimates are reported in Table A.2. While the estimate is statistically insignificant, children in malaria-endemic areas tend to be girls as a result of the war. This finding may be consistent with the under-reporting problem of male births and their immediate deaths. Alternatively, unfavorable conditions in wartime might have increased the likelihood that females were born due to the biological process of sex determination at conception (Trivers and Willard (1973)) or lowered frequency of sexual intercourse (e.g., James (1971)).

5.5 Long-term selection on adult health

Exploring long-term health consequences for respondents who were conceived in malaria-endemic areas after the outbreak of the war may help in further assessing the magnitude of the mortality effect. This is because in the long term, the immediate mortality effect may make it possible for only those who were genetically strong or in good health at conception and/or during their maturation process to survive until the present, thus exerting a *positive* relationship between in-utero exposure to malaria infection risk and present health status.

To investigate this possibility, this study utilized the 2013 DHS data. The data set includes extensive information on the respondents conceived after the outbreak of the war (approximately 33% of the 2013 sample) because the DHS's target population is women aged 15—49 years. By focusing on female respondents as a unit of analysis (rather than children born to those respondents, as exploited in the previous estimations) and by replacing the outcome of specification (1) with the respondents' height-for-age (z-scores) at the point of the survey, similar DID estimations were performed in Table 7. The height-for-age is a measure frequently exploited to detect chronic malnutrition or *stunting*. These exercises were conducted for a sub-sample of female respondents (i.e., respondents who delivered children in the five years preceding the survey) because in the DHS, the information on height-for-age was collected only for that particular sample.

Since the parental characteristics of the interviewed women cannot be discerned from the DHS data, these exercises alternatively exploited several controls evaluated at the point when the respondents were in childhood. Therefore, the estimations controlled for the number of respondents' siblings who had passed away, those still living when the respondents were 10 years old, and the respondents' birth order.[33]

Note that not all these mothers might have conceived the DHS respondents in the surveyed community. If the measure of malaria endemicity is completely noise for this reason, the DID estimations on the respondents' present health outcomes would not reveal any meaningful results. However, the estimation results reported below make this concern less critical.

[33]The number of deceased siblings is included in line with the assumption that the mortality information may positively correlate with the respondents' poverty status in childhood. Given the mortality information, the number of existing siblings is expected to measure the respondent's parental household's financial capacity to raise the respondent and his or her siblings.

5.5.1 Parallel trend assumption

As before, the DID approach needs to assume that areas of high and low malaria endemicity experienced a similar trend in health outcomes. Figure 6 demonstrates the respondents' mean height-for-age according to their year of conception. The most recent year of conception observed in the data was 1997, and the vertical line in the figure indicates the beginning of the first war in December 1989.

Two findings deserve mentioning. First, the figure reveals a similar trend of adult health between the two areas having different degrees of malaria endemicity while supporting the identification assumption (see also Appendix B for a more formal test of the pre-war parallel trends and the results).

Second, while the height-for-age is higher in low endemic areas than in areas of high endemicity before the war,[34] the pattern was reversed once the war started. This increase in height-for-age in malaria-prone areas following the outbreak of the war is expected to represent the aforementioned selection effect. In other words, a wartime increase in malaria infection risk made it possible for only those who were healthy in childhood to grow into adulthood (while discouraging the present survival of weak infants).

5.5.2 Suggestive evidence for culling of the weakest infants

Table 7 reports the DID estimation results on the respondents' height-for-age and height (cm) in columns (a)—(c) and (d)—(f), respectively. For each outcome, the exercise in the first column exploits 10 categorical variables indicating malaria intensity in contrast to the indicator for the upper 50% quantile used in the second column and the continuous measure used in the third column. Based on the results, wartime pregnancy in malaria-endemic areas has positive correlations with health-related outcomes pertaining to adult women. This finding is robust to the inclusion of numerous geographic and climate controls (see Appendix D.2.1).

Notably, because it is possible that the negative influence of maternal malaria infection on fetal growth (e.g., low birthweight) has accumulating consequences for adult health, the DID approach only identifies net effects of this (negative) and the aforementioned (positive) selection effects. Given these two

[34]This observation is not inconsistent with the finding in Figure 2 that before the war, the mortality rate was higher in areas of low malaria endemicity compared to that in areas of high transmission. Due to the great likelihood of infant mortality, it is expected that only healthy children could survive in low endemic areas through the present. It is thus possible that the height-for-age in adulthood becomes higher in low endemic areas than that in the disease-prone areas.

possible conflicting forces, the apparent long-term health improvement may highlight the significance of the immediate mortality effects. Moreover, it is also difficult to interpret whether this positive coefficient can be entirely attributed to the post-conflict policy effort made to improve access to basic health services because the service quality is lower in malaria-prone rural areas compared to urban cities. Based on a nationwide household survey in 2008, for example, more than two-thirds of rural households need more than one hour to reach the nearest health care facility, compared to the 41% country-wide average (Lee et al. (2011)).

This better health might have facilitated learning during school-attending years (e.g., Almond et al. (2009); Field et al. (2009); Kelly (2011)). Based on the relevant analyses provided in Appendix D.1, the relationship of wartime malaria infection risk in pregnancy to subsequent educational attainment is less conclusive compared to that found for health-related outcomes.

[Here, Table 7 and Figure 6]

5.6 Assessment of bias attributed to unobservables

In subsection 3.2.2, this study mentioned that the mortality effect identified in the DID approach would be attenuated. Similarly, due to the long-term positive selection effect discussed in subsection 5.5, the true health effects of wartime pregnancy in malaria-endemic areas on adult health might have exhibited an upwards bias in the DID approach.

By taking three different approaches and reporting the relevant empirical findings in Table 8, this subsection attempts to provide evidence supporting these views, i.e., that such bias exists. For the purpose of comparison to the results obtained from these approaches, the DID estimates reported in columns (b)—(c) in Table 2 and in columns (b)—(c) and (e)—(f) in Table 7 are copied and pasted to panel (A) in Table 8.

5.6.1 Oster (2015)'s δ

First, this study evaluated the relative importance of omitted variables that share covariance properties with observed controls and that are required to explain the identified estimates. Following Altonji et al.

(2005), Oster (2015) developed a way of calculating this importance, denoted as δ (i.e., a coefficient of proportionality on selection assumptions). The $\delta > 1$ means that the unobservables are more important than the observables. A negative δ means that including the unobserved controls in regressions increases the magnitude of the estimated effect rather than absorbing the effect size.

This approach needs to assume the value of R-squared obtained from a hypothetical regression of the outcome on the treatment, observed, and unobserved controls, denoted as R_{max}. Three values of R_{max} were attempted in this study. Referring to the value of R-squared arising from a regression on the treatment and observed controls as $\tilde{R}$, Oster (2015) heuristically suggested $R_{max} = 1.3\tilde{R}$. Based on the R-squared of the OLS estimation results reported in panel (A), accordingly, this study first used 0.11, 0.17, and 0.18 as the R_{max} for the mortality outcome, height-for-age, and height, respectively, in row (1) in panel (B) in Table 8. In theory, the value of R_{max} should reflect how much including both the observed and unobserved controls (that have a proportional selection relationship) and the treatment explains the variation in the outcome. Regarding the mortality consequence, one such value of R-squared may be obtained from the estimations controlling for mother-fixed effects. Referring to the value of R-squared in the estimation of column (f) in Table 2 that included the fixed effects, this study alternatively exploited $R_{max} = 0.3$ as the second possibility in row (2) in panel (B) in Table 8. In row (3) in panel (B), finally, $R_{max} = 1$ is also used as the least conservative value.

In all the cases reported in panel (B), the values of δ are negative, thus suggesting that the DID estimates reported in panel (A) are attenuated. The negative ratio for adult health outcomes may appear somewhat at odds with the expectation that the long-term influence on adult health is biased upwards due to the positive selection effect. However, the negative ratio here solely implies that the positive selection effect is underestimated in panel (A) (while underestimating the immediate mortality effects).

5.6.2 Instrumental variable approach

By exploiting a climate-based instrumental variable for malaria endemicity and conducting two-stage least-squares (2SLS) estimations, this study assessed the possible direction of bias in the OLS estimates.

It is widely accepted that temperature and rainfall are two critical factors in explaining malaria

endemicity (e.g., Blanford et al. (2013)). As far as the range of the mean temperature recorded in the data set used in this study is concerned, a higher temperature is expected to increase the potential for malaria transmission (Craig et al. (1999)).[35] However, a higher temperature does not necessarily ensure mosquito survival in regions having low precipitation because mosquitoes cannot breed in such areas (Craig et al. (1999)). Therefore, a complementarity exists between temperature and rainfall, playing an important role in malaria transmission. Accordingly, this study exploited an interaction term between these two factors (which are also interacted with w_{ikj}^{st}) as an instrumental variable for $w_{ikj}^{st} \cdot m_j$. To give more credit to the assumption of the exclusion restriction of this instrument, this study added levels of temperature and precipitation (interacted with w_{ikj}^{st}) in regressors but not in the excluded instruments.

The estimation results of the mortality effects are reported in columns (3a) and (3b) in Table 8. The coefficient on the instrument and the F-statistics in the first-stage estimations are reported at the bottom of Table A.8, which provides full results of the second-stage estimations.[36] Based on the results, the instrument is strong[37] and this instrumental variable approach identified greater mortality effects than the OLS estimates while suggesting that the latter estimates are attenuated.

The same instrumental variable approach was applied to adult health outcomes in columns (3c)—(3f). It is expected that the valid instrument removes upward bias of the OLS estimates associated with the war-induced culling of vulnerable infants. The results provided evidence (although statistically insignificant) suggesting long-term unfavorable health consequences of wartime malaria infection risk in pregnancy.

5.6.3 Discontinuity sample

The third approach exploits the insight from a regression discontinuity design. Assuming that the influence of any confounding factors must have been nearly identical for those conceived just before and after the outbreak of the war, this study applied the DID approach for sub-samples of those conceived in 1989

[35] In general, there are lower and upper limits for temperatures suitable for malaria transmission.

[36] Unfortunately, the standard errors were not corrected to allow for intra-community correlation due to computational difficulty in these estimations.

[37] However, the first-stage estimation results revealed a strong negative correlation between the instrumental variable and $w_{ikj}^{st} \cdot m_j$, which is a counter-intuitive finding. Similar to the study Alsan (2015) conducted in the context of the Tsetse fly, this finding may suggest the importance of the *non-linear* interaction between temperature and rainfall. Alternatively, the finding may stem from the fact that in Liberia, malaria is more prevalent in mountainous areas of lower temperature and precipitation.

and 1990 (given that the first war began in December 1989).

The estimation results on the mortality effect are reported in columns (4a) and (4b) in Table 8. The estimates are greater than the OLS estimates exploiting the full sample, and they reveal a great similarity to the local effect identified in the 2SLS estimations in columns (3a) and (3b). On the other hand, exploiting discrete and continuous measures of malaria endemicity yielded inconsistent estimates for adult health outcomes. These estimates might have suffered from imprecise estimation due to the small sample size. The relevant full estimation results are reported in Table A.9.

[Here, Table 8]

6 Conclusion

In what ways does an armed conflict interrupt health accumulation? To provide one answer for this question, this study investigated whether the Liberian civil war increased infant mortality by exposing pregnant mothers to a higher risk of malaria infection, thus disrupting fetal growth. Three existing frameworks encouraged the current research to address this question. First, conflict is known to greatly increase the risk of malaria-related mortality by increasing infection risk and impairing health-service deliveries. Second, pregnant women are one of the groups at the highest risk of malaria infection. Third, maternal malaria infection tends to result in adverse perinatal outcomes.

By comparing changes in mortality for children conceived before and after the outbreak of the Liberian civil war between high and low malaria endemic areas, this study found that a one-percent increase in maternal infection risk induced by the conflict resulted in a 0.44-percent increase in one-year infant mortality. This elasticity is potentially a lower-bound effect of malaria infection risk, and it could be used as one reference point for future research exploring the influence of such risk on infant mortality.

This negative health effect gradually increased and became more statistically significant as maternal passive immunity waned. In addition, the adverse health consequences were more serious for infants conceived in rainy seasons by young mothers residing in rural, battle-intense areas. On the other hand, a significant gender difference was not observed for the mortality effect. The importance of this passive immunity and the influence on infants conceived by geographically, seasonally, and immunologically high-

risk mothers may help policymakers and practitioners determine target groups for aid programs during inter- and post-war periods.

The current study also showed that in-utero exposure to malaria infection risk had a positive correlation with adult health as measured by height-for-age and height. Assuming that the possible wartime culling of the weakest infants enables only those who are genetically strong and/or protected during their childhood to reach adulthood, this apparent long-term human-capital improvement may highlight the significance of the immediate mortality effect. This selection effect is potentially inherent in any research exploring the long-term consequences of armed conflict on survivors' health based on post-conflict data, and it likely leads to underestimation of the adverse health impacts.

This study concludes by providing a few reservations with respect to the findings reported here. First, this study evaluated the *total effect* arising from an increase in malaria infection risk in pregnancy *and* impairment of the health system that aggravated unfavorable consequences of the heightened infection risk during the war. If available, exploiting information on wartime access to health services would have been useful for disaggregating the pathways. Second, this study only identified the wars' influence on children conceived by mothers originally residing in malaria-endemic areas. Given the sizeable war-induced diaspora, this research may not necessarily capture the influence of all the malaria infection risk faced by pregnant mothers during the years of the conflict. Related to this point, this study was not able to explore whether and how the war and people's behavioral responses encouraged the spread of the malaria parasites across spaces. Relevant studies addressing this spillover effect may need to consider people's immunity and infection risk.

A Appendix: Literature review

A.1 Liberia and malaria suitability

Together with the tropical heat and humidity, Liberia lies almost entirely within the rainforest zone. The landscape is characterized by swampy plains along the coast and inland rolling plateaus leading to low mountains. Most areas lie below an altitude of 500 m, which makes malaria transmission possible in almost all parts of this country because malaria infection does not typically occur above an altitude of approximately 1,500 m (Stanley C. Oaks et al. (1991), p. 219). Throughout the year, the monthly average temperature is also higher than the minimum temperature (approximately 18 °C) required for parasite development of *P.falciparum*, a major plasmodium species in this country (Patz and Olson (2006)). While the body's inflammatory response is a typical malaria symptom, this most dangerous species also causes cytoadherence of erythrocytes to vascular walls; thus, infected blood cells are often trapped in small blood vessels. This can result in end-organ damage or blood abnormalities. In addition, microcytic anemia in adults and folic acid deficiency also commonly occur due to the splenic sequestration of the parasites (e.g., Schantz-Dunn and Nour (2009)).

A.2 Liberian civil war

In Liberia, while the True Whig Party, founded in 1869 by Americo-Liberians (i.e., former slaves liberated from the United States) has taken a monopolistic position on the political system since the late 19th century, a bloody military coup led by Samuel Doe overthrew the government in 1980. Although he attempted to legitimize his rule, which gave preferential treatment to his own ethnic group, the Krahns, corruption and brutality (e.g., crackdowns on the Gio and Mano ethnic groups) during his 10 years in power unavoidably induced the subsequent uprising against him, followed by Liberia's descent into the political abyss during the last decade of the 20th century (Zeleza and Eyoh (2003), p. 327).

Liberia's political unrest encompassed two civil wars between 1989 and 2003. The first war erupted in December 1989, when an armed group, the National Patriotic Front of Liberia (NPFL), attacked the border town of Butuo in north-central Nimba county from Côte d'Ivoire under the leadership of Charles Taylor. This war was brought to an end in July 1997 when national elections were held and Taylor

was elected president. During this war, an ethnic pattern of killings based on corresponding factional groups, which fought to control the rich natural resources of this country (e.g., diamonds, gold, iron ore, and timber), emerged and set a tone for the conflict that lasted for 14 years. A second war began in April 1999, when a Guinean-backed rebel group, the Liberians United for Reconciliation and Democracy (LURD), sparked a revolt in northwest Lofa County. This war continued until August 2003, when the warring parties signed the Accra Comprehensive Peace Agreement. During the 14-year state of chaos and fear, more than 250,000 soldiers and civilians were killed, i.e., approximately more than 12% of the 2.1 million population in 1990 (Mama (2014), p. 55).[38]

Due to the atrocious, indiscriminate nature of the fighting and human rights abuses (e.g., killings, looting, property destruction, rape, and child recruitment), it is estimated that approximately 500,000 people were internally displaced and another 780,000 sought refuge abroad.[39] Detailed accounts of egregious human rights abuses and displacement can be found in Foster et al. (2009), a document based on more than 1,600 statements by war survivors (see also Nilsson (2003) for an excellent literature review of the internally displaced persons, refugees, and returnees).

