Malaria Infection and Infant Mortality during the War: Evidence from Liberia*

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March, 2017

Abstract

Armed conflict greatly increases the risk of malaria-related mortality because people seeking to escape from battle-ridden areas often hide in and/or travel through malaria-prone bushes and forests and are unable to follow typical preventive actions. This study investigates whether the Liberian civil war increased infant mortality by exposing pregnant mothers and their children to a high risk of malaria infection. Results suggest that the war-induced, one-percent increase in infection risk caused 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, irrespective of gender, who were conceived during the rainy season by young mothers residing in rural, battle-intensive areas. The importance of maternal passive immunity and heterogeneous mortality consequences for infants conceived by geographically, seasonally, and immunologically high-risk mothers may assist policymakers and practitioners in determining target groups for aid programs during inter- and post-war periods.

Keywords: Armed conflict, fetal development, infant mortality, malaria in pregnancy and infancy

JEL classification: I15

*I thank Rulof Burger, James Fenske, Seiro Ito, Berber Kramer, Tomohiro Machikita, Saumik Paul, Akira Sato, William Seitz, Yoshito Takasaki, Shinichi Takeuchi, Miwa Tsuda, Anthony Wray, and participants at the CSAE 2017 (Oxford), 2nd Hitotsubashi Summer Institute 2016 (Tokyo) and NOVAFRICA 2016 (Lisbon), and seminars at University of Dar es Salaam, GRIPS, Hitotsubashi, IDE-JETRO, and Keio for valuable comments and suggestions. My great thanks also go to Souknilanh Keola for helping me process geo-coded data. Financial support from the IDE-JETRO and the Japan Association for African Studies (JAAS) for this research is gratefully acknowledged. My great thanks in my field trip to Bududuram in Ghana goes to Frederick Mills, Alexander Nimo Wiredu, and Liberian respondents. The findings, interpretations, and conclusions expressed in this paper are entirely those of the author and do not represent the views of the IDE-JETRO. All errors are my own.

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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., 2012; Bundervoet et al., 2009; Minoiu and Shemyakina, 2012), one recent, leading question is how an armed conflict harms human capital stock rather than whether such harm occurs at all (Blattman and Miguel, 2010, p. 42). This question is important when designing effective assistance programs both during and after the conflict and to better understand the long-term influence of war on economic development. Therefore, an emerging body of empirical research has only recently illuminated the significance of the limited effectiveness of consumption-smoothing strategies (Verpoorten, 2009), reduced dietary intake (Alderman et al., 2006; Dabalen and Paul, 2014), economic loss (Minoiu and Shemyakina, 2014), and prenatal maternal stress (Camacho, 2008; Koppensteiner and Manacorda, 2016; Mansour and Rees, 2012) as mechanisms underlying conflict-induced health damage.

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

By focusing on the Liberian civil war, this study explores whether the armed conflict increased infant mortality by exposing pregnant mothers and their children to a high risk of malaria infection. This war provides a promising setting for the purpose of the current study. First, Liberia is an ideal breeding ground for malaria-carrying mosquitoes (see also the supplemental appendix S.1.1). Almost throughout the country, malaria transmission is possible during the entire year.[1] Furthermore, due to the brutal violence, approximately 60% of the country's pre-war population was either internally or externally displaced during the 14-year conflict. Therefore, the Liberians were exposed 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 causing the deaths and flights of health professionals (Kruk et al., 2010).

Infant mortality is worth exploring for two reasons. First, among adults, mobile and pregnant populations are among the highest risk groups for malaria infection. It has been sufficiently established that maternal malaria infection hinders fetal development and is highly associated with unfavorable birth outcomes (e.g., low birthweight),

[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).

which consequently increase the risk of newborn deaths. Second, infants are also at a considerably high risk of contracting malaria.

To address the study's question, it examines child-level data based on the respondent females' full birth history in two rounds (2007 and 2013) of the Standard 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) and it produces the most deadly health consequences among human malaria parasites (WHO, 2013, p. 72).

Assuming that pregnant mothers and their children hiding in and/or escaping from malaria-endemic areas were 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 both pregnancy and infancy, which increased in mothers' original places of residence during the war. To achieve this purpose, this study compares 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, along with community-level fixed effects) and shows that wartime pregnancy in areas of high infection risk exhibited increased infant mortality. This finding, obtained using the differences-in-differences (DID) approach, is robust to the utilization of both the indicator and the continuous measure of malaria infection risk, alternative controls (e.g., geographic and climate controls sourced from numerous geo-coded data sets, mother-fixed effects), time trends specific to each community, 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 also shows that war-induced infant mortality gradually increased and could be seen more clearly from six months after childbirth. Children born to a mother infected with malaria are expected to be very susceptible to several infectious diseases due to their low birthweights. While antibodies passed from the mother to the fetus protect such low-birthweight newborns, this maternal passive immunity wanes six to 12 months after the child's birth. Therefore, this decreased immunity might make infants' vulnerability more evident while gradually increasing their mortality rate.

Moreover, the mortality effect was more pronounced for children conceived during the rainy season by mothers residing in battle-intensive, rural areas. Based on the knowledge of human biology, this study also investigates whether (maternal) parity- and age-dependent immunity affects the mortality effect and provides evidence that highlights the importance of maternal age at birth. However, the current research finds no significant gender

differences with respect to adverse mortality effect.

It is also argued that the DID estimate may be a lower-bound effect of wartime malaria infection risk; the relevant analysis considers the following three approaches: assessment of the importance of unobservables (relative to the observed controls) required to explain the identified mortality effect (Oster, forthcoming); exploitation of insight obtained from a regression discontinuity design; and an instrumental variable approach.

This study consolidates the following three strands of the extant economic literature. First, as describe above, empirical studies have recently explored transmission channels driving human capital consequences of wars. Second, the knowledge and understanding of socio-economic impacts of malaria have considerably increased in recent studies focusing on Africa (e.g., Barofsky et al., 2015; Cervellati et al., 2017; Depetris-Chauvin and Weil, forthcoming; Kudamatsu et al., 2016) or elsewhere (e.g., Barreca, 2010; Hong, 2013; Venkataramani, 2012). Third, a rapidly growing body of research has demonstrated negative influences of adverse prenatal conditions on human capital stocks (e.g., Almond, 2006; Almond and Mazumder, 2011).

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

2 Channels from war to infant mortality

The conceptual framework underlying the subsequent empirical analysis can be schematically summarized as occurring through the following series of events: "fighting (subsection 2.1) ⇒ an increase in malaria infection risk (subsection 2.2) ⇒ infection of pregnant mothers and their children with malaria (subsection 2.3) ⇒ an increase in mortality of weak infants associated with a compromised health system (subsection 2.4)." This section reviews literature relevant to each of these stages (see also the supplemental appendix S.1 for more details).