The displacement typically proceeded through phases of hiding, internal displacement within Liberia, and refuge in other countries. In response to the sound of gunfire, government warnings or rumors of an assault, and surprise attacks, fleeing populations often hid in swamps, bushes, and mountains for days, weeks, or even months. Rebel groups also exploited the forced displacement as a military tactic to clear areas for their occupation (Foster et al. (2009), p. 131). It is argued that women more disproportionately suffered from difficulties stemming from the displacement compared to men and boys, who were often forced to join various fighting forces (Dabo (2012)). This cycle of fighting, hiding, and relocation was also repeated during the long years of the conflict.

Most internally displaced persons (IDPs) headed towards Monrovia, the capital (Nilsson (2003)). IDPs often took shelter in public buildings and other available spaces (e.g., church compounds, embassy compounds, sports stadiums, and university campuses) in overcrowded, impoverished conditions. After

[38]Information on the total population is sourced from "World Population Prospects: The 2012 Revision" (`http://esa.un.org/wpp/unpp/panel_population.htm`).

[39]Information pertaining to the externally and internally displaced persons is drawn from the "2005 UNHCR Statistical Year Book" (`http://www.unhcr.org/464478a72.html`) and "Liberia: Internal displacement in brief" as of December 2013, Internal Displacement Monitoring Center (IDMC) (`http://www.internal-displacement.org/sub-saharan-africa/liberia/summary`), respectively.

the signing of the Accra Peace Agreement in 2003, most IDPs relocated to formal camps located close to Monrovia and along the main road in Bong County. The World Food Programme (WFP) registered approximately 320,000 Liberians at these camps (see Jesuit Refugee Service (2007) for a good review of issues relevant to IDPs).

At the beginning of the war, refugees successfully settled in asylum countries, and they were economically integrated into their host communities (e.g., Dick (2002); Leach (1992); Kuhlman (2002)). Based on the research Damme (1999) conducted in the Forest Region of Guinea, for example, less than 20 percent of the refugees were residing in camps in 1996. As refugees came to outnumber their hosts, however, anti-refugee sentiment became visible, sometimes forcing refugees to return to Liberia even though it remained unsafe (Lawrie and Damme (2003)). As the war persisted, refugees often arrived suffering from exhaustion because they tended to choose the option of leaving the country only after enduring several years of internal displacement and the associated hardships (Damme (1999)).

Many factors (e.g., limited access to education and health services, severance of family and community links, concerns of gender-based violence, and absence of job opportunities) have discouraged IDPs and refugees from returning to their places of origin after the war ended (e.g., Wright and Savage (2007)). Consequently, it took more than three years for the majority of the IDPs to return to their homes or settle elsewhere following the 2003 Peace Agreement.[40] In addition, the UN refugee agency completed a repatriation program in 2012 that facilitated the return of more than 155,000 refugees by the end of that year (Momodu (2013)). It is also reported that those returnees sometimes went through daunting hardships when resettling in Liberia (e.g., Jesuit Refugee Service (2007); Omata (2012)).

A.3 Factors affecting infant mortality

Based on a recent estimate, approximately 44% of deaths in children under the age of five years occur within the first four weeks following birth (Liu et al. (2015)).[41] Public health research has reported the

[40]The IDP camps were officially closed in April 2006, although this does not necessarily mean all the IDPs had returned to their places of origin by this time.

[41]The corresponding ratio was 37% in 1990; thus, an increasing proportion of under-five deaths is now attributed to newborn deaths (e.g., Lawn et al. (2005); Lawn et al. (2010)). With 32 deaths per 1,000 live births estimated in 2012, the neonatal mortality rate (NMR) in sub-Saharan Africa is the highest in major developing areas, and this region has also seen the lowest (28%) reduction in NMRs from 1990 to 2012 (UNICEF (2013), p. 13). Explicitly referring to Liberia, Lawn et al. (2005) also noted that the high incidence of neonatal deaths in Africa is particularly seen in countries that experienced violent political turmoil.

following direct causes of neonatal mortality (excluding stillbirths) worldwide: severe infections (e.g., sepsis, pneumonia, tetanus, and diarrhea) (29%), preterm births (i.e., delivery at less than 37 weeks of completed gestation) (29%), asphyxia (23%), and congenital abnormality (8%) (Lawn et al. (2010)).

Pneumonia, diarrhea, and malaria also constitute approximately 23%, 16%, and 13% of the remaining proportion (i.e., 56% = 100% − 44%) of under-five mortality, respectively (Liu et al. (2015)). In Profile 2 countries (e.g., Guinea and Sierra Leone) characterized by Black et al. (2003), it is also estimated that pneumonia, diarrhea, malaria, and neonatal deaths each account for approximately 20—26% of deaths in children younger than five years.

Low birthweight (i.e., a weight at birth less than 2,500 g) is also an important secondary factor that affects neonatal and infant deaths. The short gestation and intrauterine growth restriction (i.e., restricted growth of a fetus during pregnancy) and a combination of these factors lead to low birthweight, and approximately 60—80% of newborn deaths are cases of low-birthweight neonates (Lawn et al. (2005)).

Because these factors are closely inter-related, it is difficult to identify a single reason for each neonatal and infant death. For example, the incidence of preterm births increases that newborn's difficulty in feeding and maintaining his or her body temperature while raising the risk of contracting infectious diseases (Lawn et al. (2005); Lawn et al. (2010)). Prematurity and in-utero growth failure or disruption also raise the risk of neonatal deaths especially attributed to infection (Lawn et al. (2005)). Many infants recorded as dying from infectious diseases are also premature (Lawn et al. (2010)).

A.4 Pathological pathways from maternal malaria infection to fetal growth retardation

Umbers et al. (2011) provided a good pathological review on the underlying factors that link placental malaria with fetal growth restriction. For instance, placental infection is likely to impair placental development while prompting maternal hypertension and placental vascular dysfunction. In addition, placental inflammation attenuates levels of hormones that regulate placental functions. Of those hormones, some affect fetal cell proliferation, maternal appetite, metabolism, and fat accumulation. Moreover, a high density of parasites in placental blood and the associated immune response may decrease levels of nutrients (e.g., amino acids, lipids, and glucose) delivered to the fetus. Similarly, the transplacental transfer of maternal antibodies and cellular immune responses to several infectious diseases (e.g., measles, tetanus)

can also be prevented by placental infection (e.g., Brair et al. (1994); de Moraes-Pinto et al. (1998)). Placental changes such as thickening of cytrophoblastic membranes may also hinder the effectiveness of nutrient transporters (e.g., Guyatt and Snow (2004)).

A.5 Seasonal influence of maternal malaria infection on fetal growth

Analyses in the fifth and sixth columns in Table 5 showed that wartime pregnancy in malaria-endemic areas raised infant mortality if conception occurred in the rainy season. While this finding may suggest a higher infection risk in malaria-prone seasons and associated unfavorable birth outcomes, some caution is needed in asserting such a link because the seasonal influence of maternal malaria infection on fetal growth could be affected by multiple factors, a possibility supported by the studies reviewed in this section.

In Africa, birthweights are likely to exhibit seasonal fluctuations (e.g., Rayco-Solon et al. (2005)). While maternal malaria infection may be just one factor that underlies this observation, it could possibly have a seasonal influence on fetal growth for three reasons. First, some gestational months may biologically reveal a higher risk of contracting the disease than in the remaining months. Second, even if the inherent risk of infection is identical throughout the gestation period, epidemic-prone seasons may also exist. Third, the magnitude of the adverse influence on fetal growth may also vary by gestational months of infection.

Based on the review of epidemiological literature made by Desai et al. (2007) and others (e.g., Brabin (1983); Rogerson et al. (2000); Walker et al. (2014); Zhou et al. (2002)), it appears that the infection risk is highest during the second trimester. The peak prevalence in this period may also be attributed to an increasing susceptibility in the first trimester (e.g., Cohee et al. (2014); Dicko et al. (2003)).

Typically, malaria transmission positively correlates with relatively predictable patterns of precipitation, although the infection risk may continue at lower levels during the dry season (Stanley C. Oaks et al. (1991), pp. 217-218). While malaria infection is possible throughout the year in Liberia due to its climatically suitable nature for the vector-mediated transmission, this seasonal pattern seems to remain even in this country. *Anopheles gambiae*, a principal anopheles species in Liberia (WHO (2013), p. 149),

is most abundant during the rainy seasons in this country (Fahmy et al. (2015); Gelfand (1955); Hogh et al. (1993); Somah (2005)).

Compared to the health consequences of malaria in pregnancy in general, it is relatively unknown which gestational period of infection has the largest unfavorable effects (e.g., Cottrell et al. (2007); Kalilani et al. (2010); Huynh et al. (2011)). As Desai et al. (2007) pointed out, intrauterine growth restriction and low birthweight may be attributed to infection in the second and third trimesters, whereas low birthweight and preterm delivery may result from infection in the third trimester. While the adverse effects of infection in the first trimester are relatively unclear, it may also explain the decrease in the birthweight of newborns (e.g., Huynh et al. (2011)).

B Appendix: Parallel trend assumption

The key identification assumption of the DID approach is a parallel trend of the outcomes. While Figure 2 (infant mortality) and Figure 6 (height-for-age) provide an informal assessment of and support for the assumption, this subsection statistically tests whether the pre-war trend of the outcomes significantly varied with malaria endemicity. To achieve this purpose, this study restricted its attention to data pertaining to persons conceived in or before 1989 (because the war began in December 1989) and estimated the following equation for the outcomes y_{ij} of individual i in community j,

$$y_{ij} = \beta_1 + \beta_2^h \sum_h a_{ij}^h + \beta_3^h \sum_h a_{ij}^h \cdot m_j + v_j + u_{ij}, \tag{B.1}$$

where a_{ij}^h is an indicator equal to one if the person was conceived in year h and zero otherwise (the reference group was all those conceived in and before 1980); v_j is community-level fixed effects; and u_{ij} is a stochastic error. The unit of analysis is either children born to the DHS respondents or the DHS respondents themselves, depending upon the outcomes of interest. The parallel trend is consistent with the estimated β_3^h, which is insignificantly different from zero. The estimation results of the one-year mortality of children born to the DHS respondents, the respondents' height-for-age, height, and education were presented in columns (a) through (d) in Table A.1, respectively. As is clear from the table, these

exercises provided no evidence undermining the assumption of parallel trends in the pre-war periods.

C Appendix: Robustness checks

C.1 Non-linear empirical models

As shown in Table A.3, this study attempted to determine whether the findings provided in Table 2 were robust to non-linear empirical models (and controls). First, this study separated the sample into two groups, namely children conceived before and after the outbreak of the conflict, and independently estimated the influence of maternal malaria infection risk based on a probit model. The sample separation facilitates a straightforward calculation of the marginal effects reported in columns (a)—(g) and (i)—(o) in Table A.3, respectively (Ai and Norton (2003)). As seen in the table, several survival periods were investigated by varying the value of Z from one (i.e., neonatal mortality) to 60 (i.e., five-year infant mortality). To avoid computational obstacles resulting from estimating non-linear models with numerous indicator coefficients as well as the potential incidental parameter problem that biases the estimates and the associated standard errors (e.g., Greene (2004); Lancaster (2000)), the probit estimations replaced the community-fixed effects with county-level fixed effects (16 groups) while including a community's geographical position (latitude, longitude) and elevation in meters.[42]

As the results show, the malaria infection risk in pregnancy exhibited statistically positive relationships with child mortality only after the war's outbreak. The estimates on malaria endemicity and the corresponding statistical significance also gradually increased as the exercises examined longer survival periods. These findings are consistent with those provided by Table 2 and Figure 4.

By exploiting a Cox proportional hazard model, this study also analyzed the duration (months) from birth to death in columns (h) and (p) in Table A.3, whereby the estimated hazard ratio is reported and a ratio greater (smaller) than one means that the variable induces (prevents) mortality.[43] Due to the proportional hazard assumption, the ratio should be interpreted as the hazard ratio at any point in time

[42]Similar to the identification of a district corresponding to each DHS community (see footnote 21), the communities were categorized into 15 counties plus one group for which the ArcGIS failed to identify the corresponding county. The estimations in Table A.3 included the unidentified group as one county that corresponded only to 3% of the entire sample.

[43]Unlike the previous estimations that exploited data pertaining to children born more than Z months before the timing of the DHS interview, this survival analysis used all the available observations.

between two individuals that only varies by one unit of covariates. Schoenfeld residual (p-values) reported at the bottom of Table A.3 failed to reject the assumption of proportional hazards. The results again highlight that malaria infection risk in pregnancy decreases the survival likelihood of children conceived only after the outbreak of the war (with 5% statistical significance).

C.2 Alternative measures of malaria infection risk

This study collected estimates of endemicity levels of *P.falciparum* in each year between 2000 and 2010 from another study of the Malaria Atlas Project, Bhatt et al. (2015).[44] Despite a similar Bayesian geostatistical model and the map resolution exploited by Gething et al. (2011) and Bhatt et al. (2015), these two studies provided different estimates in 2010. In the data set used in this study, for example, the community-level mean 2010 endemicity was 0.41 based on Gething et al. (2011) and 0.31 based on Bhatt et al. (2015), respectively. In part, this difference is attributed to differences in a geographical scope of these two studies as well as the timing of the *Plasmodium falciparum* parasite rate (PfPR) surveys that input data of the parasite rate into the geostatistical model. For instance, Bhatt et al. (2015) considered the PfPR surveys taking place between 1995 and 2014 in Africa, and Gething et al. (2011) was based on PfPR surveys conducted between 1985 and 2010 primarily in (but not limited to) Africa.

Due to these differences and a great difficulty in estimating endemicity in general, this study avoids a hasty judgment about which of these (or other possible) studies provides more precise estimates of endemicity.[45] Instead, these alternative estimates were exploited to check the robustness of the main findings. Accordingly, this study estimated the specification (1) based on endemicity estimates provided by Bhatt et al. (2015) and reported the estimated α_2 in Table A.10. In each panel from (A) to (K), endemicity levels sourced from different years were exploited for the estimations.

As seen in the first two columns, this study confirmed that the wartime conception in malaria-endemic areas increased the likelihood of infant mortality. In addition, the estimates are almost identical across panels. This finding supports the discussion in subsection 3.1.2 that the endemicity levels of *P.falciparum* have not markedly changed between 2000 and 2010 in Liberia despite a global malaria control effort.

[44] The data are available from `http://www.map.ox.ac.uk/`.

[45] Relatedly, similar to the endemicity estimates of Gething et al. (2011), those based on Bhatt et al. (2015) should also be seen as just another proxy for the pre-war levels of endemicity in the present study.

Regressing Bhatt et al. (2015)'s endemicity estimates in 2010 on those in 2000 at the community-level yielded a coefficient of 0.937 (std. 0.029), which is close to one.

D Appendix: Long-term selection on adult human capital stocks

D.1 Selection effects on education

In subsection 5.5, this study showed that pre-natal exposure to malaria infection risk positively correlated with long-term health outcomes. Then, better health might also have facilitated learning during school-attending years. Based on the previous studies, unfavorable prenatal conditions appear to hinder children's cognitive development (e.g., Almond et al. (2009); Field et al. (2009); Kelly (2011)).

By applying a similar DID approach to that demonstrated in subsection 5.5, this study investigated the long-term consequences of wartime malaria infection risk in pregnancy on the respondents' completed years of education in Table A.4. In contrast to analyses of adult health outcomes reported in Table 7, the data pertaining to all the respondents in the 2013 DHS were exploited for the exercises in Table A.4 (recall that in the DHS, the information on height-for-age was collected only for respondents that delivered children in the five years preceding the survey).

Exploiting both the discrete (upper 50% quantile) and continuous measures of malaria infection risk provided evidence supporting the positive selection effect at around 10% significance level in columns (b) and (c) in Table A.4, respectively. However, this positive relationship appears to be less conclusive because, based on the estimation result in column (a) in Table A.4, which uses 10 categorical variables of malaria endemicity (the reference group is the lowest percentile), the statistically significant positive and negative effects were found for relatively lower levels of malaria endemicity. This finding may indicate that the linkage between fetal development and educational attainment may not be as straightforward during the time of the war. Following discussions presented in subsection 5.6, the results of the 2SLS estimations [columns (d)—(e)] and of the estimations exploiting data on those conceived in 1989 and 1990 [columns (f)—(g)] are also reported in the remaining columns in Table A.4.