2.1 Liberian civil war

Liberia's political unrest encompassed two civil wars between 1989 and 2003, with the first war being fought between December 1989 and July 1997 and the second between April 1999 and August 2003. The 14-year state of chaos and fear is believed to have led to the deaths of more than 250,000 soldiers and civilians, that is, 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 fighting and human rights abuses (e.g., killings, looting, property destruction, rape, child recruitment), approximately 500,000 people are estimated to have been internally displaced and another 780,000 had to seek refuge abroad during the war.[3] Many factors (e.g., limited access to education and health services, severing of family and community ties, concerns over gender-based violence, and absence of job opportunities) discouraged the displaced populations from returning to their homes after the end of the war. It consequently took more than three years for the majority of the internally displaced persons (IDPs) to return or settle elsewhere once the Accra Peace Agreement was signed in 2003. 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). 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 considered 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). Although 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).[4]

The risk of malaria infection is expected to greatly increase due to conflict. To avoid areas of military operations, 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 face difficulty when engaging in typical risk-reducing strategies, such as staying inside the 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 civil war, fleeing populations often hid in swamps, bushes, and mountains for days, weeks, and even months in response to gunfire, government warnings, assault rumors, and surprise attacks (Foster et al., 2009).

[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]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 times of crisis are fundamentally the same as those typically observed in developing countries (e.g., diarrhea, respiratory infection, and malaria), although the conflict leads to great increases in such risks. In emergency settings, malaria control is one of the most important health-related policy concerns (WHO, 2005).

Concealment was usually followed by internal displacement within Liberia and/or refuge in other countries. This cycle of fighting, hiding, and relocating was being continuously 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 contributed to and enhanced the incidence of malaria.

2.3 Malaria infection of pregnant mothers and children

The present study particularly focuses on an increase in malaria infection risk that must have affected both pregnant mothers (and thus, fetuses) and their children. It is well acknowledged that children under five years of age are at great risk of malaria infection. As estimated in Black et al. (2003), for example, malaria accounts for approximately 25% of deaths in children younger than five years in Profile 2 countries (e.g., Guinea, Sierra Leone) characterized by them.

Among adults, pregnant women are presumed to be at considerably high risk of contracting malaria.[5] Based on Desai et al. (2007), at least one in four women in malaria-endemic areas of Africa is estimated to be infected with the disease at the time of childbirth. Although adult females may be asymptomatic owing to immunity acquired in childhood, their immune systems still weaken during pregnancy, particularly for primigravidae (Schantz-Dunn and Nour, 2009).

It is widely accepted that pregnant women infected with malaria are prone to various adverse perinatal outcomes, including miscarriage, stillbirth, intrauterine growth retardation, premature delivery, and low birthweight (e.g., Uneke, 2007b). Among these, low birthweight (i.e., a birthweight less than 2,500 g) is one of the most important factors affecting neonatal and infant deaths.[6] Maternal malaria infection is believed to reduce the birthweight of newborns by affecting gestational length and/or causing fetal growth restriction (or a combination of these factors). Low-birthweight children are vulnerable to various infectious diseases, such as pneumonia, diarrhea, and malaria, all of which increase the infant mortality risk (e.g., Lawn et al., 2005; Liu et al., 2015).

2.4 Influence of health system impairment on weak infants

The wartime impairment of the country's health system was expected to increase the aforementioned mortality risk attributed to malaria that struck pregnant mothers and their children. Due to the near-total destruction of

[5] Other high-risk groups include HIV/AIDS patients, non-immune migrants, mobile populations, and travelers (`http://www.who.int/malaria/areas/high_risk_groups/en/`).

[6] 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 be caused through the materno-fetal circulation or direct penetration through the chorionic villi. It was previously presumed that congenital transmission was rare, but more recently, an increasing number of cases have been reported (e.g., Menendez and Mayor, 2007).

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 by the end of 2003 (Lee et al., 2011). Furthermore, the number of public health sector employees decreased from 3,526 in 1988 to 1,396 in 1998; many doctors and nurses died or fled during the conflict (Varpilah et al., 2011). Nine out of 10 doctors were estimated to have left the country (Downie, 2012), leaving only 30 physicians to serve a population of three million at the end of the war (Kruk et al., 2010). The brutal conflict also deprived training institutions of the appropriate resources required to train health care workers (Duale and Mataya, 2007). While some humanitarian aid agencies provided health services during the fighting, most of the population, especially that in the rural areas, had little or no access to such services (Kruk et al., 2010; National Transitional Government of Liberia, 2004).

While limited evidence is available, the wartime chaos might also have 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. Based on the author's field interviews, conducted with Liberians in the Buduburam refugee settlement in Ghana, pre-war access to formal health services was limited in remote areas and people usually took traditional medicines (e.g., herbs) to self-treat a malaria infection.[7]

3 Empirical strategy

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

This study alternatively attempts to estimate the influence of war on children born to mothers residing in malaria-endemic areas at the beginning of the conflicts. Compared with pregnant mothers and their children hiding in and/or escaping from less-endemic areas, those doing so in and/or from disease-prone areas must have faced greater exposure to infection risk. Moreover, during wartime, newborns must have suffered due to the destruction of the country's health system; this problem can be assumed to be more serious in areas of high malaria transmission than in those of low transmission for two reasons. First, children from the former area had greater malaria infection risk due to its high endemicity. Second, children from the former area were also expected

[7] 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 for approximately 30 minutes each. Information was collected regarding the respondents' brief life-history since they left Liberia and their preventive and treatment strategies against malaria before, during, and after the war.

to have low birthweights due to their mothers' infections during pregnancy, and therefore, were more vulnerable to numerous deadly infectious diseases (including malaria). This study jointly examines these assumptions and the related mortality consequences while estimating an ITT effect of malaria infection risk in both pregnancy and infancy, which increased in the mothers' original places of residence during the war.

3.1 Specification

To estimate the aforementioned effect, this study primarily uses data drawn from two rounds (2007 and 2013) of the Standard 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 allows different units of analysis (e.g., household, women, and children), the main empirical analysis focuses on data regarding the respondents' previous children, that is, the mother's full history of births (up to 20 entries).[8] 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 that is equal to one if the month s in the year t of conception belongs to the period 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[9]; 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 similarly affected mortality between areas of high and low malaria risks, such as economic sanctions (e.g., Cortright et al., 2000, pp. 189—193).

While the mortality consequences may stem from, or be potentially entirely driven by, maternal malaria infection and the resulting fetal growth retardation, one primary goal of this study is to identify the total effects of malaria

[8] In the data set exploited in this study, births more than 17 were not recorded.
[9] Information on respondents' ethnicity is not available in the Liberian DHS data.

infection risk affecting both pregnant mothers and their children. Compared to exploiting an indicator equal to one if the child was "born" after the outbreak of the war as a key explanatory variable, focusing on the timing of conception w_{ikj}^{st} enables the estimated mortality effects to include the influence of malaria infection in pregnancy.[10][11] Given the large-scale violence observed in Liberia, the benchmark specification also attempts to estimate the war's average mortality consequences for the whole country, which may still vary by malaria infection risk. The DHS data contain information on birth year and month for every child born to a female respondent. Presuming that a mother became impregnated nine months before delivery, this study utilizes the estimated month of conception. In areas of high malaria transmission in Africa, the immunity acquired by women 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 time of conception.

While the periods following the outbreak of the war can be divided into four categories (first war, ceasefire, second war, and post-war), this study avoided this disaggregation for two reasons. First, the end of the war did not necessarily indicate the immediate recovery of the economic and relevant health situations (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 in the supplemental appendix S.3.3.

Regarding the malaria infection risk, this study uses an index that ranges from zero to one sourced from the Malaria Atlas Project (Gething et al., 2011). 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.[12] 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 on this endemicity map.