D.2 Robustness checks

D.2.1 Inclusion of geographic and climate controls

Analyses in subsection 5.5 and Appendix D.1 showed that in-utero exposure to malaria infection risk had a positive correlation with health and (with less confidence) educational attainment in adulthood. This section checks the robustness of this finding to the inclusion of geographic and climate controls. For this purpose, this study further examined height-for-age (z-scores), height (cm), and education (years) in Table A.5, Table A.6, and Table A.7, respectively. In these tables, a similar empirical model to that in Table 7 and Table A.4 is estimated. As before, estimation results exploiting an indicator for high malaria infection risk (upper 50% quantile) and a continuous measure of malaria endemicity are reported together with the relevant coefficients. Coefficients on suppressed controls are available from the author upon request.

This study performed similar exercises to those conducted for infant mortality in subsection 5.3.3. In other words, the estimations also included interaction terms between an indicator for the respondents conceived after the outbreak of the war and several variables representing a community's climatology [columns (a)—(b)], landscape [columns (c)—(d)], soil quality [columns (e)—(f)], and GPS-based coordinates [columns (g)—(h)].

The statistical significance of the estimated coefficients is not always strong likely due to the small sample size (relative to that used in analyses of infant mortality) and the high correlation between the measures of malaria endemicity and geographic and climatic conditions. Nevertheless, these estimation results continued to confirm positive relationships between pre-natal exposure to malaria infection risk and adult human capital stocks.

D.2.2 Alternative measures of malaria infection risk

By exploiting alternative endemicity estimates provided by Bhatt et al. (2015) (see subsection C.2), this study assessed the robustness of the long-term selection effects reported in Table A.10, whereby only the relevant coefficients are reported. As seen from the estimation results in the third through eighth columns in panels (A)—(K), in-utero exposure to malaria infection risk in wartime had positive correlations with

adult human capital stocks.

E Appendix: Violence seasonality

While it is difficult to obtain precise data on the frequency of crimes committed during the time of conflict, this study assessed the seasonal pattern of violence by exploiting two independent data sources, namely the Truth and Reconciliation Commission (TRC) and (as described in Section 4) the ACLED.[46] With assistance from the Human Rights Data Analysis Group (HRDAG) and the Benetech, the TRC built an electronic database of violence events based on statements collected from victims, witnesses, and perpetrators of human rights violations during the war (see Cibelli et al. (2009) for details of descriptive statistics of the data).[47]

The TRC team has made an extensive effort to collect as many statements as possible. The statement givers provided the TRC with 17,160 testimonies, including 163,615 total violations that existed between April 1979 and December 2003 across 15 of the country's counties. As summarized in Table A.12, in the TRC data, violence is classified into 23 categories.[48] Figure A.2 reports the number of acts of sexual violence (grouping rape, gang rape, multiple rape, sexual abuse, and sexual slavery) and the total number of violent acts by year of incidence.[49] While sexual violence constitutes approximately 3—4% of the reported total human rights violations, Figure A.2 shows a similar episodic tendency between sexual and total violence, with three spikes: one in 1990 at the onset of the first civil war, one in 1994, and one in 2003 linked to attacks on Monrovia by the rebel groups.

The year of incidence was discerned from the TRC data for all human rights violations. However, of the recorded 6,534 violations relevant to sexual violence, only 1,190 observations (approximately 18%) contained information on the month of incidence. With this admitted limitation, Figure A.3 provided the details of the time trend of sexual violence (see also Figure A.4 for a similar breakdown of the total violence). Apparently, the figure provides some evidence suggesting that in wartime, sexual crimes were more intensively committed in rainy seasons (May to October).

[46] The TRC data and the relevant documents can be downloaded from `https://hrdag.org/liberia-data/`.
[47] See `https://hrdag.org/` and `http://benetech.org/` for the HRDAG and Benetech, respectively.
[48] In Table A.12, this study grouped rape, gang rape, and multiple rape into one category.
[49] Notably, not only females but also males can be victims of sexual violence; in addition, sexual abuse includes a case of stripping victims, which could be seen as a means of humiliating people during the war.

To test this finding more formally, this study constructed balanced panel data of human rights violations at the county-month level, consisting of 15 counties and 297 months from April 1979 and December 2003 (yielding 4,455 observations). By exploiting this panel data, the seasonal pattern of the human rights violations was checked in columns (a) through (f) in Table A.11. Notably, the number of human rights violations includes cases only when both the timing and location of the alleged violation are discerned from the TRC dataset; therefore, the regression analyses in Table A.11 using the fixed effects of year/month-of-incidence and each county are seriously subject to this issue (as well as a problem of selective statements explained in the next paragraph).[50] Acknowledging this limitation, the results still show that violence was more intensive in rainy seasons during the time of conflict. The analyses in columns (i) through (n) disaggregated the periods following the outbreak of the war into four periods relevant to the first war, ceasefire, second war, and post war and repeated similar exercises. The obtained implications remained unchanged.

Unavoidably, the statements reported to the TRC cannot be seen as representing the general population of human rights violations (Cibelli et al. (2009)). First, the statement givers are not randomly selected. Second, some crimes—such as sexual violence and murders—may be under-recorded unless appropriate witnesses are found and/or the victims tell the truth. Consequently, estimations using the TRC data might have failed to provide a true picture of the seasonal pattern of violence. Hence, to further the analysis of violence seasonality, this study also exploited data drawn from the ACLED.

The seasonal pattern of the total number of battle events was checked in columns (g) and (h) and, with a similar breakdown of the periods following the outbreak of the war, in columns (o) and (p) in Table A.11. Similar to the analysis using the TRC data, this study established balanced panel data consisting of 14 counties and 284 months from January 1980 to August 2003 (yielding 3,976 observations). In contrast to the TRC data, the ACLED data contain information on both the month and year of incidence for all battles; therefore, it is less likely that the analysis using the latter data suffers from the problem attributed to difficulty utilizing the representative violence data. Based on the results, it appears that the frequency of the battles was higher in rainy seasons compared to the remaining periods after the war

[50]For example, only 31,001 of the total 163,615 human rights violations (approximately 19%) included all the information on year, month, and place of incidence.

started, particularly during the time of the first war, although the statistical significance is absent.

F Appendix: Geo-coded variables

This section describes community-level geo-coded variables used in this study and the original sources.

F.1 Malaria endemicity

The data on the endemicity levels of *P.falciparum* are provided by the Malaria Atlas Project. The spatial resolution exploited in this study is between 30 seconds of a longitude/latitude degree (≈ 1 km^2). Gething et al. (2011)'s estimates are available from `http://www.map.ox.ac.uk/browse-resources/endemicity/Pf_mean/world/`. Bhatt et al. (2015)'s estimates are available from `http://www.map.ox.ac.uk/`.

F.2 Battle intensity

The data on battle intensity are provided by the Armed Conflict Location and Event Database (ACLED), available from `https://www.prio.org/Data/Armed-Conflict/Armed-Conflict-Location-and-Event-Data/`. This study calculated the number of battle events that occurred within a 25-km radius from each DHS community.

F.3 Administrative areas

A county and district corresponding to each DHS community was identified by matching a community's GPS latitude/longitude coordinate with a map of Liberia sourced from DIVA-GIS (`http://www.diva-gis.org/datadown`).

F.4 Climatology

The data on climatology is provided by "WorldClim - Global Climate Data" (Hijmans et al. (2005); `http://www.worldclim.org/current`). The spatial resolution exploited in this study is between 30 seconds of a longitude/latitude degree (≈ 1 km^2).

Temperature: average monthly temperature (multiplied by 10 °C) between 1950 and 2000

Precipitation: average monthly precipitation (mm) between 1950 and 1990

F.5 Land scape

Elevation: elevation (m) of each community based on the Shuttle Radar Topography Mission (SRTM) Digital Elevation Model (DEM) and directly available in the DHS data

Slope: slope (percent) provided by Nunn and Puga (2012) (`http://diegopuga.org/data/rugged/#grid`); the spatial resolution exploited in this study is between 30 seconds of a longitude/latitude degree ($\approx$ 1 km^2).

Terrain roughness: terrain roughness (percent) provided by Nunn and Puga (2012) (`http://diegopuga.org/data/rugged/#grid`); the spatial resolution exploited in this study is between 30 seconds of a longitude/latitude degree ($\approx$ 1 km^2).

F.6 Soil quality

The data on soil quality is provided by the "Harmonized World Soil Database" (Fisher et al. (2008); `http://webarchive.iiasa.ac.at/Research/LUC/External-World-soil-database/HTML/index.html?sb=1`). The spatial resolution exploited in this study is between 30 seconds of a longitude/latitude degree ($\approx$ 1 km^2).

Nutrient availability: an indicator for nutrient availability, which takes one if a community is characterized as having "moderate, severe, or very severe constraint" (the reference group is "no or slight constraint")

Nutrient retention capacity: an indicator for nutrient retention capacity, which takes a value of one if a community is characterized as having "moderate, severe, or very severe constraint" (the reference group is "no or slight constraint")

Rooting conditions: an indicator for rooting conditions, which takes a value of one if a community is characterized as having "moderate, severe, or very severe constraint" (the reference group is "no or slight constraint")

Oxygen availability to roots: an indicator for oxygen availability to roots, which takes a value of one

if a community is characterized as having "moderate, severe, or very severe constraint" (the reference group is "no or slight constraint")

Excess salts: an indicator for excess salts, which takes a value of one if a community is characterized as having "moderate, severe, or very severe constraint" (the reference group is "no or slight constraint")

Field-management constraint: an indicator for field-management constraint (workability), which takes a value of one if a community is characterized as having "moderate, severe, or very severe constraint" (the reference group is "no or slight constraint")

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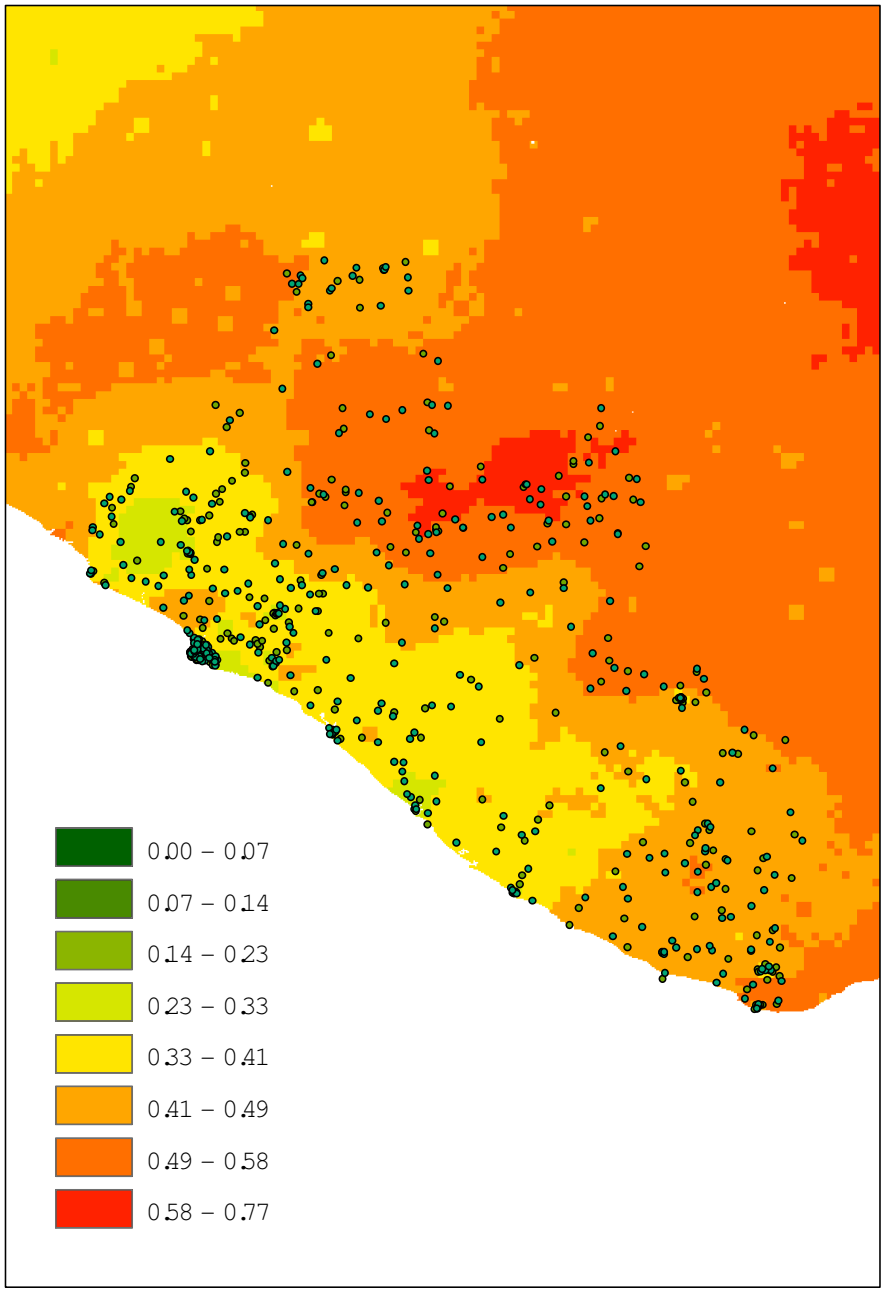

Figure 1: Malaria endemicity map (*Plasmodium falciparum*) in 2010 and locations of DHS communities

Source: Gething et al. (2011))

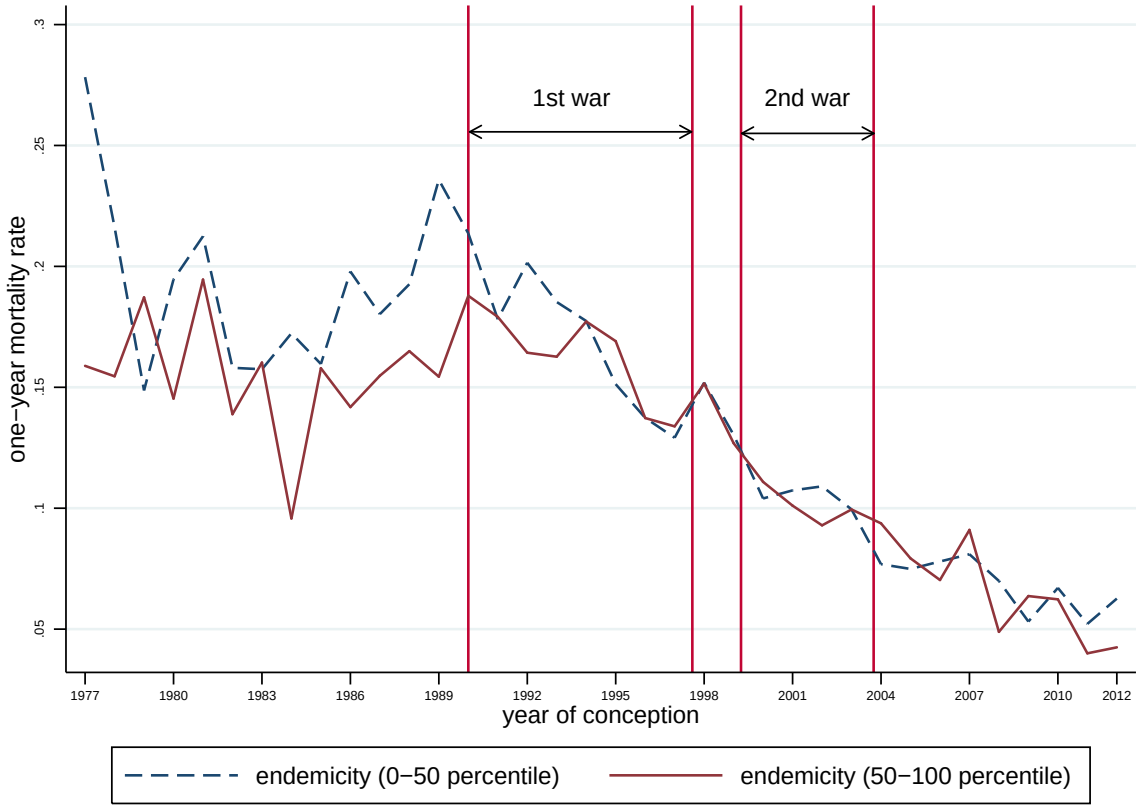

Figure 2: One-year mortality rate by the timing of conception

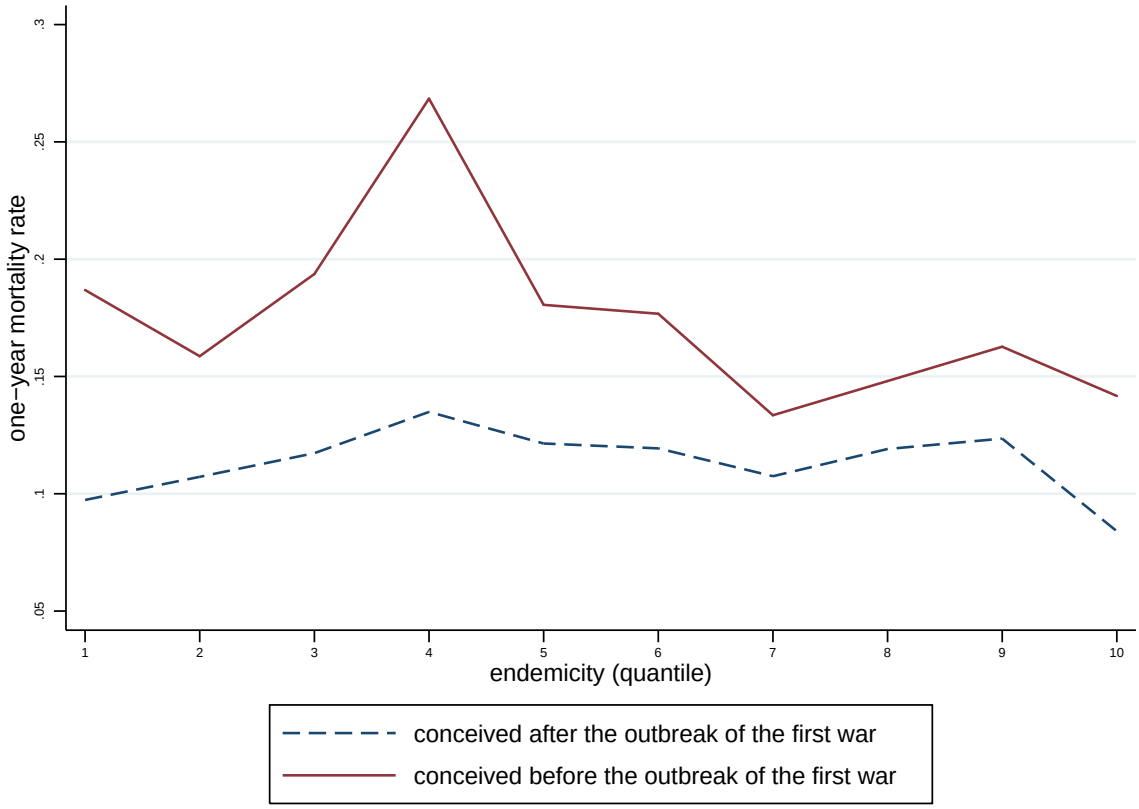

Figure 3: One-year mortality rate by levels of endemicity

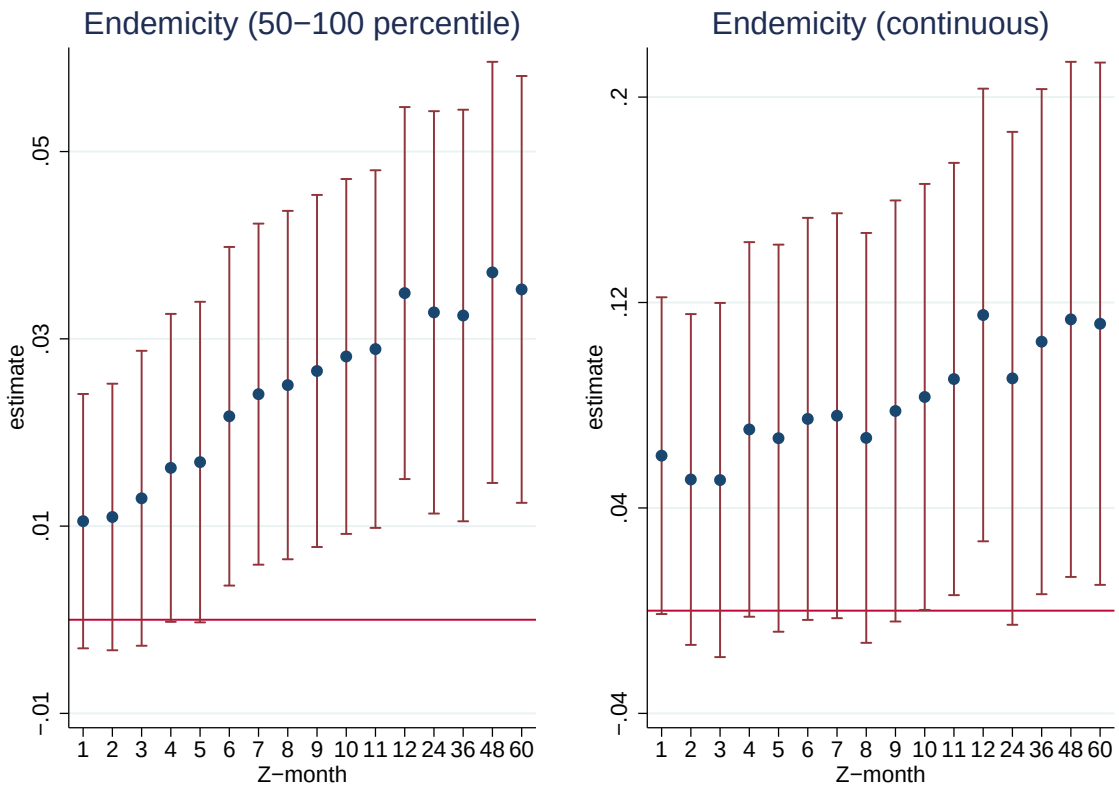

Figure 4: Impacts of wartime pregnancy in malaria endemic areas on Z-month infant mortality (LPM)

Notes: (1) This figure reports the estimated α_2 with 95% confidence intervals by varying the investigated survival periods. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community.

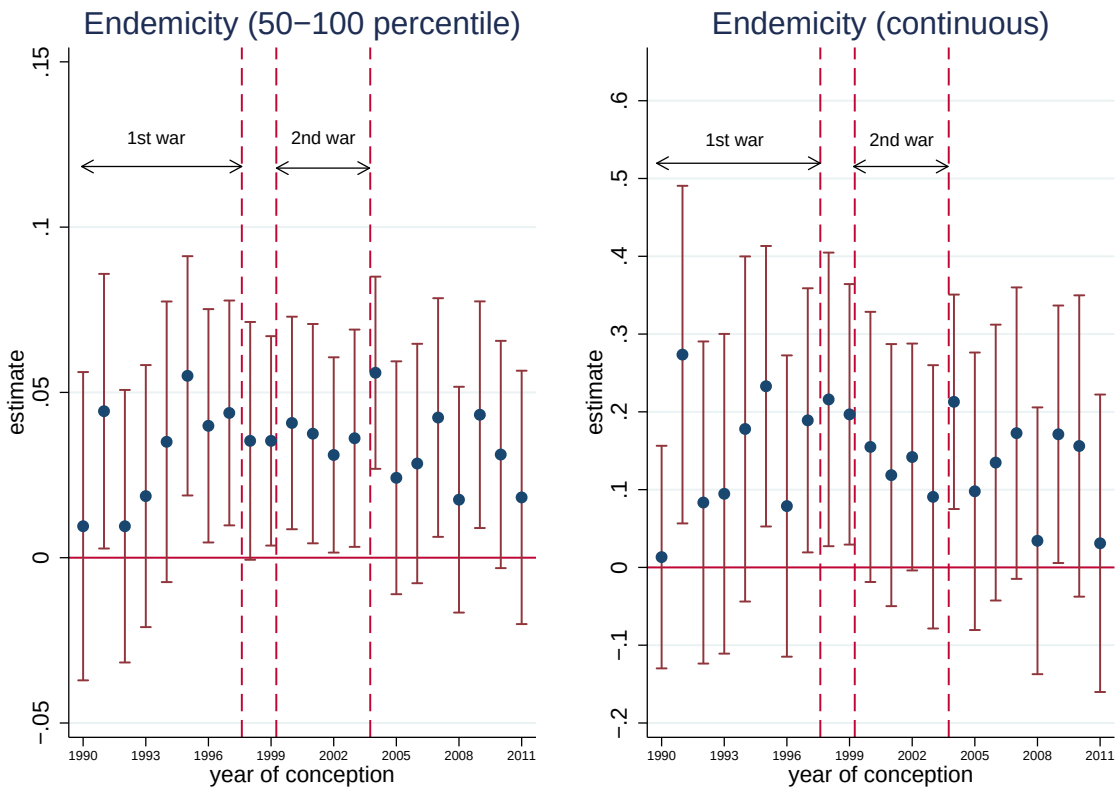

Figure 5: Impacts of wartime pregnancy in malaria endemic areas on one-year mortality by the year of conception (LPM)

Notes: (1) This figure reports the estimated α_2 specific to the year of conception following the outbreak of the first war, with 95% confidence intervals. (2) Infants conceived in December 1989 (i.e., the beginning of the first war) were included in a category of those conceived in 1990. (3) Standard errors are robust to heteroskedasticity and clustered residuals within each community.

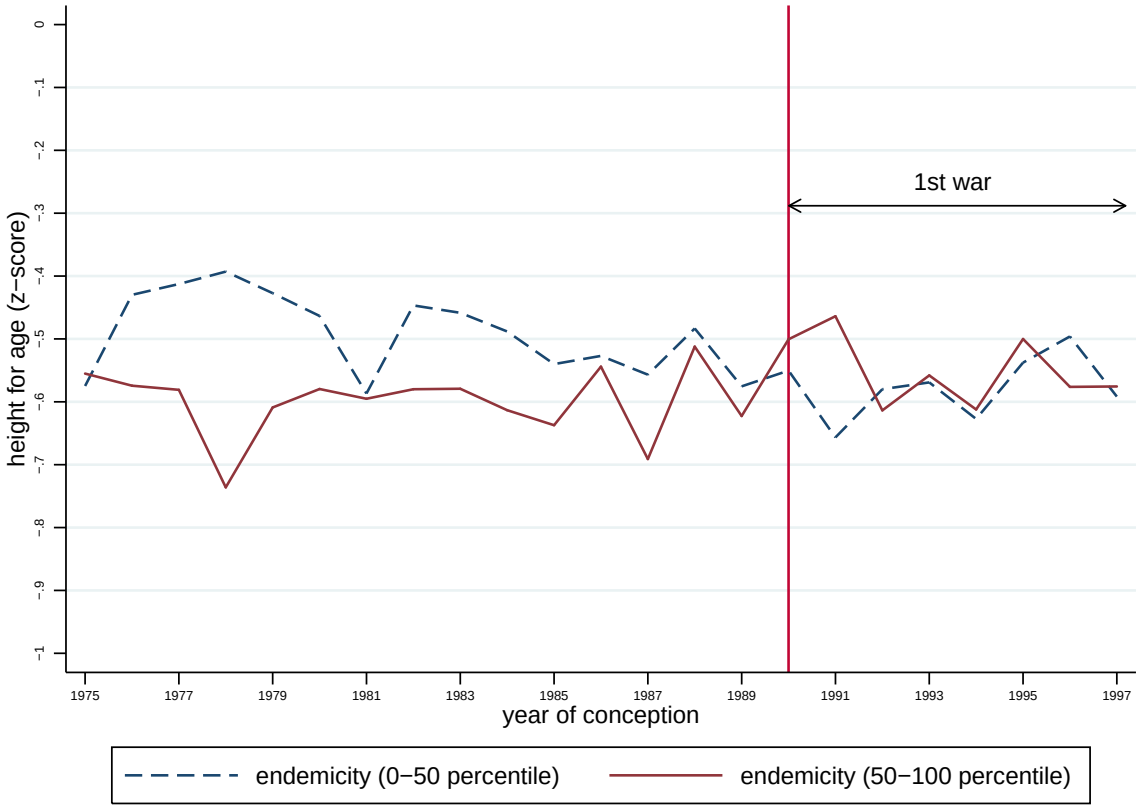

Figure 6: Height-for-age (z-score) by the timing of conception (2013 DHS only)

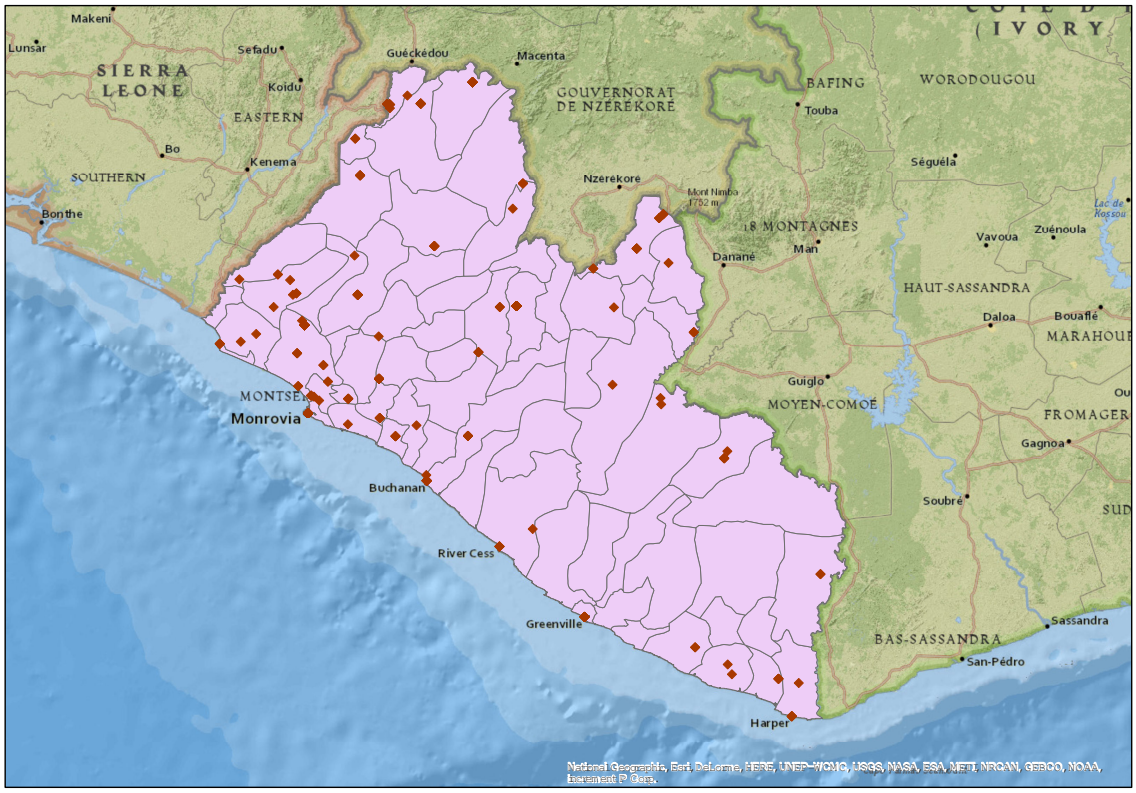

Figure A.1: Spatial distribution of battle events with district boundaries

Source: Armed Conflict Location and Event Database (ACLED)
Notes: (1) In the data set, 265 conflicts are recorded. (2) The map of Liberia is sourced from DIVA-GIS (`http://www.diva-gis.org/datadown`).