Exploiting the 2010 endemicity levels in the respondents' present residential community requires three assumptions. First, the estimated endemicity is an appropriate proxy for malaria infection risk. Second, malaria endemicity has not changed noticeably over the last two decades. This second assumption is needed because data on the endemicity levels during the pre- and inter-war periods was not available. On one hand, Liberia's "local" conflict

[10]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 thus, are vulnerable to several infectious diseases, including malaria. In this case, the influence of an infant's own infection would include effects of maternal infection.

[11]While the results are not reported, this study also replaced w_{ikj}^{st} with an indicator equal to one if the child was born after the outbreak of the war. The estimated mortality effects were attenuated compared to the case using the w_{ikj}^{st}. Moreover, simultaneously exploiting both w_{ikj}^{st} and the alternative indicator (interacted by malaria endemicity) highlighted the significance of the timing of conception (so, in-utero exposure to malaria infection risk) more pronouncedly than that of birth (so, postnatal exposure).

[12]More precisely, this index averages a monthly proportion of children aged 2—10 years who are infected with $P.falciparum$ in the general population 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/`).

experience in the last decade of the 20th century may not significantly affect Gething et al. (2011)'s "global" endemicity estimates relying on the *Plasmodium falciparum* parasite rate (PfPR) surveys conducted between 1985 and 2010 in many countries not limited to Africa. On the other hand, the situation of malaria control 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. However, based on Noor et al. (2014) (Figure 6), the mean PfPR in Liberia was almost identical between 2000 and 2010. In addition, this global concerted campaign of malaria control only reached peak intensity after 2010 (Bhatt et al., 2015).[13] Nevertheless, in subsection 5.3.3, this study discusses that the violation of these two assumptions results in underestimation of the mortality effects.

Third, all the populations displaced during the conflict returned to their original places of residence after the war (although this assumption allows for the possibility that they resettled elsewhere in Liberia but were not widespread enough to invalidate the empirical analysis). This assumption will be assessed in subsection 5.3.5.

In contrast to standard nonlinear models such as logit and probit, the LPM enables the coefficients on the interaction term to have a straightforward interpretation (Ai and Norton, 2003). To avoid a censoring problem, the analysis also utilizes 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]

3.2 Identification

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 DID estimation is that in the absence of the conflict, infant mortality for children conceived in both disease-prone and remaining areas would have followed parallel trends.

Utilizing 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 outbreak of the first war (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). The post-1977 fraction was presented in the figure because the annual number of children conceived before 1977 was minor (less than 100);

[13]Relatedly, the Liberian Government established a National Health Policy in 2007 that planned to deliver a free basic package of health services to citizens, including services intended to control communicable diseases such as HIV/AIDS and malaria. However, the corresponding report of the 2007 DHS (p. 158) refers to a huge gap between the national target considered within the national malaria policy and the present coverage of relevant interventions. Significant improvement in these initiatives appears to take more time in this country (Kruk et al., 2010; Lee et al., 2011).

thus, the estimated mortality rate is likely to be imprecise.

First, a remarkably similar trend of infant mortality was observed between the malaria-endemic and the remaining areas both before and after the outbreak of the war. Based on a more formal test described in the supplemental appendix S.2, the mortality trend before the fighting began was not significantly different between the high and low malaria-endemic areas. This finding makes the key identification assumption of the DID approach greatly trustworthy.

Second, while the pre-war mortality rate of children conceived in malaria-endemic areas was lower than the corresponding rate in less endemic areas, after the outbreak of the war, this difference disappeared. This finding may suggest unfavorable health consequences of wartime pregnancy in high-infection-risk areas; the present study aims to identify these consequences.

In addition to these main findings, the fact that, before the war, the one-year mortality rate in areas of low malaria endemicity was higher than in those of high endemicity is worth repeating. This is possible, presuming that the influence of maternal malaria infection is significant. In other words, the immunity acquired by women by the time of their pregnancy tends to be weak if they have grown up in low-malaria-transmission areas. In such areas, malaria infection during pregnancy is believed to cause 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 have made at least some progress in improving maternal and child health in Liberia, 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, and more likely, selective omission of child deaths from the data set may also explain the downward trend, as noted in the 2007 and 2013 DHS reports. Two major reasons cause this omission: 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 omission mechanisms lead to the underestimation of the mortality rate during the war.[14]

[14]The gradual decline in mortality levels can also be attributed to such wartime deaths not recorded in the DHS beginning with the weakest mothers and infants and slowly moving to stronger ones.

[Here, Figure 2]

4 Data

This study primarily uses the repeated cross-sectional data drawn from the Standard DHS 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).[15] The interviews were conducted from December 2006 to April 2007 for the 2007 survey and from March to July 2013 for the latter round.

A two-stage sample design was used in both rounds. The first stage of the respective round selected communities (clusters) from enumeration areas of the 1984 and 2008 Population Census; in the second stage, 25 (2007 round) and 30 (2013 round) households from each community were systematically sampled. The survey team aimed to interview all women aged between 15 to 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. Utilizing the history of children born to all the female respondents (up to 20 births), the current research considers the children to be the main analytical unit.

4.1 Immunity, infection risk, and mortality

As Figure 2 demonstrated, before the war, the one-year mortality rate in low endemic areas was higher than in high-endemic areas; this finding highlighted the weak (strong) immunity acquired by mothers who grew up in the former (latter) areas and its significance in determining infant mortality. Before providing summary statistics, this subsection discusses in more detail the relationships between mothers' immunity, infection risk (measured by malaria endemicity), and infant mortality because this work may facilitate an understanding of the effects this study aims to identify.

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. Two findings are notable. First, the relationship between endemicity and mortality is non-monotonic for both sampled periods. This is expected because at very low endemicity levels, the infection risk is too low to increase 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

[15] Data and relevant documents are publicly available at `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.

mortality even if the infection risk is high. The mortality rate is consequently the highest for intermediate levels of endemicity. Notably, while the relationship between "levels" of infection risk and mortality is non-monotonic, "changes" in risk and mortality do not necessarily have non-monotonic relationships. As seen in the subsequent empirical analysis, the increase in war-induced infection risk increased 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, which shows that mortality levels have declined over the last two decades in both high and low malaria-endemic areas. 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 the two groups characterized by the endemicity of *P.falciparum*.[16] The sample was separated 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).

Before proceeding with the empirical analysis, a few observations are worth noting. First, before the war, the one-year mortality rate in areas of low malaria transmission was significantly higher than the corresponding rate in malaria-endemic regions; however, this difference disappeared after the war began. Second, in both high and low endemic areas, mortality rates have reduced over time. In addition to these observations, which are consistent with the findings illustrated in Figure 2, malaria parasites were more prevalent in rural (and likely forested and mountainous) areas of high altitude. These findings are intuitive because such environmental characteristics aid the parasite's development.

The present study also used 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,

[16] In Table S.1 in the supplemental appendix, an attempt was also made to assess 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, and the resulting DID estimates were reported.

malaria-endemic areas show lower temperatures with lower 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 obtained from the Armed Conflict Location and Event Database (ACLED).[17] ACLED is a well-known data set used in the scientific analysis of conflict (e.g., Dabalen and Paul, 2014); it contains information regarding 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 are recorded in this data set (see Figure S.1 in the supplemental appendix for battle locations). As revealed in the summary statistics, the fighting occurred more intensively in less malaria-endemic areas at lower altitudes (more urban land). This information will be utilized in the empirical analysis explained in subsection 5.2.