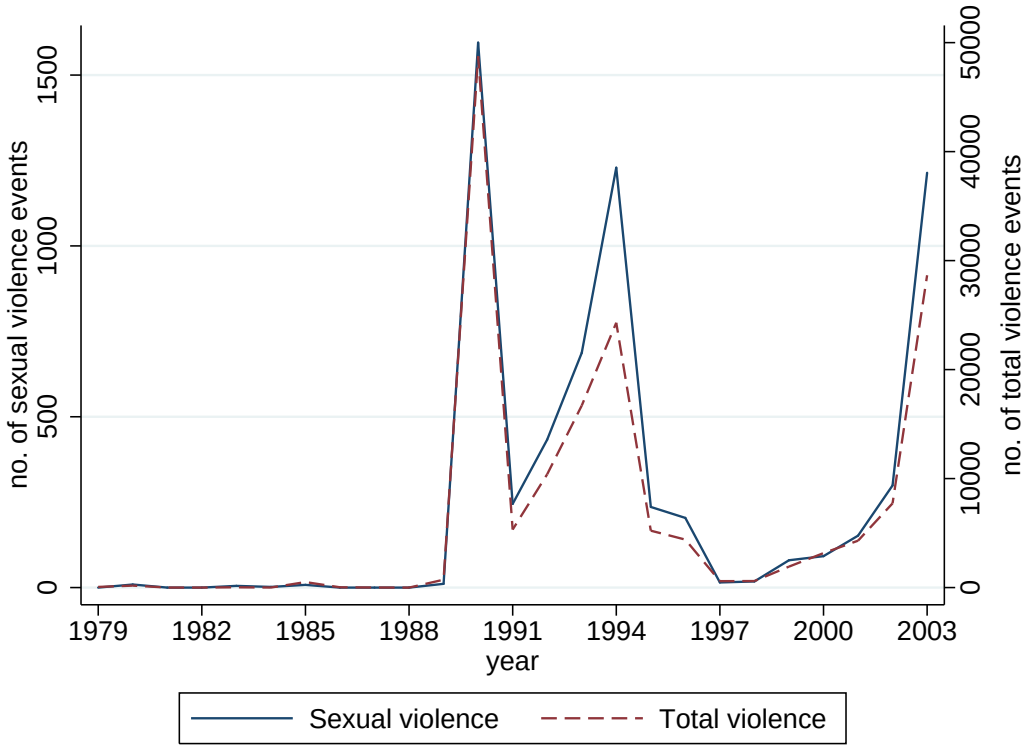

Figure A.2: Violence events by year

Source: Truth and Reconciliation Commission (TRC)
Note: Sexual violence includes rape, gang rape, multiple rape, sexual abuse and sexual slavery (See also Table A.12). The figure includes all the violence events recorded (163,615 human rights violations).

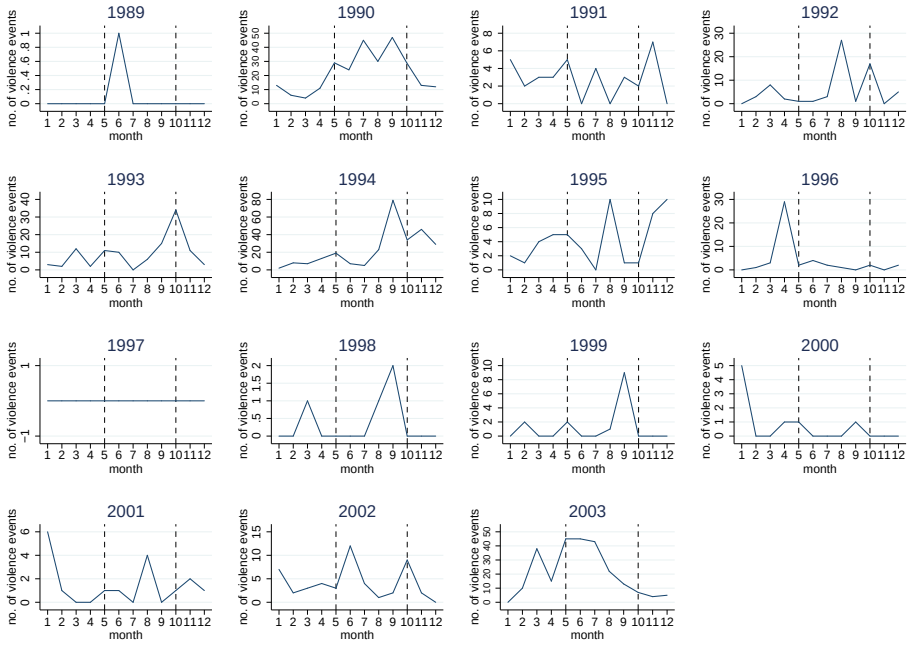

Figure A.3: Violence seasonality (sexual violence)

Source: Truth and Reconciliation Commission (TRC)
Notes: (1) Sexual violence includes rape, gang rape, multiple rape, sexual abuse and sexual slavery. (2) The number of violence events do not include cases if the year and month of alleged violation are not recorded in the dataset. Of the recorded 6,534 sexual violence cases, only 1,190 observations (approximately 18%) included information on both the year and month of incidence.

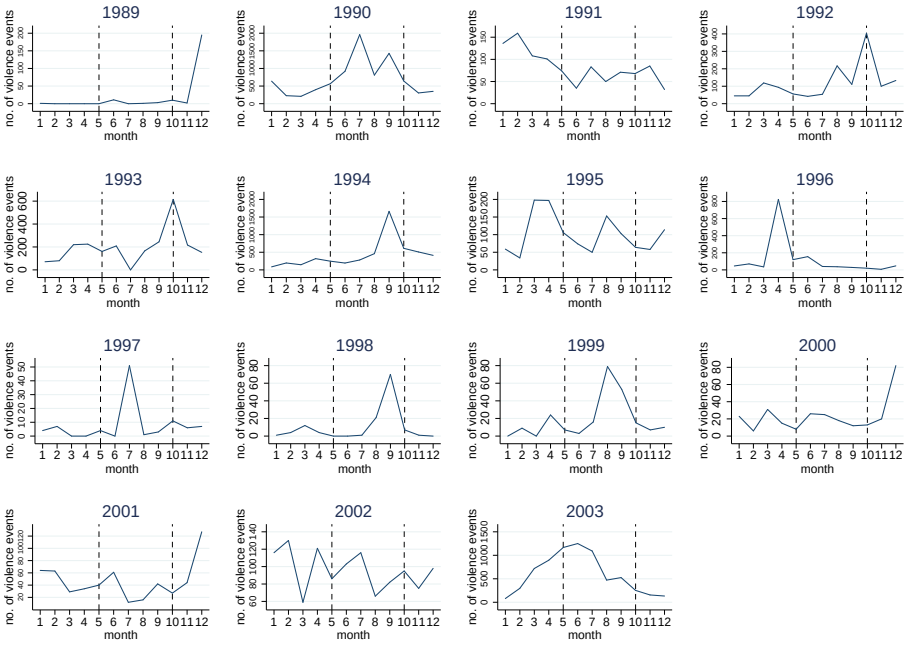

Figure A.4: Violence seasonality (total violence)

Source: Truth and Reconciliation Commission (TRC)
Notes: The number of violence events do not include cases if the year and month of alleged violation is not recorded in the dataset. Of the recorded 163,615 human rights violations, only 31,173 observations (approximately 19%) included information on both the year and month of incidence.

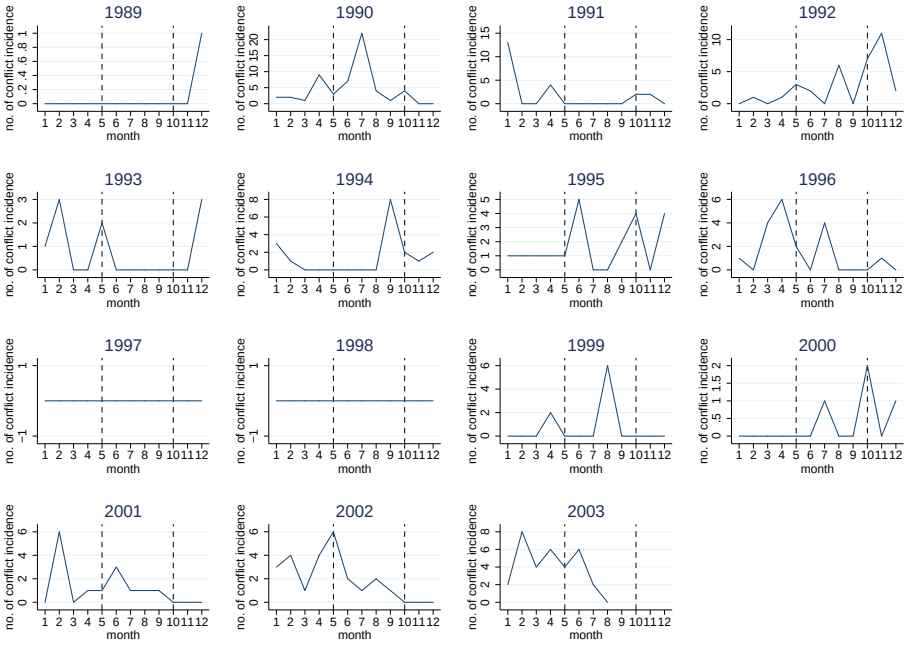

Figure A.5: Violence seasonality (conflict incidence)

Source: Armed Conflict Location and Event Database (ACLED)
Note: In the data set, 265 conflicts are recorded.

Table 1: Summary statistics

	Endemicity (50-100 percentile)			Endemicity (0-50 percentile)		
	Mean	Std.	No. of obs.	Mean	Std.	No. of obs.
Panel (A): Conceived before the war						
Die within 12 months (dummy)	0.15***	0.36	4477	0.19	0.39	4862
Male (dummy)	0.52	0.49	4477	0.51	0.49	4862
Birth order	2.46	1.63	4477	2.46	1.66	4862
Single birth (dummy)	0.96*	0.17	4477	0.97	0.16	4862
Mother's age at birth (years)	19.92	4.21	4477	19.96	4.16	4862
Mother's education (years)	2.10	5.99	4477	2.26	4.90	4862
Mother's experience of terminated pregnancy (dummy)	0.29*	0.45	4457	0.30	0.46	4861
Conceived in rainy season (dummy)	0.58	0.49	4477	0.57	0.49	4862
Urban (dummy)	0.20***	0.40	4477	0.47	0.49	4862
Longitude	-8.82***	0.94	4328	-10.17	0.90	4862
Latitude	6.28	1.19	4328	6.28	0.57	4862
Elevation (m)	213.91***	145.27	4328	64.89	63.28	5862
No. of battles ($<$ 25 km)	4.85***	7.84	4477	20.03	19.25	4862
Malaria endemicity (0—1)	0.49***	0.04	4477	0.34	0.05	4862
Mean temperature ($\times$ 10 °C), 1950—2000	253.86***	6.20	4477	258.08	3.30	4862
Mean precipitation (mm), 1950—2000	210.93***	46.29	4477	291.44	60.74	4862
Panel (B): Conceived after the outbreak of the war						
Die within 12 months (dummy)	0.11	0.31	21927	0.11	0.31	21661
Male (dummy)	0.50	0.49	21927	0.51	0.49	21661
Birth order	3.66***	2.41	21927	3.48	2.38	21661
Single birth (dummy)	0.95	0.19	21927	0.96	0.19	21661
Mother's age at birth (years)	25.39***	6.76	21927	25.18	6.66	21661
Mother's education (years)	2.21***	4.62	21927	2.97	5.46	21661
Mother's experience of terminated pregnancy (dummy)	0.22**	0.42	21873	0.23	0.42	21631
Conceived in rainy season (dummy)	0.54**	0.49	21927	0.53	0.49	21661
Urban (dummy)	0.21***	0.41	21927	0.46	0.49	21661
Longitude	-8.84***	0.93	21631	-10.18	0.89	21661
Latitude	6.29***	1.21	21631	6.27	0.59	21661
Elevation (m)	217.68***	145.94	21631	64.01	61.61	21661
No. of battles ($<$ 25 km)	4.68***	7.35	21927	18.94	18.90	21661
Malaria endemicity (0—1)	0.49***	0.04	21927	0.34	0.05	21661
Mean temperature ($\times$ 10 °C), 1950—2000	253.19***	6.21	21927	257.92	3.27	21661
Mean precipitation (mm), 1950—2000	214.35***	45.97	21927	292.38	58.18	21661

Note: The equality of means between the high and low endemicity areas is examined for children conceived before and after the outbreak of the war, respectively. *** denotes significance at 1%, ** at 5%, and * at 10%.

Table 2: Impacts of wartime pregnancy in malaria endemic areas on one-year infant mortality (OLS)

Dependent variable:	One if die within 12 months after the birth							
Sample:	All	All	All	All	All	All	2007 DHS only & Born to a mother living in her birth place	All excluding Greater Monrovia District
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Conceived after the outbreak of the war								
× Malaria endemicity (10-20 percentile)	0.029	-	-	-	-	-	-	-
	(0.022)							
× Malaria endemicity (20-30 percentile)	0.024	-	-	-	-	-	-	-
	(0.023)							
× Malaria endemicity (30-40 percentile)	-0.028	-	-	-	-	-	-	-
	(0.027)							
× Malaria endemicity (40-50 percentile)	0.016	-	-	-	-	-	-	-
	(0.023)							
× Malaria endemicity (50-60 percentile)	0.035	-	-	-	-	-	-	-
	(0.021)							
× Malaria endemicity (60-70 percentile)	0.049**	-	-	-	-	-	-	-
	(0.023)							
× Malaria endemicity (70-80 percentile)	0.047**	-	-	-	-	-	-	-
	(0.022)							
× Malaria endemicity (80-90 percentile)	0.041**	-	-	-	-	-	-	-
	(0.021)							
× Malaria endemicity (90-100 percentile)	0.045**	-	-	-	-	-	-	-
	(0.022)							
× Malaria endemicity (50-100 percentile)	-	0.035***	-	-	0.035***	0.047***	0.037**	0.036***
		(0.010)			(0.010)	(0.015)	(0.016)	(0.011)
× Malaria endemicity (continuous measure)	-	-	0.115**	0.148***	-	-	-	-
			(0.046)	(0.052)				
Male (dummy)	0.017***	0.017***	0.017***	0.017***	0.017***	0.016***	0.014**	0.017***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.006)	(0.003)
Birth order	0.013***	0.013***	0.013***	0.017***	0.013***	-0.015***	0.013***	0.013***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.003)	(0.003)	(0.001)
Single birth (dummy)	-0.240***	-0.239***	-0.239***	-0.232***	-0.240***	-0.255***	-0.248***	-0.246***
	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)	(0.018)	(0.031)	(0.015)
Mother's age at birth	-0.006***	-0.006***	-0.006***	-0.007***	-0.006***	-0.004	-0.005***	-0.006***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.007)	(0.001)	(0.000)
Mother's education (years)	-0.001	-0.001	-0.001	-0.001	-0.001	-	0.000	-0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)		(0.001)	(0.001)
Mother's experience of terminated pregnancy	-	-	-	-	0.009**	-	-	-
					(0.004)			
Mother's religion FE	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
Mother FE	No	No	No	No	No	Yes	No	No
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE (YFE)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes
Community FE	Yes	Yes	Yes	No	Yes	No	Yes	Yes
District FE (DFE)	No	No	No	Yes	No	No	No	No
DFE × YFE	No	No	No	Yes	No	No	No	No
R-squared	0.085	0.085	0.085	0.107	0.085	0.318	0.114	0.087
No. of obs.	49185	49185	49185	49185	49089	49429	11721	44299

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) In column (d), this study identified a district corresponding to each DHS community by matching a community's GPS latitude/longitude position with a map of Liberia. Consequently, the communities were categorized into 65 districts plus one group for which the ArcGIS failed to identify the corresponding district, enabling the estimations to include 66 district-level fixed effects.

Table 3: Trend of the mortality effects and month-of-birth effects (OLS)

Dependent variable:	One if die within 12 months after the birth					
Malaria endemicity:	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)
Malaria endemicity × Conceived:						
during the 1st war	0.036***	0.128***	-	-	-	-
(Dec 1989 to July 1997)	(0.011)	(0.048)				
during the ceasefire	0.031**	0.098*	-	-	-	-
(Aug 1997 to Mar 1999)	(0.014)	(0.052)				
during the 2nd war	0.034***	0.097*	-	-	-	-
(Apr 1999 to Aug 2003)	(0.012)	(0.053)				
during the post-war	0.036***	0.105*	-	-	-	-
(After Sep 2003)	(0.012)	(0.054)				
Conceived after the outbreak of the war						
× Malaria endemicity	-	-	-	-	0.068***	0.120*
					(0.020)	(0.064)
Born after the outbreak of the war						
× Malaria endemicity	-	-	0.026**	0.078*	-0.039*	-0.006
			(0.011)	(0.046)	(0.021)	(0.063)
Male (dummy)	0.017***	0.017***	0.017***	0.017***	0.017***	0.017***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Birth order	0.013***	0.013***	0.013***	0.013***	0.013***	0.013***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Single birth (dummy)	-0.240***	-0.239***	-0.239***	-0.239***	-0.239***	-0.239***
	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)	(0.014)
Mother's age at birth	-0.006***	-0.006***	-0.006***	-0.006***	-0.006***	-0.006***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Mother's education	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001
(years)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Mother's religion FE	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE (YFE)	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.085	0.085	0.085	0.085	0.085	0.085
No. of obs.	49185	49185	49185	49185	49185	49185

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community.

Table 4: The mortality effects with geographic and climate controls (OLS)

Dependent variable:	One if die within 12 months after the birth							
Malaria endemicity:	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Conceived after the outbreak of the war								
× Malaria endemicity	0.038***	0.199**	0.034***	0.095	0.027**	0.116	0.034***	0.125**
	(0.013)	(0.088)	(0.013)	(0.062)	(0.013)	(0.076)	(0.013)	(0.063)
× Temperature (× 10 °C)	-0.000	-0.000	-	-	-	-	-	-
	(0.000)	(0.000)						
× Precipitation (mm)	0.000	0.000	-	-	-	-	-	-
	(0.000)	(0.000)						
× Elevation (m)	-	-	0.000	0.000	-	-	-	-
			(0.000)	(0.000)				
× Slope (%)	-	-	-0.004	-0.003	-	-	-	-
			(0.016)	(0.017)				
× Terrain ruggedness index (100 m)	-	-	0.004	0.001	-	-	-	-
			(0.042)	(0.043)				
× Nutrient availability	-	-	-	-	-0.052	-0.086	-	-
					(0.053)	(0.059)		
× Nutrient retention capacity	-	-	-	-	0.044	0.046	-	-
					(0.034)	(0.033)		
× Rooting conditions	-	-	-	-	0.013	0.015	-	-
					(0.023)	(0.024)		
× Oxygen availability to roots	-	-	-	-	-0.019	-0.021	-	-
					(0.014)	(0.014)		
× Excess salts	-	-	-	-	-0.014	-0.011	-	-
					(0.016)	(0.019)		
× Workability	-	-	-	-	-0.005	-0.003	-	-
					(0.020)	(0.020)		
× Longitude	-	-	-	-	-	-	0.001	0.005
							(0.006)	(0.006)
× Latitude	-	-	-	-	-	-	-0.003	-0.002
							(0.008)	(0.008)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother characteristics	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother's religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.085	0.085	0.085	0.085	0.087	0.087	0.085	0.085
No. of obs.	49185	49185	48769	48769	47135	47135	48769	48769

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The individual controls include a gender dummy, birth order, a single-birth dummy, and mothers' age at birth. (4) The mother characteristics include mothers' education (years) and religion dummies.

Table 5: Heterogeneity: environmental risk (OLS)

Dependent variable:	One if die within Z months after the birth					
Sample:	No. of battles (<25 km)		Residential area		Conceived in	
	Above median	Below median	Rural	Urban	Rain season	Dry season
	(1a)	(1b)	(1c)	(1d)	(1e)	(1f)
Panel (A): $Z = 12$						
Malaria endemicity (50-100 percentile)	0.036**	0.030**	0.042***	0.024	0.039***	0.031**
× Conceived after	(0.016)	(0.015)	(0.014)	(0.018)	(0.013)	(0.013)
the outbreak of the war						
R-squared	0.087	0.087	0.091	0.077	0.094	0.103
No. of obs.	23030	26155	32562	16623	26653	22532
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)
Panel (B): $Z = 36$						
Malaria endemicity (50-100 percentile)	0.039**	0.019	0.034**	0.028	0.040***	0.026*
× Conceived after	(0.018)	(0.016)	(0.015)	(0.019)	(0.015)	(0.015)
the outbreak of the war						
R-squared	0.096	0.090	0.097	0.083	0.101	0.111
No. of obs.	20690	23354	29163	14881	23942	20102
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)
Panel (C): $Z = 60$						
Malaria endemicity (50-100 percentile)	0.037*	0.023	0.038**	0.029	0.045***	0.026
× Conceived after	(0.019)	(0.018)	(0.016)	(0.020)	(0.016)	(0.016)
the outbreak of the war						
R-squared	0.097	0.091	0.097	0.085	0.104	0.115
No. of obs.	18384	20473	25631	13226	21086	17771
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)
Panel (D): $Z = 12$						
Malaria endemicity (continuous)	0.116	0.067	0.147*	0.095	0.176***	0.087
× Conceived after	(0.071)	(0.069)	(0.078)	(0.062)	(0.067)	(0.060)
the outbreak of the war						
R-squared	0.087	0.086	0.091	0.077	0.094	0.103
No. of obs.	23030	26155	32562	16623	26653	22532
	(5a)	(5b)	(5c)	(5d)	(5e)	(5f)
Panel (E): $Z = 36$						
Malaria endemicity (continuous)	0.135*	-0.005	0.127	0.073	0.169**	0.073
× Conceived after	(0.076)	(0.078)	(0.083)	(0.072)	(0.077)	(0.066)
the outbreak of the war						
R-squared	0.096	0.090	0.097	0.083	0.101	0.111
No. of obs.	20690	23354	29163	14881	23942	20102
	(6a)	(6b)	(6c)	(6d)	(6e)	(6f)
Panel (F): $Z = 60$						
Malaria endemicity (continuous)	0.128	0.008	0.142*	0.068	0.198**	0.068
× Conceived after	(0.078)	(0.082)	(0.085)	(0.071)	(0.079)	(0.068)
the outbreak of the war						
R-squared	0.097	0.091	0.097	0.084	0.104	0.114
No. of obs.	18384	20473	25631	13226	21086	17771
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Mother characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) A rainy (dry) season is defined as periods between May to October (November to April). (4) The individual controls include a gender dummy, birth order, a single-birth dummy, and mothers' age at birth. (5) The mother characteristics include mothers' education (years) and religion dummies.

Table 6: Heterogeneity: human biology (OLS)

Dependent variable:	One if die within Z months after the birth					
	Birth order		Mother's age at birth		Gender	
	3rd and above	1st or 2nd	Above median	Below median	Male	Female
	(1a)	(1b)	(1c)	(1d)	(1e)	(1f)
Panel (A): $Z = 12$						
Malaria endemicity (50-100 percentile)	0.034**	0.036***	0.017	0.033***	0.037***	0.034**
× Conceived after	(0.015)	(0.012)	(0.017)	(0.012)	(0.013)	(0.014)
the outbreak of the war						
R-squared	0.105	0.097	0.103	0.099	0.096	0.097
No. of obs.	26881	22304	23759	25426	25173	24012
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)
Panel (B): $Z = 36$						
Malaria endemicity (50-100 percentile)	0.036**	0.030**	0.008	0.031**	0.029*	0.038**
× Conceived after	(0.017)	(0.014)	(0.021)	(0.013)	(0.015)	(0.015)
the outbreak of the war						
R-squared	0.111	0.108	0.106	0.106	0.104	0.103
No. of obs.	23737	20307	20663	23381	22529	21515
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)
Panel (C): $Z = 60$						
Malaria endemicity (50-100 percentile)	0.035**	0.035**	0.002	0.038***	0.033**	0.039**
× Conceived after	(0.018)	(0.015)	(0.022)	(0.014)	(0.016)	(0.016)
the outbreak of the war						
R-squared	0.113	0.112	0.111	0.107	0.104	0.108
No. of obs.	20519	18338	17540	21317	19915	18942
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)
Panel (D): $Z = 12$						
Malaria endemicity (continuous)	0.108	0.122**	0.037	0.111*	0.135**	0.103*
× Conceived after	(0.067)	(0.058)	(0.078)	(0.057)	(0.065)	(0.060)
the outbreak of the war						
R-squared	0.105	0.097	0.103	0.099	0.096	0.096
No. of obs.	26881	22304	23759	25426	25173	24012
	(5a)	(5b)	(5c)	(5d)	(5e)	(5f)
Panel (E): $Z = 36$						
Malaria endemicity (continuous)	0.143*	0.091	0.019	0.110*	0.119	0.101
× Conceived after	(0.073)	(0.065)	(0.088)	(0.061)	(0.075)	(0.064)
the outbreak of the war						
R-squared	0.109	0.106	0.103	0.106	0.104	0.103
No. of obs.	26881	22304	23759	25426	22529	21515
	(6a)	(6b)	(6c)	(6d)	(6e)	(6f)
Panel (F): $Z = 60$						
Malaria endemicity (continuous)	0.132*	0.105	-0.021	0.140**	0.131*	0.099
× Conceived after	(0.075)	(0.069)	(0.094)	(0.063)	(0.077)	(0.065)
the outbreak of the war						
R-squared	0.113	0.112	0.111	0.107	0.104	0.107
No. of obs.	20519	18338	17540	21317	19915	18942
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Mother characteristics	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The individual controls include a gender dummy, birth order, a single-birth dummy, and mothers' age at birth. (4) The mother characteristics include mothers' education (years) and religion dummies.

Table 7: Long-term selection on adult health: 2013 DHS only (OLS)

Dependent variables:	Height for age (z-score)			Height (cm)		
	(a)	(b)	(c)	(d)	(e)	(f)
Conceived after the outbreak of the war						
× Malaria endemicity	0.224	-	-	0.878	-	-
(10-20 percentile)	(0.143)			(1.053)		
× Malaria endemicity	0.080	-	-	0.427	-	-
(20-30 percentile)	(0.132)			(0.807)		
× Malaria endemicity	0.114	-	-	0.596	-	-
(30-40 percentile)	(0.139)			(0.852)		
× Malaria endemicity	0.181	-	-	1.043	-	-
(40-50 percentile)	(0.135)			(0.818)		
× Malaria endemicity	0.206	-	-	1.038	-	-
(50-60 percentile)	(0.157)			(0.956)		
× Malaria endemicity	0.007	-	-	-0.013	-	-
(60-70 percentile)	(0.153)			(0.932)		
× Malaria endemicity	0.312*	-	-	1.811*	-	-
(70-80 percentile)	(0.159)			(0.967)		
× Malaria endemicity	0.398**	-	-	2.368**	-	-
(80-90 percentile)	(0.166)			(1.013)		
× Malaria endemicity	0.288**	-	-	1.596*	-	-
(90-100 percentile)	(0.137)			(0.914)		
× Malaria endemicity	-	0.124*	-	-	0.766*	-
(50-100 percentile)		(0.067)			(0.434)	
× Malaria endemicity	-	-	0.802***	-	-	5.099**
(continuous measure)			(0.306)			(2.003)
Birth order	0.009	0.009	0.009	0.049	0.050	0.048
	(0.012)	(0.012)	(0.012)	(0.072)	(0.072)	(0.072)
No. of alive siblings	0.006	0.006	0.006	0.049	0.050	0.051
at age 10	(0.011)	(0.011)	(0.011)	(0.067)	(0.067)	(0.067)
No. of late siblings	-0.019	-0.017	-0.017	-0.159	-0.155	-0.157
at age 10	(0.019)	(0.019)	(0.018)	(0.133)	(0.133)	(0.133)
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.135	0.133	0.134	0.142	0.140	0.141
No. of obs.	4576	4576	4576	4582	4582	4582

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community.

Table 8: Assessment of bias attributed to unobservables

Dependent variables:	One if die within 12 months after the birth		Height for age (z-score)		Height (cm)	
Malaria endemicity:	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure
	(1a)	(1b)	(1c)	(1d)	(1e)	(1f)
Panel (A): Main results from Tables 2 and 7 (OLS)						
Conceived after the outbreak of the war	0.035***	0.115**	0.124*	0.802***	0.766*	5.099**
× Malaria endemicity	(0.010)	(0.046)	(0.067)	(0.306)	(0.434)	(2.003)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141
No. of obs.	49185	49185	4576	4576	4582	4582
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)
Panel (B): Oster (2015)'s δ						
(1) $R_{max} = 1.3\tilde{R}$	-1.28	-0.54	-1.52	-0.27	-0.91	-0.46
(2) $R_{max} = 0.3$	-0.19	-0.09	-0.41	-0.12	-0.25	-0.19
(3) $R_{max} = 1.0$	-0.04	-0.02	-0.08	-0.03	-0.04	-0.04
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)
Panel (C): Instrumental variable approach						
Conceived after the outbreak of the war	0.049***	0.297***	-0.003	-0.015	-0.390	-2.138
× Malaria endemicity	(0.018)	(0.105)	(0.152)	(0.832)	(0.944)	(5.170)
R-sqaured	0.085	0.085	0.133	0.133	0.140	0.140
No. of. obs	49185	49185	4576	4576	4582	4582
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)
Panel (C): Discontinuity sample conceived in 1989 and 1990 (OLS)						
Conceived after the outbreak of the war	0.064*	0.226	-0.174	2.367	-1.035	14.144
× Malaria endemicity	(0.037)	(0.141)	(0.832)	(2.199)	(4.964)	(13.114)
R-sqaured	0.272	0.272	0.660	0.668	0.660	0.668
No. of. obs	2556	2556	325	325	325	325
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes
Mother characteristics	Yes	Yes	NA	NA	NA	NA
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	NA	NA	NA	NA
Community FE	Yes	Yes	Yes	Yes	Yes	Yes

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community in columns (1a)—(1f) and (4a)—(4f) and to heteroskedasticity in columns (3a)—(3b). (3) The individual controls in columns (1a)—(1b), (3a)—(3b), and (4a)—(4b) include a gender dummy, birth order, a single-birth dummy, and mothers' age at birth. The individual controls in columns (1c)—(1f), (3c)—(3f), and (4c)—(4f) include birth order, no. of alive siblings at age 10, and no. of late siblings at age 10. (4) The mother characteristics include mothers' education (years) and religion dummies.

Table A.1: Checking on the pre-war trends of outcomes (OLS)

Dependent variables:	One if die within 12 months after the birth	Height for age (z-score)	Height (cm)	Education (years)
	(a)	(b)	(c)	(d)
Conceived in 1989	0.017	-0.155	-0.926	2.114***
	(0.028)	(0.106)	(0.635)	(0.304)
Conceived in 1988	-0.014	0.008	0.047	2.259***
	(0.027)	(0.176)	(1.052)	(0.338)
Conceived in 1987	-0.039	-0.149	-0.887	2.084***
	(0.029)	(0.140)	(0.834)	(0.344)
Conceived in 1986	-0.036	0.014	0.086	1.933***
	(0.028)	(0.139)	(0.830)	(0.379)
Conceived in 1985	-0.061**	-0.084	-0.502	1.936***
	(0.028)	(0.168)	(1.001)	(0.419)
Conceived in 1984	-0.039	0.016	0.096	1.507***
	(0.030)	(0.129)	(0.766)	(0.302)
Conceived in 1983	-0.069**	-0.031	-0.184	1.635***
	(0.029)	(0.130)	(0.777)	(0.339)
Conceived in 1982	-0.068**	0.055	0.328	0.465
	(0.029)	(0.164)	(0.976)	(0.379)
Conceived in 1982	-0.019	-0.174	-1.035	0.247
	(0.036)	(0.129)	(0.766)	(0.357)
Conceived in 1981	-0.018	-0.084	-0.506	0.302
	(0.038)	(0.167)	(0.999)	(0.444)
Malaria endemicity (50-100 percentile)				
× Conceived in 1989	-0.033	-0.003	0.027	-0.200
	(0.038)	(0.149)	(0.890)	(0.395)
× Conceived in 1988	0.026	0.013	0.132	0.074
	(0.039)	(0.235)	(1.402)	(0.464)
× Conceived in 1987	0.028	0.034	0.243	-0.107
	(0.039)	(0.172)	(1.025)	(0.484)
× Conceived in 1986	0.025	-0.070	-0.371	-0.429
	(0.038)	(0.215)	(1.287)	(0.471)
× Conceived in 1985	0.058	-0.096	-0.467	-0.402
	(0.039)	(0.216)	(1.293)	(0.502)
× Conceived in 1984	-0.027	-0.088	-0.288	-0.532
	(0.041)	(0.178)	(1.076)	(0.409)
× Conceived in 1983	0.062	-0.053	-0.125	-0.661
	(0.040)	(0.177)	(1.075)	(0.429)
× Conceived in 1982	0.045	-0.205	-0.951	-0.109
	(0.042)	(0.216)	(1.352)	(0.441)
× Conceived in 1982	0.035	0.061	0.481	0.063
	(0.049)	(0.172)	(1.034)	(0.468)
× Conceived in 1981	0.001	0.042	-0.089	0.221
	(0.050)	(0.244)	(1.605)	(0.543)
Round FE	Yes	NA	NA	NA
Community FE	Yes	Yes	Yes	Yes
R-squared	0.122	0.150	0.147	0.358
Unit of obs.	children	respondents	respondents	respondents
No. of obs.	9342	3135	3138	6237

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community.