[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 malaria endemicity value, 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 malaria endemicity experienced significantly increased one-year mortality rates by approximately 4—5%. The estimation in column (b), which alternatively used an indicator for the highest 50 percentiles of malaria endemicity, also revealed a 3% increase in the mortality rate in the corresponding areas. Thus, it can be determined from this estimate that the war 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.[18]

It is difficult to speculate on this magnitude with respect to the number of infants killed due to a malaria infection during the war and to find estimates comparable to this elasticity. However, the impact size is non-negligible, presuming that the estimated coefficient does not capture the complete effect of malaria infection.

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

[18] This elasticity is obtained as follows. As the one-year mortality rate of children conceived before the war was approximately 17% based on the examined data, the three-percent increase in infant mortality accounts for about 17.6% of the pre-war mortality. Furthermore, the estimated mean proportion of the infected population aged 2—10 years (i.e., malaria endemicity index values) is about 0.34 and 0.49 in areas belonging to the lowest and highest 50 percentiles of malaria endemicity, respectively. Therefore, combining these numbers with the 17.6% change in the mortality rate gives the above elasticity.

Because not all mothers and infants residing in malaria-endemic areas contract the disease, the treatment effect of malaria infection is likely to be greater 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. Utilizing the continuous measure also confirmed that after the war, children conceived by mothers residing in malaria-endemic areas more evidently died within one year after birth compared to those conceived by mothers in low endemic areas.

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

In the estimation results reported in column (e), community-specific time trends were additionally included in regressors. Given the decline in the mortality rate following the outbreak of the war shown in Figure 2, the time trends were considered in a quadratic manner, resulting in inclusion of the years of conception (a continuous variable) and the squared values multiplied by indicators for each community. The estimate is close to that in column (b) and statistically significant even in this demanding specification.

The statistically significant impacts on three-year and five-year mortality were also reported in columns (f) and (g), respectively. Notably, as the estimated effects are similar to those on one-year mortality, this finding suggests that infant deaths primarily occurred during the 12 months following childbirth. To further assess the lengths of child survival, the specification (1) was also estimated for the values of Z varying from one to 12; Figure 4 reports the estimated α_2 with 95% confidence intervals. The analysis produced two interesting findings. First, the unfavorable effects of wartime pregnancy in malaria-endemic areas gradually increased as longer survival periods were studied. Second, the mortality effects became more statistically significant as the examined survival periods grew longer than six months.

Children are presumably vulnerable to malaria and other infectious diseases. This applies particularly to those born to a mother infected with malaria, because these children are expected to have low birthweights. Although antibodies that have passed from a mother to the fetus through the placenta still protect newborns, 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 in association with the increasing vulnerability of such (likely low-birthweight) children.

Further robustness checks were also conducted. First, in Table 3, this study additionally included interaction terms between w_{ikj}^{st} and numerous geo-coded variables relevant to climatology, landscape, and soil quality in the vicinity of each DHS community (see the supplemental appendix S.6 for details), along with a community's GPS-based coordinates. Only the relevant coefficients are presented in this table. The "WorldClim - Global Climate Data" provided information on mean monthly precipitation amounts (mm) and temperatures (multiplied by 10 °C) from 1950 to 2000 with a spatial resolution of 30 seconds ($\approx$ 1 km^2), with longitude/latitude in a raster format (Hijmans et al., 2005). In addition to information on each community's elevation (m), which was directly available in the DHS data, this study also obtained 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 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").[19] As the results in Table 3 show, the inclusion of (time-invariant) variables relevant to climatology [column (a)], landscape [column (b)], soil quality [column (c)], and GPS coordinates [column (d)] almost did not affect the previously identified effects of wartime malaria infection risk. Although these results have not been reported due to space restrictions, simultaneously including all these geographic and climate controls also left the obtained implications unchanged.

By utilizing probit and Cox proportional hazard models, it was also assessed whether the aforementioned findings were robust to non-linear empirical models (and controls). This study also examined whether exploiting alternative estimates of malaria endemicity provided by Bhatt et al. (2015) yielded similar findings to those relying on Gething et al. (2011)'s endemicity estimates. The corresponding estimation results, as described in the supplemental appendix S.3.1 and S.3.2, also supported the previous findings.

[Here, Table 2, Table 3, and Figure 4]

5.2 Heterogeneity

While the Liberian civil war was a nationwide state of emergency, the unfavorable mortality effect may still be more pronounced in areas that were more frequently affected by the battle events. In addition, the mortality effect may

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

also be more significant in rural rather than urban areas due to possible high infection risk and/or limited access to health services (e.g., few health facilities, dirt roads difficult to traverse in rainy seasons). Moreover, considering that malaria transmission positively correlates with relatively predictable precipitation patterns (Stanley C. Oaks et al., 1991, pp. 217—218), seasons of conception may also yield different mortality consequences.

In addition to these environmental factors, (maternal) parity- and age-dependent immunity as well as the oft-cited biological "weakness" of male infants relative to females (e.g., Waldron, 1983) may also make the mortality effects heterogeneous. More precisely, women acquire parity-dependent immunity; therefore, it is observed that first- and second-time mothers are at greater risk of contracting malaria than multigravidae (e.g., Desai et al., 2007; Schantz-Dunn and Nour, 2009; Uneke, 2007a). Young maternal age also independently increases the risk of infection due to the acquisition of age-associated immunity.

The relevant exercises to explore these possibilities were conducted in the supplemental appendix S.4 and a significant mortality effect was more evident for infants conceived in the rainy season by young mothers residing in rural and war-torn areas. On the other hand, 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 had not experienced normal deliveries and/or who had died early in infancy and were thus not reported to the DHS team. Similarly, if boys are more vulnerable than girls, the obtained "no gender-difference" result may suggest that the identified mortality consequences are underestimates of the total population effect. This may be attributed to the possible failure of this study to consider the mortality effect on boys who were not a result of normal pregnancy and/or who died shortly after birth and therefore were not recorded in the DHS data.

5.3 Selection issues

This section discusses five important selection issues for the causal identification.

5.3.1 Selection into wartime fertility

In malaria-endemic and the remaining areas, unobserved parental characteristics (e.g., health safety) might have affected wartime childbearing decisions in different ways. To control for all such time-invariant characteristics, first, this study controlled for mother-fixed effects and then, re-estimated the specification (1) in column (e) in Table 3. The obtained implication is robust to this specification.[20]

[20]It is significant to note that, of the 13,260 mothers of children used in the estimations in Table 3, approximately 80% had multiple children; therefore, exploitation of the within-mother variation of data is less likely to unnecessarily produce selection bias attributed to unobserved characteristics possessed by these mothers that may differ from those held by mothers having a single child. On the other hand, approximately 27% of the mothers having multiple children delivered babies both before and after 1990 (recall that the first war began in December 1989). Thus, the mortality effects can be identified by using data relevant to these mothers.