Table A.2: Summary statistics (DID estimates)

Dependent variables:	Coefficient	Std.	R-sqd	No. of obs.
Male (dummy)	-0.012	(0.012)	0.000	52927
Birth order	0.178***	(0.068)	0.034	52927
Single birth (dummy)	0.004	(0.007)	0.000	52927
Mother's age at birth (years)	0.260*	(0.156)	0.094	52927
Mother's education (years)	-0.598***	(0.200)	0.005	52927
Mother's experience of terminated pregnancy (dummy)	0.009	(0.018)	0.004	52822
Conceived in rainy season (dummy)	-0.002	(0.012)	0.001	52927
Urban (dummy)	0.025	(0.024)	0.071	52927
Longitude	-0.022	(0.053)	0.347	52482
Latitude	0.021	(0.054)	0.000	52482
Elevation (m)	4.662	(6.292)	0.319	52482
No. of battles ($<$ 25km)	0.913	(0.792)	0.201	52927
Malaria endemicity (0-1)	-0.000	(0.003)	0.700	52927
Mean temperature ($\times$ 10 °C), 1950-2000	0.208	(0.305)	0.142	52927
Mean precipitation (mm), 1950-2000	2.479	(2.958)	0.356	52927

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community.

Table A.3: Impacts of wartime pregnancy in malaria endemic areas (probit and hazard models)

Dependent variables:	One if die within Z months after the birth							Months to death
	$Z = 1$	$Z = 6$	$Z = 12$	$Z = 24$	$Z = 36$	$Z = 48$	$Z = 60$	
	Probit (ME)	Probit (ME)	Probit (ME)	Probit (ME)	Probit (ME)	Probit (ME)	Probit (ME)	Hazard ratio
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Panel (A): Conceived before the war								
Malaria endemicity	0.008	-0.004	-0.002	0.003	0.002	-0.001	0.001	0.957
(50-100 percentile)	(0.009)	(0.014)	(0.018)	(0.020)	(0.020)	(0.020)	(0.021)	(0.099)
Male (dummy)	0.008*	0.017**	0.021**	0.026***	0.026***	0.027***	0.029***	1.135***
	(0.005)	(0.007)	(0.008)	(0.009)	(0.009)	(0.009)	(0.009)	(0.051)
Birth order	0.008***	0.018***	0.025***	0.027***	0.029***	0.030***	0.031***	1.128***
	(0.002)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.024)
Single birth (dummy)	-0.210***	-0.276***	-0.299***	-0.314***	-0.335***	-0.338***	-0.330***	0.434***
	(0.035)	(0.038)	(0.040)	(0.039)	(0.037)	(0.037)	(0.038)	(0.055)
Mother's age at birth	-0.004***	-0.009***	-0.012***	-0.014***	-0.014***	-0.014***	-0.015***	0.937***
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.009)
Mother's education	-0.002**	-0.007***	-0.002	-0.003	-0.004	-0.004	-0.004	0.969
(years)	(0.001)	(0.001)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.020)
Longitude	0.004	0.003	-0.020	-0.024	-0.017	-0.022	-0.022	0.958
	(0.012)	(0.017)	(0.021)	(0.024)	(0.024)	(0.025)	(0.026)	(0.139)
Latitude	0.022	0.032	0.036	0.041	0.069*	0.067*	0.070*	1.563**
	(0.022)	(0.029)	(0.035)	(0.039)	(0.038)	(0.039)	(0.040)	(0.303)
Elevation (m)	-0.000*	-0.000	-0.000	-0.000	-0.000**	-0.000*	-0.000*	0.998**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)
Shoenfeld res. (p-val.)	-	-	-	-	-	-	-	0.259
R-squared	0.070	0.077	0.059	0.055	0.055	0.055	0.054	-
No. of obs.	9084	9126	9126	9126	9126	9126	9126	8607
	(i)	(j)	(k)	(l)	(m)	(n)	(o)	(p)
Panel (B): Conceived after the outbreak of the war								
Malaria endemicity	-0.003	0.004	0.012	0.018*	0.019*	0.022*	0.025**	1.200**
(50-100 percentile)	(0.004)	(0.007)	(0.009)	(0.010)	(0.011)	(0.012)	(0.010)	(0.099)
Male (dummy)	0.011***	0.012***	0.016***	0.017***	0.019***	0.020***	0.022***	1.085***
	(0.002)	(0.002)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.030)
Birth order	0.007***	0.011***	0.017***	0.020***	0.021***	0.023***	0.024***	1.136***
	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.013)
Single birth (dummy)	-0.142***	-0.195***	-0.215***	-0.230***	-0.236***	-0.232***	-0.235***	0.424***
	(0.014)	(0.015)	(0.016)	(0.016)	(0.017)	(0.018)	(0.019)	(0.028)
Mother's age at birth	-0.003***	-0.005***	-0.007***	-0.008***	-0.009***	-0.010***	-0.010***	0.944***
	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.004)
Mother's education	-0.001*	-0.001***	-0.001**	-0.001*	-0.001**	-0.002**	-0.002**	0.988**
(years)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.006)
Longitude	0.004	0.001	-0.009	-0.020*	-0.022*	-0.020	-0.021	0.883
	(0.005)	(0.008)	(0.010)	(0.011)	(0.012)	(0.013)	(0.013)	(0.076)
Latitude	0.004	0.012	0.005	-0.007	-0.004	-0.009	-0.012	1.009
	(0.008)	(0.011)	(0.016)	(0.018)	(0.019)	(0.021)	(0.022)	(0.141)
Elevation (m)	-0.000	-0.000	-0.000	0.000	0.000	0.000	0.000	1.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)
Shoenfeld res. (p-val.)	-	-	-	-	-	-	-	0.264
R-squared	0.062	0.067	0.064	0.060	0.059	0.055	0.053	-
No. of obs.	42363	41129	39643	37125	34533	31806	29387	41230
Mother's religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
County FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) This study identified a county corresponding to each DHS community by matching a community's GPS latitude/longitude position with a map of Liberia. Consequently, the communities were categorized into 15 counties plus one group for which the ArcGIS failed to identify the corresponding county, enabling the estimations to include 16 county-level fixed effects.

Table A.4: Long-term selection on education: 2013 DHS only

Dependent variable:	Education (years)						
Sample:	All	All	All	All	All	Conceived in 1989 and 1990	Conceived in 1989 and 1990
	OLS	OLS	OLS	2SLS	2SLS	OLS	OLS
	(a)	(b)	(c)	(d)	(e)	(f)	(g)
Conceived after the outbreak of the war							
× Malaria endemicity	-0.726*	-	-	-	-	-	-
(10-20 percentile)	(0.440)						
× Malaria endemicity	-0.330	-	-	-	-	-	-
(20-30 percentile)	(0.484)						
× Malaria endemicity	0.753**	-	-	-	-	-	-
(30-40 percentile)	(0.377)						
× Malaria endemicity	0.674	-	-	-	-	-	-
(40-50 percentile)	(0.440)						
× Malaria endemicity	0.946**	-	-	-	-	-	-
(50-60 percentile)	(0.404)						
× Malaria endemicity	-0.007	-	-	-	-	-	-
(60-70 percentile)	(0.352)						
× Malaria endemicity	0.430	-	-	-	-	-	-
(70-80 percentile)	(0.401)						
× Malaria endemicity	0.537	-	-	-	-	-	-
(80-90 percentile)	(0.389)						
× Malaria endemicity	0.241	-	-	-	-	-	-
(90-100 percentile)	(0.441)						
× Malaria endemicity	-	0.319*	-	0.364	-	0.113	-
(50-100 percentile)		(0.190)		(0.341)		(0.896)	
× Malaria endemicity	-	-	1.574	-	2.006	-	-0.270
(continuous measure)			(0.957)		(1.880)		(2.833)
× Temperature (× 10 °C)	-	-	-	-0.001	-0.004	-	-
				(0.003)	(0.005)		
× Precipitation (mm)	-	-	-	-0.001	0.000	-	-
				(0.002)	(0.002)		
Birth order	-0.018	-0.021	-0.021	-0.020	-0.020	-0.049	-0.047
	(0.027)	(0.027)	(0.027)	(0.024)	(0.024)	(0.136)	(0.137)
No. of alive siblings	-0.015	-0.012	-0.012	-0.012	-0.013	0.104	0.101
at age 10	(0.028)	(0.028)	(0.028)	(0.024)	(0.024)	(0.147)	(0.151)
No. of late siblings	0.025	0.032	0.031	0.031	0.030	-0.109	-0.110
at age 10	(0.042)	(0.042)	(0.042)	(0.038)	(0.038)	(0.236)	(0.237)
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.369	0.367	0.367	0.367	0.367	0.633	0.633
No. of obs.	9197	9197	9197	9197	9197	640	640
1st-stage coefficient on:							
Conceived after the outbreak of the war	-	-	-	-0.000***	-0.000***	-	-
× Temperature (× 10 °C)				(0.000)	(0.000)		
× Precipitation (mm)							
1st-stage F-statistics	-	-	-	3223.10	4686.00	-	-

Notes: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community in all columns excluding (d) and (e).

Table A.5: Long-term selection on height-for-age with geographic and climate controls: 2013 DHS only (OLS)

Dependent variable:	Height for age (z-scores)							
Malaria endemicity:	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Conceived after the outbreak of the war								
× Malaria endemicity	0.069	0.732	0.081	0.761*	0.031	0.371	0.095	0.920**
	(0.086)	(0.540)	(0.091)	(0.430)	(0.087)	(0.493)	(0.098)	(0.442)
× Temperature (× 10 °C)	0.002	0.000	-	-	-	-	-	-
	(0.001)	(0.002)						
× Precipitation (mm)	-0.001	-0.000	-	-	-	-	-	-
	(0.001)	(0.001)						
× Elevation (m)	-	-	0.000	-0.000	-	-	-	-
			(0.000)	(0.000)				
× Slope (%)	-	-	0.288*	0.269	-	-	-	-
			(0.170)	(0.173)				
× Terrain ruggedness index (100m)	-	-	-0.754*	-0.708*	-	-	-	-
			(0.389)	(0.398)				
× Nutrient availability	-	-	-	-	0.537	0.361	-	-
					(0.480)	(0.539)		
× Nutrient retention capacity	-	-	-	-	-0.192	-0.171	-	-
					(0.344)	(0.349)		
× Rooting conditions	-	-	-	-	-0.038	-0.031	-	-
					(0.160)	(0.159)		
× Oxygen availability to roots	-	-	-	-	-0.052	-0.030	-	-
					(0.094)	(0.097)		
× Excess salts	-	-	-	-	-0.213*	-0.167	-	-
					(0.112)	(0.130)		
× Workability	-	-	-	-	0.053	0.053	-	-
					(0.108)	(0.106)		
× Longitude	-	-	-	-	-	-	0.022	0.002
							(0.046)	(0.041)
× Latitude	-	-	-	-	-	-	0.026	-0.025
							(0.055)	(0.057)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.134	0.134	0.134	0.135	0.132	0.132	0.133	0.134
No. of obs.	4576	4576	4576	4576	4450	4450	4576	4576

Note: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The individual controls include birth order, no. of alive siblings at age 10, and no. of late siblings at age 10.

Table A.6: Long-term selection on height with geographic and climate controls: 2013 DHS only (OLS)

Dependent variable:	Height (cm)							
Malaria endemicity:	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Conceived after the outbreak of the war								
× Malaria endemicity	0.345	4.071	0.458	4.711*	0.172	2.528	0.556	5.690**
	(0.527)	(3.366)	(0.555)	(2.674)	(0.530)	(3.121)	(0.604)	(2.727)
× Temperature (× 10 °C)	0.012	0.003	-	-	-	-	-	-
	(0.008)	(0.012)						
× Precipitation (mm)	-0.006	-0.002	-	-	-	-	-	-
	(0.004)	(0.005)						
× Elevation (m)	-	-	0.000	-0.001	-	-	-	-
			(0.002)	(0.002)				
× Slope (%)	-	-	1.437	1.316	-	-	-	-
			(1.055)	(1.082)				
× Terrain ruggedness index (100m)	-	-	-4.512*	-4.210*	-	-	-	-
			(2.346)	(2.411)				
× Nutrient availability	-	-	-	-	3.149	1.924	-	-
					(2.932)	(3.365)		
× Nutrient retention capacity	-	-	-	-	-1.069	-0.924	-	-
					(2.119)	(2.155)		
× Rooting conditions	-	-	-	-	-0.072	-0.017	-	-
					(1.061)	(1.051)		
× Oxygen availability to roots	-	-	-	-	-0.232	-0.069	-	-
					(0.567)	(0.597)		
× Excess salts	-	-	-	-	-1.423*	-1.085	-	-
					(0.733)	(0.853)		
× Workability	-	-	-	-	0.254	0.244	-	-
					(0.797)	(0.780)		
× Temperature (× 10 °C)	-	-	-	-	-	-	0.151	0.016
							(0.286)	(0.254)
× Precipitation (mm)	-	-	-	-	-	-	0.199	-0.125
							(0.348)	(0.360)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.141	0.141	0.142	0.142	0.138	0.138	0.140	0.141
No. of obs.	4582	4582	4582	4582	4456	4456	4582	4582

Note: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The individual controls include birth order, no. of alive siblings at age 10, and no. of late siblings at age 10.

Table A.7: Long-term selection on education with geographic and climate controls: 2013 DHS only (OLS)

Dependent variable:	Education (years)							
Malaria endemicity:	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure	50-100 percentile	continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Conceived after the outbreak of the war								
× Malaria endemicity	0.231	2.312	0.132	0.760	0.102	0.466	0.262	0.875
	(0.222)	(1.462)	(0.217)	(1.188)	(0.220)	(1.403)	(0.212)	(1.100)
× Temperature (× 10 °C)	-0.000	-0.005	-	-	-	-	-	-
	(0.003)	(0.005)						
× Precipitation (mm)	-0.001	0.000	-	-	-	-	-	-
	(0.002)	(0.002)						
× Elevation (m)	-	-	0.001*	0.001	-	-	-	-
			(0.001)	(0.001)				
× Slope (%)	-	-	-0.527	-0.537	-	-	-	-
			(0.408)	(0.407)				
× Terrain ruggedness index (100m)	-	-	1.239	1.266	-	-	-	-
			(1.014)	(1.010)				
× Nutrient availability	-	-	-	-	0.404	0.228	-	-
					(0.695)	(0.927)		
× Nutrient retention capacity	-	-	-	-	-0.679***	-0.659***	-	-
					(0.205)	(0.230)		
× Rooting conditions	-	-	-	-	-0.530	-0.522	-	-
					(0.374)	(0.375)		
× Oxygen availability to roots	-	-	-	-	0.073	0.078	-	-
					(0.235)	(0.236)		
× Excess salts	-	-	-	-	-0.675**	-0.656*	-	-
					(0.310)	(0.361)		
× Workability	-	-	-	-	0.338	0.353	-	-
					(0.346)	(0.346)		
× Longitude	-	-	-	-	-	-	0.012	0.043
							(0.100)	(0.094)
× Latitude	-	-	-	-	-	-	0.133	0.144
							(0.131)	(0.134)
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.367	0.367	0.367	0.367	0.369	0.369	0.367	0.367
No. of obs.	9197	9197	9197	9197	8961	8961	9197	9197

Note: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The individual controls include birth order, no. of alive siblings at age 10, and no. of late siblings at age 10.