Second, whether fertility trend varied with malaria endemicity was also assessed. An ordered probit model was estimated for the total number of children born to female respondents; the relevant estimates (not marginal effects) are presented in Table S.8 in the supplemental appendix.[21] The ordered probit model suits an analysis of fertility and is often seen in demographic literature. Note that, in this exercise, female respondents, rather than children born to these respondents, were considered the units of analysis. Furthermore, respondents included those who had never given birth, in which case, the number of children was recorded as zero. Since the DHS data did not reveal the interviewed women's parental characteristics, these exercises alternatively used several controls evaluated at the point when the respondents were children. Therefore, the estimations controlled for the number of respondents' siblings who had passed away and those still living when the respondents were 10 years old as well as the respondents' birth order.[22]

Compared with females born in and before 1965 (reference group), those belonging to the subsequent birth cohorts increasingly tended to have fewer children, as observed from the significantly negative coefficients on the birth-cohort dummies. However, as the interaction terms between the cohort dummies and malaria endemicity largely yielded insignificant coefficients, this tendency equally affected both malaria-endemic and the remaining areas. It consequently appears that no systematic difference was observed due to malaria endemicity for the wartime fertility decisions.

Nevertheless, coefficients on those interaction terms are mostly negative in columns (a) to (f) in Table S.8. Furthermore, based on p-values reported at the bottom of the table, the null hypothesis that all coefficients on those interaction terms were zero was sometimes rejected, while suggesting that fewer children were born in malaria-endemic areas due to the war, compared to those in the remaining areas. As mentioned previously, mothers tend to not report pregnancies that resulted in miscarriage, stillbirth, and deaths early in infancy to the DHS team. Therefore, the findings may refer to the likelihood that in wartime, such unsuccessful pregnancies more frequently occurred in areas with high malaria infection risk rather than in areas with low risk. This possibility causes the underestimation of the mortality effects in the DID approach.

[21] The estimated positive (negative) coefficients in this model suggest that the variables reduce (increase) the likelihood of having no children while increasing (decreasing) the probability of having many children. In other words, the variables characterized by the positive (negative) coefficients shift the distribution of fertility toward the right (left).

[22] The number of deceased siblings is included to correspond with the assumption that the mortality information may positively correlate with the respondents' poverty status in childhood. Considering 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 her siblings.

5.3.2 Selected mortality on mothers' acquired immunities

As indicated from Figure 2 (pre-war infant mortality) and Figure 3 (non-monotonic relationship between malaria endemicity and infant mortality), women originating from areas of low (high) malaria endemicity have weak (strong) immunity to malaria. Therefore, the birth outcomes tend to be more (less) serious for mothers residing in less (more) malaria-endemic areas, yielding the possible underestimation of the mortality effects.[23] The aforementioned estimation result using the mother-fixed effects, which would control for mothers' acquired immunities, mitigates this concern.

5.3.3 Selected infection risk on human immunities and global malaria control

Nevertheless, people's acquired immunities may also make Gething et al. (2011)'s endemicity estimates underestimate true infection risk in areas of high malaria transmission because the estimates are the proportion of the infected populations. Other considerations also suggest this possibility. For example, people living in malaria-endemic areas tend to have sickle cell genes that enhance their resistance to malaria (e.g., Depetris-Chauvin and Weil, forthcoming; Piel et al., 2010) and thus, the strong genetic immunities may reduce the infection probability in such areas. In addition, strenuous effort expended to target malaria-endemic areas in the Roll Back Malaria Initiative might have reduced the infection probability in such disease-prone areas more pronouncedly. If all these concerns are true, the adverse health consequences of malaria infection risk would be underestimated.[24]

5.3.4 Mothers' health and its long-term selection on the war

In the long term, the immediate mortality effect may enable only those who are genetically strong or in good health at conception and/or during their maturation process to survive until the present. If these war survivors give birth to "strong" children and this tendency differs by malaria endemicity, the mortality effects may be biased in the previous analysis exploiting data on children born to the DHS respondents.

By focusing on female respondents as the units of analysis (rather than children born to the DHS respondents, as in the mortality equation) and by replacing the outcome of specification (1) with the respondents' height-for-

[23] Related to this point, as the war extended, malaria parasites might have been transmitted from high to low endemic areas (e.g., from rural immune to urban non-immune populations) due to substantial migration, thus 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. The presence of such spillover effects may be investigated by comparing the changes in mortality for children conceived before and after the outbreak of the war between war-ridden, 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 periods of investigation.

[24] To see this, assume the following empirical model: $y = \alpha m + \epsilon$, along with $m = r\gamma m^* + (1-r)m^*$ and $E(\epsilon) = E(m^*\epsilon) = 0$, whereby $\gamma < 1$, m^* is the true level of malaria infection risk, and r is a proportion corresponding to people residing in areas of high malaria infection risk. A standard OLS algebra yields $\text{plim}_{n\to\infty}\ \hat{\alpha} = \alpha(1 + r(\gamma - 1)) < \alpha$.

age (z-scores) at the time of the survey, similar DID estimations were performed, as detailed in the supplemental appendix S.5. The estimation results in Table S.9 in the supplemental appendix showed a "positive" relationship between in-utero and/or postnatal exposure to wartime malaria infection risk and the respondents' present health status in a statistically significant manner, suggesting that the war-induced culling of the weakest infants existed and was more significant in malaria-endemic areas.

However, as described above, the estimated mortality effects were robust to the inclusion of mother-fixed effects, which would control for the respondents' unobserved characteristics relevant to their survival. Moreover, if the "strong" mothers in malaria-prone areas deliver "strong" children, this concern would make the DID estimates in the mortality equation attenuated.

5.3.5 Selected resettlement on infection risk

As the respondents might have originally resided in locations different than their current DHS communities, the measured malaria endemicity includes some noise that may be correlated with infant mortality. If this noise systematically differs by malaria endemicity, this is another source of bias. To determine the sensitivity of previous findings toward this concern, three exercises were performed.

First, this study utilized data pertaining only to children born to those mothers who were identified as permanent residents of the surveyed community in the 2007 DHS and re-estimated the specification (1) in column (f) in Table 3. Based on the 2007 DHS, approximately 55% of the respondent females were identified as permanent residents of the surveyed community (corresponding information was unavailable in the 2013 DHS). Notably, this does not necessarily mean that the remaining females currently live in places far away from their wartime residential places because those females include two groups: (1) young respondents who must have recently relocated into the surveyed community in the vicinity of their birthplaces owing to (widely observed) patrilocal marriage and (2) those who were not permanent residents but who had settled in the DHS community before the war (again, due to the patrilocal marriage, for example). The main findings of this study are robust to utilizing this sub-sample.[25]

Second, during the conflict, most IDPs headed toward the capital, Monrovia (Nilsson, 2003). Moreover, after the war, Liberian refugees were repatriated to the capital under a program run by the UN refugee agency. However, in the post-war periods, some of those people preferred to stay in the capital instead of returning to their original

[25] Admittedly, limiting analytical attention to sub-samples may introduce selection bias owing to the endogenous selection of residential places. In the present context, the permanent residents might have resided in less conflict-affected areas. However, constructing an indicator for children conceived by mothers identified to be permanent residents and regressing this indicator 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 districts) yielded a coefficient 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 indicator.

homes, and thus the current residents of the capital area must have shown a great tendency to have resided in different places during the years of the conflict. This is particularly true if none of their relatives lived in their original communities as a result of the war (Jesuit Refugee Service, 2007; Omata, 2012). Given this likelihood, another exercise conducted in column (g) in Table 3 excluded those currently living in the Greater Monrovia District from the estimated sample. This additional analysis also yielded similar implications to those obtained previously.