Table A.8: Impacts of wartime pregnancy in malaria endemic areas (2SLS)

Dependent variables:	One if die within 12 months after the birth		Height for age (z-score)		Height (cm)	
Malaria endemicity:	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)
Conceived after the outbreak of the war						
× Malaria endemicity	0.049***	0.297***	-0.003	-0.015	-0.390	-2.138
	(0.018)	(0.105)	(0.152)	(0.832)	(0.944)	(5.170)
× Temperature (× 10 °C)	-0.000	-0.001**	0.002	0.002	0.015*	0.019
	(0.000)	(0.000)	(0.001)	(0.002)	(0.008)	(0.015)
× Precipitation (mm)	0.000	0.000	-0.001	-0.001	-0.009*	-0.010
	(0.000)	(0.000)	(0.001)	(0.001)	(0.005)	(0.007)
Male (dummy)	0.017***	0.017***	-	-	-	-
	(0.003)	(0.003)				
Birth order	0.013***	0.013***	0.008	0.008	0.045	0.045
	(0.001)	(0.001)	(0.011)	(0.011)	(0.068)	(0.068)
Single birth (dummy)	-0.240***	-0.240***	-	-	-	-
	(0.011)	(0.011)				
Mother's age at birth	-0.006***	-0.006***	-	-	-	-
	(0.000)	(0.000)				
Mother's education	-0.001***	-0.001***	-	-	-	-
(years)	(0.000)	(0.000)				
No. of alive siblings	-	-	0.007	0.007	0.053	0.054
at age 10			(0.011)	(0.011)	(0.068)	(0.068)
No. of late siblings	-	-	-0.018	-0.018	-0.162	-0.161
at age 10			(0.017)	(0.017)	(0.108)	(0.108)
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	NA	NA	NA	NA
Community FE	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.085	0.085	0.133	0.133	0.140	0.140
Unit of obs.	children	children	respondents	respondents	respondents	respondents
No. of obs.	49185	49185	4576	4576	4582	4582
1st-stage coefficient on:						
Conceived after the outbreak of the war	-0.000***	-0.000***	-0.000***	-0.000***	-0.000***	-0.000***
× Temperature (× 10 °C)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
× Precipitation (mm)						
1st-stage F-statistics	5210.75	7005.99	1578.33	2300.56	1571.50	2292.42

Note: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity in columns (a) and (b).

Table A.9: Impacts of wartime pregnancy in malaria endemic areas: discontinuity sample (OLS)

Dependent variables:	One if die within 12 months after the birth		Height for age (z-score)		Height (cm)	
Malaria endemicity:	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure
	(a)	(b)	(c)	(d)	(e)	(f)
Conceived after the outbreak of the war						
× Malaria endemicity	0.064*	0.226	-0.174	2.367	-1.035	14.144
	(0.037)	(0.141)	(0.832)	(2.199)	(4.964)	(13.114)
Male (dummy)	0.027	0.026	-	-	-	-
	(0.019)	(0.019)				
Birth order	0.022***	0.022***	0.004	0.002	0.026	0.011
	(0.008)	(0.008)	(0.117)	(0.118)	(0.700)	(0.701)
Single birth (dummy)	-0.400***	-0.400***	-	-	-	-
	(0.068)	(0.069)				
Mother's age at birth	-0.014***	-0.014***	-	-	-	-
	(0.003)	(0.003)				
Mother's education	-0.001	-0.001	-	-	-	-
(years)	(0.002)	(0.002)				
No. of alive siblings	-	-	-0.051	-0.037	-0.302	-0.221
at age 10			(0.139)	(0.135)	(0.828)	(0.808)
No. of late siblings	-	-	-0.058	-0.076	-0.347	-0.453
at age 10			(0.186)	(0.193)	(1.111)	(1.150)
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	NA	NA	NA	NA
Community FE	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.272	0.272	0.660	0.668	0.660	0.668
Unit of obs.	children	children	respondents	respondents	respondents	respondents
No. of obs.	2556	2556	325	325	325	325

Note: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The estimations exploit data on children [columns (a)—(b)] or respondents [columns (c)—(f)] conceived in 1989 and 1990.

Table A.10: Robustness to Bhatt et al. (2015)'s endemicity estimates of *P.falciparum* (OLS)

Dependent variables:	One if die within 12 months after the birth		Height for age (z-score)		Height (cm)		Education (years)	
Malaria endemicity:	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure	50-100 percentile	Continuous measure
	(1a)	(1b)	(1c)	(1d)	(1e)	(1f)	(1g)	(1h)
Panel (A): Endemicity in 2000								
Conceived after the outbreak of the war	0.020**	0.076**	0.146**	0.701**	0.848**	4.861**	0.668***	2.453***
× Malaria endemicity	(0.010)	(0.037)	(0.067)	(0.275)	(0.427)	(1.886)	(0.189)	(0.750)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141	0.368	0.368
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)	(2g)	(2h)
Panel (B): Endemicity in 2001								
Conceived after the outbreak of the war	0.022**	0.081**	0.123*	0.701***	0.807*	4.828***	0.687***	2.367***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.267)	(0.431)	(1.827)	(0.190)	(0.734)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141	0.368	0.368
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)	(3g)	(3h)
Panel (C): Endemicity in 2002								
Conceived after the outbreak of the war	0.026**	0.084**	0.110	0.733***	0.734*	5.013***	0.631***	2.319***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.266)	(0.432)	(1.818)	(0.191)	(0.736)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141	0.368	0.368
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)	(4g)	(4h)
Panel (D): Endemicity in 2003								
Conceived after the outbreak of the war	0.026**	0.086**	0.118*	0.734***	0.778*	5.121***	0.616***	2.218***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.259)	(0.431)	(1.838)	(0.192)	(0.733)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141	0.368	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(5a)	(5b)	(5c)	(5d)	(5e)	(5f)	(5g)	(5h)
Panel (E): Endemicity in 2004								
Conceived after the outbreak of the war	0.025**	0.080**	0.124*	0.744***	0.820*	5.151***	0.608***	2.126***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.261)	(0.432)	(1.855)	(0.189)	(0.745)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141	0.368	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(6a)	(6b)	(6c)	(6d)	(6e)	(6f)	(6g)	(6h)
Panel (F): Endemicity in 2005								
Conceived after the outbreak of the war	0.014	0.072**	0.150**	0.723***	0.990**	4.931***	0.537***	2.076***
× Malaria endemicity	(0.010)	(0.036)	(0.068)	(0.265)	(0.435)	(1.849)	(0.190)	(0.763)
R-squared	0.085	0.085	0.133	0.134	0.141	0.141	0.367	0.367
No. of obs.	49185	49185	4576	4576	4582	4582		
	(7a)	(7b)	(7c)	(7d)	(7e)	(7f)	(7g)	(7h)
Panel (G): Endemicity in 2006								
Conceived after the outbreak of the war	0.011	0.067*	0.167**	0.712***	1.088**	4.754**	0.454**	1.975**
× Malaria endemicity	(0.010)	(0.037)	(0.068)	(0.269)	(0.436)	(1.840)	(0.191)	(0.781)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141	0.367	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(8a)	(8b)	(8c)	(8d)	(8e)	(8f)	(8g)	(8h)
Panel (H): Endemicity in 2007								
Conceived after the outbreak of the war	0.016	0.066*	0.225***	0.696***	1.413***	4.573**	0.477**	1.984**
× Malaria endemicity	(0.010)	(0.037)	(0.067)	(0.265)	(0.433)	(1.776)	(0.192)	(0.777)
R-squared	0.085	0.085	0.135	0.134	0.142	0.141	0.367	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(9a)	(9b)	(9c)	(9d)	(9e)	(9f)	(9g)	(9h)
Panel (I): Endemicity in 2008								
Conceived after the outbreak of the war	0.021**	0.068*	0.206***	0.670***	1.308***	4.302**	0.303	1.968***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.257)	(0.432)	(1.686)	(0.193)	(0.755)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141	0.367	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(10a)	(10b)	(10c)	(10d)	(10e)	(10f)	(10g)	(10h)
Panel (J): Endemicity in 2009								
Conceived after the outbreak of the war	0.018*	0.074**	0.195***	0.665***	1.247***	4.237**	0.315	1.983***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.255)	(0.430)	(1.646)	(0.192)	(0.751)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141	0.367	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
	(11a)	(11b)	(11c)	(11d)	(11e)	(11f)	(11g)	(11h)
Panel (K): Endemicity in 2010								
Conceived after the outbreak of the war	0.016	0.080**	0.178***	0.663**	1.139***	4.163**	0.378*	2.239***
× Malaria endemicity	(0.010)	(0.037)	(0.067)	(0.265)	(0.430)	(1.685)	(0.193)	(0.771)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141	0.367	0.367
No. of obs.	49185	49185	4576	4576	4582	4582	9197	9197
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother characteristics	Yes	Yes	NA	NA	NA	NA	NA	NA
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	NA	NA	NA	NA	NA	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Note: (1) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (2) Standard errors are robust to heteroskedasticity and clustered residuals within each community. (3) The individual controls in the first two columns include a gender dummy, birth order, a single-birth dummy, and mothers' age at birth. The individual controls in the remaining columns include birth order, no. of alive siblings at age 10, and no. of late siblings at age 10. (4) The mother characteristics include mothers' education (years) and religion dummies.

Table A.11: Violence seasonality (OLS)

Dependent variables:	No. of							
	total violence		sexual violence		non-sexual violence		Conflict incidence	
Data sources:	TRC (Apr 1979 to Dec 2003)						ACLED (Jan 1998 to Aug 2003)	
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Rainy seasons	6.873**	7.055***	0.294***	0.288***	6.578**	6.767***	0.010	0.011
×After the outbreak of the war	(2.317)	(2.302)	(0.084)	(0.083)	(2.240)	(2.225)	(0.027)	(0.025)
Rainy seasons (dum.)	-0.253*	-	-0.002	-	-0.251*	-	-0.002	-
	(0.127)		(0.003)		(0.125)		(0.002)	
After the outbreak of the war	8.517***	-	0.315***	-	8.202***	-	0.104***	-
	(1.051)		(0.056)		(1.006)		(0.025)	
R-squared	0.045	0.144	0.037	0.105	0.045	0.144	0.042	0.075
No. of observations	4455	4455	4455	4455	4455	4455	3976	3976
	(i)	(j)	(k)	(l)	(m)	(n)	(o)	(p)
Rainy seasons								
× During the 1st war	9.339**	9.119***	0.383***	0.359***	8.957**	8.760**	0.021	0.017
	(3.187)	(3.034)	(0.116)	(0.115)	(3.100)	(2.950)	(0.030)	(0.027)
× During the ceasefire	0.779	1.675	-0.001	0.020	0.780	1.655	0.002	0.010
	(0.516)	(1.018)	(0.012)	(0.027)	(0.507)	(0.995)	(0.002)	(0.007)
× During the 2nd war	3.728	7.005*	0.234	0.340**	3.494	6.665*	-0.013	0.001
	(3.016)	(3.380)	(0.136)	(0.142)	(2.902)	(3.260)	(0.037)	(0.035)
× During the post-war periods	17.153	-14.471	0.369	-0.832*	16.784	-13.638	-	-
	(10.236)	(11.388)	(0.216)	(0.449)	(10.293)	(11.219)		
Rainy seasons (dum.)	-0.253*	-	-0.002	-	-0.251*	-	-0.002	-
	(0.127)		(0.003)		(0.125)		(0.002)	
During the 1st war	10.740***	-	0.418***	-	10.322***	-	0.124***	-
(Dec 1989 to July 1997)	(1.468)		(0.076)		(1.405)		(0.031)	
During the ceasefire	-0.030	-	0.014	-	-0.044	-	-0.002	-
(Aug 1997 to Mar 1999)	(0.225)		(0.014)		(0.216)		(0.002)	
During the 2nd war	8.077***	-	0.254***	-	7.822***	-	0.115**	-
(Apr 1999 to Aug 2003)	(1.672)		(0.075)		(1.613)		(0.039)	
During the post-war periods	8.770***	-	0.296*	-	8.474***	-	-	-
(After Sep 2003)	(2.225)		(0.161)		(2.138)			
R-squared	0.058	0.147	0.047	0.108	0.057	0.146	0.049	0.075
No. of observations	4455	4455	4455	4455	4455	4455	3976	3976
Year-fixed effects	No	Yes	No	Yes	No	Yes	No	Yes
Month-fixed effects	No	Yes	No	Yes	No	Yes	No	Yes
County-fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: (1) Unit of observations is a county-year-month. (2) Figures ( ) are standard errors. *** denotes significance at 1%, ** at 5%, and * at 10%. (3) Standard errors are robust to heteroskedasticity and clustered residuals within each county. (4) In columns (a) to (f) and (i) to (n), counties are classified into 15 groups, i.e., Bomi, Bong, Gbarpolu, Grand Bassa, Grand Cape Mount, Grand Gedeh, Grand Kru, Lofa, Margibi, Maryland, Montserrado, Nimba, Rivercess, River Gee, and Sinoe. (5) In columns (g) to (h) and (o) to (p), counties are classified into 14 groups, i.e., Bomi, Bong, Grand Bassa, Grand Cape Mount, Grand Gedeh, Grand Kru, Lofa, Margibi, Maryland, Montserrado, Nimba, Rivercess, River Gee, and Sinoe. (6) In columns (a) to (f) and (i) to (n), the dependent variables (i.e., the number of violence events) do not include cases if time and location of the alleged violation are not recorded in the TRC dataset. Of the recorded 163,615 human rights violations, only 31,001 observations (approximately 19%) observations included all the information on year, month and place of incidence.

Table A.12: Recorded human rights violations

	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991
Abduction	1	13	0	0	1	0	58	9	1	0	56	2762	404
Amputation	1	0	0	0	0	0	0	0	0	0	0	13	1
Arbitrary detention	12	22	0	0	7	0	73	1	3	0	14	1043	185
Assault	6	24	0	0	12	2	68	1	4	0	38	3293	444
Cannibalism	0	0	0	0	0	0	0	0	0	0	0	22	2
Drugging	0	0	0	0	0	0	0	0	0	0	0	11	0
Exposure/deprivation	0	0	0	0	0	0	0	0	0	0	3	653	74
Extortion	0	2	0	0	0	0	12	2	0	0	8	662	71
Forced displacement	6	22	1	3	0	0	72	4	0	0	394	20256	1777
Forced labor	0	4	0	0	0	0	8	0	0	0	5	1580	205
Forced recruitment	0	0	0	0	0	0	0	9	0	0	4	415	76
Ingesting taboo item	0	0	0	0	0	0	0	0	0	0	1	69	14
Killing	29	55	2	0	2	2	133	0	1	0	104	9407	1001
Looting	3	13	0	0	0	0	12	0	0	1	16	2116	222
Missing	0	3	0	0	0	0	6	1	1	0	7	521	62
Property destruction	3	2	0	0	0	0	5	0	0	0	21	1557	184
Robbery	1	0	0	0	0	0	13	0	0	0	21	1535	172
Torture	0	11	0	0	0	1	37	0	0	0	20	1240	189
Rape/gang rape/multiple rape	0	8	0	0	0	2	1	0	0	0	5	764	121
Sexual abuse	0	1	0	0	5	0	7	0	0	0	4	600	79
Sexual slavery	0	0	0	0	0	0	0	0	0	0	2	231	45
All crimes	62	180	3	3	27	7	505	27	10	1	723	48750	5328

	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	Total
Abduction	965	1578	2227	501	273	56	37	186	324	406	984	2203	13045
Amputation	2	10	8	2	4	0	0	1	1	0	6	9	58
Arbitrary detention	288	454	579	153	132	32	31	47	44	46	147	704	4017
Assault	797	1314	2252	458	401	76	59	121	166	243	531	2912	13222
Cannibalism	6	12	20	4	3	0	0	0	2	1	5	9	86
Drugging	4	15	4	1	0	0	0	2	2	1	12	29	81
Exposure/deprivation	209	260	281	61	74	8	2	25	40	49	94	215	2048
Extortion	123	170	235	59	30	19	28	19	25	30	81	519	2095
Forced displacement	3740	5522	8167	1409	1274	131	172	861	1365	1834	2759	9080	58849
Forced labor	461	910	1235	283	232	29	36	66	144	302	621	1439	7560
Forced recruitment	104	127	202	54	31	2	22	43	72	129	216	527	2033
Ingesting taboo item	11	20	39	10	4	1	1	5	4	3	6	67	255
Killing	1897	3221	4138	1077	1052	117	109	236	446	587	893	3533	28042
Looting	335	557	852	205	194	33	13	43	110	104	238	2552	7619
Missing	93	118	185	32	34	4	1	13	35	67	61	192	1436
Property destruction	321	650	1038	295	181	16	37	58	154	185	308	866	5881
Robbery	312	470	748	167	156	29	23	58	74	117	271	1650	5817
Torture	295	603	860	214	126	25	12	61	63	46	197	937	4937
Rape/gang rape/multiple rape	289	375	675	112	119	11	11	41	56	90	174	626	3480
Sexual abuse	99	186	367	89	62	3	4	30	12	39	73	371	2031
Sexual slavery	45	126	187	35	23	1	3	9	24	23	52	217	1023
All crimes	10396	16698	24299	5221	4405	593	601	1925	3163	4302	7729	28657	163615

Source: Truth and Reconciliation Commission (TRC)