Third, to see if the post-conflict resettlement systematically changed population characteristics between areas of high and low malaria transmission, this study appended the 1986 Standard DHS data (5,239 respondents in 156 communities) to the 2007 and 2013 DHS data and compared a community's demographic characteristics (i.e., age, education, religion, marital status, fertility, household size), which may have some correlation with infant mortality, before (1986 DHS) and after (2007 and 2013 DHS) the war between areas of high and low malaria endemicity. These characteristics are measured by the mean of the respondent females in a community and the relevant DID estimates are reported in columns (a)—(f) in Table S.10 in the supplemental appendix. As seen from the interaction terms between the post-conflict dummy, which takes one if the community was surveyed in either 2007 or 2013 DHS, and malaria endemicity, the war-induced significant reduction in household size (partly due to high infant mortality) [column (f)] and no impact on fertility observed in malaria endemic areas [column (e)] are consistent with the previous findings. As a whole, the interaction terms yielded insignificant coefficients for most outcomes.

Relatedly, recall that the large-scale violence in Liberia encouraged this study to avoid using the within-country variation of battle intensity in the benchmark specification and to estimate the war's average mortality consequences for the whole country, which may still differ by malaria endemicity. If a systematic difference with respect to changes in the population characteristics attributable to the post-conflict resettlement exists based on battle intensity rather than malaria endemicity, this benchmark specification may suffer less from this resettlement issue.[26]

Finally, a study conducted by Acemoglu et al. (2014) in Sierra Leone may also mitigate the concern that the post-conflict resettlement makes the positional information on the DHS communities useless in the present analysis. Using the post-conflict DHS data including only information on the respondents' present communities as well as other data including information on their places of birth, Acemoglu et al. (2014) provided a coherent story of

[26]Related to this point, malaria infection risk may cause violence (e.g., Cervellati et al., forthcoming). However, regressing the number of battle events that occurred within a 25-km radius from a community on a community's malaria endemicity with controls of its temperature and precipitation as well as fixed effects of the DHS round and local political units (called clans) yielded insignificant coefficients. On the other hand, the temperature and precipitation had positive correlation with the incidence of battles with strong statistical significance, as indicated in Cane et al. (2014) and Hsiang et al. (2013), for example.

interest, suggesting that the DHS respondents returned to their original residential places after the civil war (1991—2002) involving the large-scale population displacement and the post-conflict resettlement. Considering this neighboring country's similar experience of the war, this implication may be true even in Liberia.

5.4 Assessment of bias attributed to unobservables

By considering three statistical approaches and reporting the relevant empirical findings in Table 4, direction of bias possibly included in the previous estimates is examined in this subsection. To be compared with the results obtained from these approaches, the DID estimates reported in columns (b)—(c) in Table 2 are copied and pasted to panel (A) in Table 4. The relevant full estimation results corresponding to panel (C) and panel (D) are reported in Table S.11 in the supplemental appendix.

First, following Oster (forthcoming), this study evaluated the relative importance of omitted variables that share covariance properties with observed controls and that are required to explain the identified effects. This importance is denoted as δ, i.e., a coefficient of proportionality on selection assumptions. This approach needs to assume the value of R-squared that is 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 (forthcoming) 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 as the R_{max} in row (1) in panel (B) in Table 4. In theory, the value of R_{max} should reflect the extent to which including both the observed and unobserved controls (that have a proportional selection relation) and the treatment explains outcome variation. 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 corresponding estimation of column (e) in Table 3, this study alternatively used $R_{max} = 0.3$ as the second possibility in row (2). In row (3), 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.[27] As a negative δ indicates that including the unobserved controls in regressions increases the magnitude of the estimated effect rather than absorbing the effect size, these results suggest the attenuation of the DID estimates reported in panel (A).

The second approach uses the insight from a regression discontinuity design. Assuming a nearly identical influence of any confounding factors 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 and 1990 (given that the first war began in

[27] This study assumed that the fixed effects of the year-of-conception (ρ_t) and communities (v_j) are proportional to unobservables.

December 1989). The estimation results on the (local) mortality effect are reported in panel (C) in Table 4. The estimates are greater than the OLS estimates utilizing the full sample.

Third, this study employed climate-based instrumental variables (interacted with w_{ikj}^{st}) for $w_{ikj}^{st} \cdot m_j$ and conducted two-stage least-squares (2SLS) estimations in panel (D).[28] It is widely accepted that temperature and rainfall are two critical factors in explaining malaria endemicity (e.g., Blanford et al., 2013). The relationship between these factors and malaria infection risk is potentially non-monotonic. For example, while the large precipitation increases the likelihood of mosquito survival, too much rainfall can be detrimental to mosquito longevity, because it flushes away breeding habitats and thus, eggs or larvae of the mosquitoes (e.g., Usher, 2010). Considering this point, this study constructed malaria suitability statistics for the temperature and precipitation separately, as $1 - \frac{|q - q_{\text{opt}}|}{|q - q_{\text{opt}}|_{\max}}$, whereby q is a community's temperature or precipitation; q_{opt} is optimal levels of these factors for malaria transmission, which are considered to be 22 °C and 80 mm of the monthly precipitation (e.g., Grover-Kopec et al., 2006; Small et al., 2003); and $|q - q_{\text{opt}}|_{\max}$ is the maximum value of $|q - q_{\text{opt}}|$ among all the surveyed communities. Therefore, malaria suitability increases with this statistic which ranges from zero to one.

As seen from the F-statistics in the first-stage estimations and p-values of the Hansen test reported in columns (a)—(d) in Table S.11 in the supplemental appendix, the instruments positively affect malaria endemicity with strong statistical significance and satisfy the overidentifying restriction. As the panel (D) in Table 4 reports, the estimated mortality effects are similar to or slightly greater than the OLS estimates, although their statistical differences may not be significant. Overall, all the exercises performed in Table 4 may collectively provide the view that the OLS estimates are attenuated if any bias exists.

[Here, Table 4]

5.5 Interpretation

The previous estimates should be interpreted as the total effect arising from an increase in malaria infection risk (that struck both pregnant mothers and their children) and the impairment of the health system that aggravated the unfavorable consequences of the heightened infection risk during the war. Exploiting the malaria endemicity rather than malarious weather (e.g., Kudamatsu et al., 2016) helps in assessing the direct influence of the malaria infection risk. Nevertheless, this interpretation of the estimates requires more careful discussion, as demonstrated in this subsection.

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

First, during the war, acts of sexual and gender-based violence against women and girls were exploited as weapons of warfare. This raises the issue of children born of these unwanted events being abandoned or killed shortly following birth (e.g., Denov, 2015), more pronouncedly in malaria-endemic areas. However, the current study considers this possibility as less likely for several reasons. First, such cases of infanticide have not been supported by the relevant qualitative research in Liberian contexts (e.g., Foster et al., 2009; UNFPA, 2011). Second, the fighting was "less" intense in malaria-endemic areas (see Table 1). Third, if this alternative interpretation is considered true, newborn deaths were more likely to frequently occur during the immediate months following these births. However, 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 female survivors' health, as discussed in subsection 5.3.4.

Second, if malaria-endemic areas were prone to other tropical diseases, the unfavorable consequences for infant mortality may also be attributed to these. Among "neglected tropical diseases" including hookworm, ascariasis, trichuriasis, schistosomiasis, lymphatic filariasis, onchocerciasis, and dengue, in Liberia, (only) the estimated number of people infected with schistosomiasis and onchocerciasis is comparable to those infected with malaria (e.g., Hotez, 2015). However, morbidity of pregnant women and their children attributable to the former is largely unknown (Friedman et al., 2007) and a major symptom of the latter is blindness, both of which have not been highlighted in 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).

Finally, if the malaria-endemic areas were insecure with respect to food during the war, maternal and infants' nutritional deprivation may also explain the previous findings (e.g., Abu-Saad and Fraser, 2010; Villar et al., 2003).[29] On one hand, the robustness of the estimated mortality effects to the inclusion of geographic and climate controls (i.e., columns (a) to (d) in Table 3) as well as the community-specific time trends (i.e., column (e) in Table 2) may alleviate this concern to some extent, if those controls are considered to be proxies for community-level food availability. On the other hand, this undernutrition issue also increases the risk of morbidity and mortality resulting from malaria infection; therefore, these two factors may not be mutually exclusive. Similarly, malaria infection in pregnancy during wartime may also increase maternal stress, thus further reducing neonates' birthweight (Camacho, 2008; Mansour and Rees, 2012). In these cases, the estimated mortality effects may be seen as the total effects of

[29]The pronounced mortality effect revealed in Table S.6 in the supplemental appendix for children conceived in the rainy season may also be consistent with the fact that, in Liberia, this season almost overlaps with hunger periods resulting from heavy agricultural workloads (Owadi et al., 2010, Figure 4.7) due to the frequent heavy rains, inaccessible roads, limited access to markets, food stock depletion, food price hikes, and so on; however, the relationship between mothers' dietary intake and positive or negative birth outcomes may differ according to pregnancy stages, types of nutrients (e.g., energy, protein, iron, zinc, vitamin A, folate, vitamin D), and a combination of these factors.

these forces.

6 Conclusion

In what ways does an armed conflict interrupt health accumulation? To provide one answer to this question, this study investigated whether infant mortality during the Liberian civil war increased due to pregnant mothers and their children being exposed to a higher risk of malaria infection. Two existing frameworks encouraged the examination of this question in the current research. 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 and infants are one of the groups that are highly vulnerable to malaria infection, along with maternal malaria infection that tends to cause 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 observed that a one-percent increase in conflict-induced malaria infection risk caused 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 a reference point in future research exploring the influence of such risk on infant mortality.

As maternal passive immunity waned, this negative health effect gradually increased and became more statistically significant. Moreover, infants conceived in rainy seasons by young mothers residing in rural, battle-intense areas faced more serious adverse health consequences. On the other hand, a significant gender difference for the mortality effect was not observed. The importance of this passive immunity and heterogeneous mortality consequences for infants conceived by geographically, seasonally, and (possibly) immunologically high-risk mothers may assist policymakers and practitioners in determining target groups for aid programs during inter- and post-war periods.

In this study, Liberia was selected due to its climatically suitable nature for vector-mediated transmission as well as its experience of brutal violence, which was expected to make the war-induced infection risk and the resulting mortality consequences the greatest. On the other hand, mothers' immunities acquired in childhood may also be greater in this country, compared to those of other countries and this strong immunity might have reduced the mortality consequences of the wartime malaria infection risk. Therefore, the external validity of the findings may depend upon these two conflicting forces.

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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)	1.84***	3.27	4465	2.16	3.80	4857
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 (× 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)	2.08***	3.06	21899	2.82	3.97	21628
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 (× 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 infant mortality (OLS)

Dependent variable:	One if die within Z months after the birth						
	$Z = 12$	$Z = 12$	$Z = 12$	$Z = 12$	$Z = 12$	$Z = 36$	$Z = 60$
	(a)	(b)	(c)	(d)	(e)	(f)	(g)
Conceived after the outbreak of the war							
× Malaria endemicity	0.029	-	-	-	-	-	-
(10-20 percentile)	(0.022)						
× Malaria endemicity	0.023	-	-	-	-	-	-
(20-30 percentile)	(0.023)						
× Malaria endemicity	-0.030	-	-	-	-	-	-
(30-40 percentile)	(0.027)						
× Malaria endemicity	0.015	-	-	-	-	-	-
(40-50 percentile)	(0.022)						
× Malaria endemicity	0.033	-	-	-	-	-	-
(50-60 percentile)	(0.021)						
× Malaria endemicity	0.050**	-	-	-	-	-	-
(60-70 percentile)	(0.023)						
× Malaria endemicity	0.046**	-	-	-	-	-	-
(70-80 percentile)	(0.022)						
× Malaria endemicity	0.040**	-	-	-	-	-	-
(80-90 percentile)	(0.020)						
× Malaria endemicity	0.043**	-	-	-	-	-	-
(90-100 percentile)	(0.022)						
× Malaria endemicity	-	0.035***	-	0.035***	0.031*	0.033***	0.035***
(50-100 percentile)		(0.010)		(0.010)	(0.017)	(0.011)	(0.012)
× Malaria endemicity	-	-	0.115**	-	-	-	-
(continuous measure)			(0.046)				
Male (dummy)	0.017***	0.017***	0.017***	0.017***	0.017***	0.020***	0.022***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.004)
Birth order	0.013***	0.013***	0.013***	0.013***	0.012***	0.016***	0.017***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)
Single birth (dummy)	-0.240***	-0.239***	-0.239***	-0.240***	-0.236***	-0.260***	-0.260***
	(0.014)	(0.014)	(0.014)	(0.014)	(0.015)	(0.015)	(0.016)
Mother's age at birth	-0.006***	-0.006***	-0.006***	-0.006***	-0.006***	-0.008***	-0.009***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)
Mother's education	-0.003***	-0.003***	-0.003***	-0.003***	-0.002***	-0.003***	-0.004***
(years)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Mother's experience of	-	-	-	0.011**	-	-	-
terminated pregnancy				(0.004)			
Mother's 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
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community-specific trend	No	No	No	No	Yes	No	No
R-squared	0.086	0.085	0.085	0.086	0.119	0.091	0.092
No. of obs.	49121	49121	49121	49025	49129	43985	38807

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 3: Robustness to geographic and climate controls, mother-fixed effects, and sub-subsamples (OLS)

Dependent variable:	One if die within 12 months after the birth						
Sample:	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)
Conceived after the outbreak of the war							
× Malaria endemicity	0.039***	0.035***	0.028**	0.035***	0.047***	0.038**	0.036***
(50-100 percentile)	(0.013)	(0.013)	(0.013)	(0.013)	(0.015)	(0.017)	(0.011)
× Temperature (× 10 °C)	-0.000	-	-	-	-	-	-
	(0.000)						
× Precipitation (mm)	0.000	-	-	-	-	-	-
	(0.000)						
× Elevation (m)	-	-0.000	-	-	-	-	-
		(0.000)					
× Slope (%)	-	-0.005	-	-	-	-	-
		(0.016)					
× Terrain ruggedness index (100m)	-	0.006	-	-	-	-	-
		(0.042)					
× Nutrient availability	-	-	-0.053	-	-	-	-
			(0.053)				
× Nutrient retention capacity	-	-	0.044	-	-	-	-
			(0.034)				
× Rooting conditions	-	-	0.013	-	-	-	-
			(0.023)				
× Oxygen availability to roots	-	-	-0.019	-	-	-	-
			(0.014)				
× Excess salts	-	-	-0.013	-	-	-	-
			(0.016)				
× Workability	-	-	-0.006	-	-	-	-
			(0.020)				
× Longitude	-	-	-	0.000	-	-	-
				(0.006)			
× Latitude	-	-	-	-0.003	-	-	-
				(0.008)			
Individual controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother characteristics	Yes	Yes	Yes	Yes	No	Yes	Yes
Mother's religion FE	Yes	Yes	Yes	Yes	No	Yes	Yes
Mother FE	No	No	No	No	Yes	No	No
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	No	Yes
Community FE	Yes	Yes	Yes	Yes	No	Yes	Yes
R-squared	0.085	0.085	0.087	0.085	0.318	0.114	0.087
No. of obs.	49121	48708	47091	48708	49429	11695	44243

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 4: Assessment of bias attributed to unobservables

Dependent variable:	One if die within 12 months after the birth	
Malaria endemicity:	50-100 percentile	Continuous measure
	(a)	(b)
Panel (A): Main results from Table 2 (OLS)		
Conceived after the outbreak of the war	0.035***	0.115**
× Malaria endemicity	(0.010)	(0.046)
R-squared	0.085	0.085
No. of obs.	49121	49121
	(c)	(d)
Panel (B): Oster (forthcoming)'s δ		
(1) $R_{max} = 1.3\tilde{R}$	-0.498	-0.171
(2) $R_{max} = 0.3$	-0.057	-0.019
(3) $R_{max} = 1.0$	-0.013	-0.004
	(e)	(f)
Panel (C): Discontinuity sample conceived in 1989 and 1990 (OLS)		
Conceived after the outbreak of the war	0.062*	0.225
× Malaria endemicity	(0.037)	(0.142)
R-sqaured	0.273	0.273
No. of obs.	2552	2552
	(g)	(h)
Panel (D): Instrumental variable approach (2SLS)		
Conceived after the outbreak of the war	0.034***	0.121***
× Malaria endemicity	(0.012)	(0.045)
R-sqaured	0.085	0.085
No. of obs.	49121	49121
Individual controls	Yes	Yes
Mother characteristics	Yes	Yes
Month-of-conception FE	Yes	Yes
Year-of-conception FE	Yes	Yes
Round FE	Yes	Yes
Community FE	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 (a)—(b) and (e)—(f) and to heteroskedasticity in columns (g)—(h). (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.

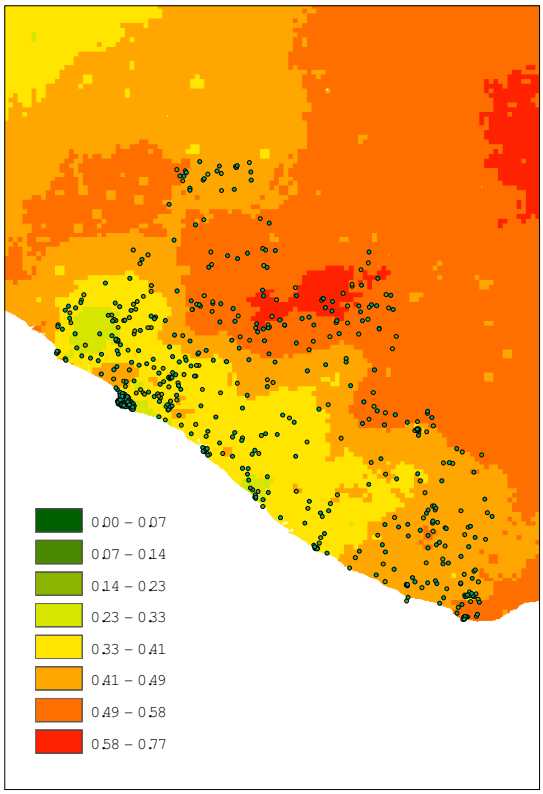

Figure 1: Malaria endemicity map (*Plasmodium falciparum*) in 2010 and locations of DHS communities

Source: Gething et al. (2011)

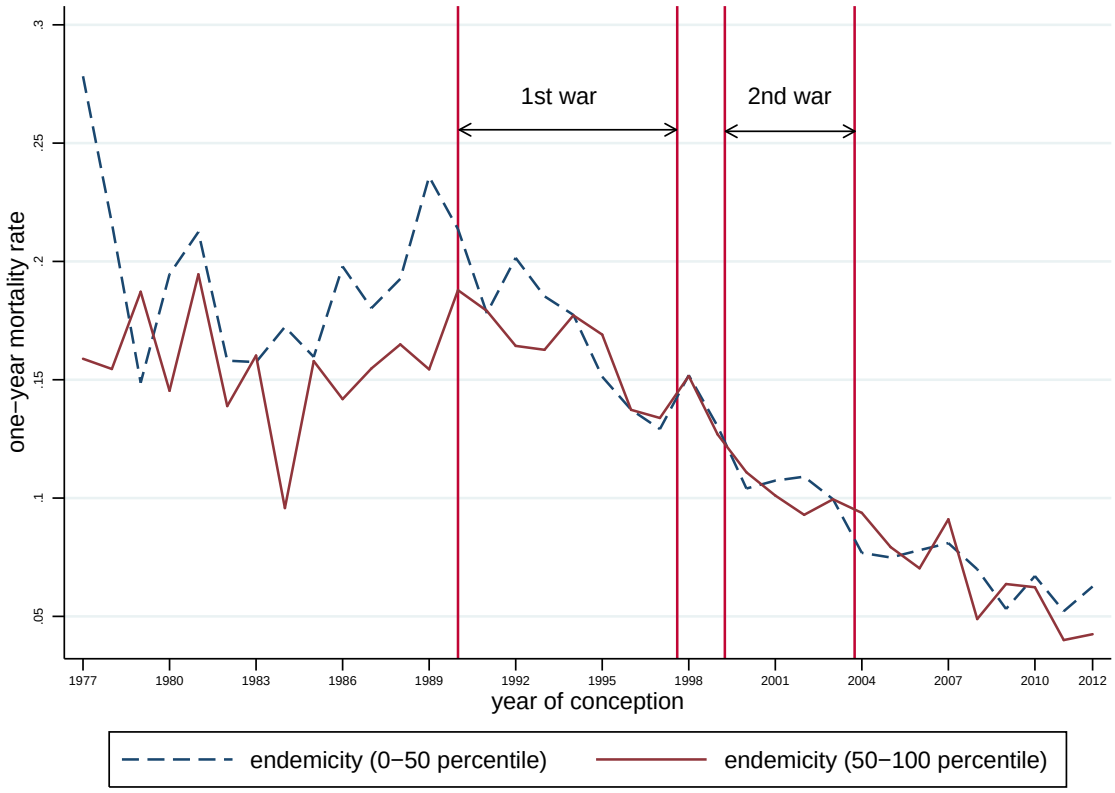

Figure 2: One-year mortality rate by the timing of conception

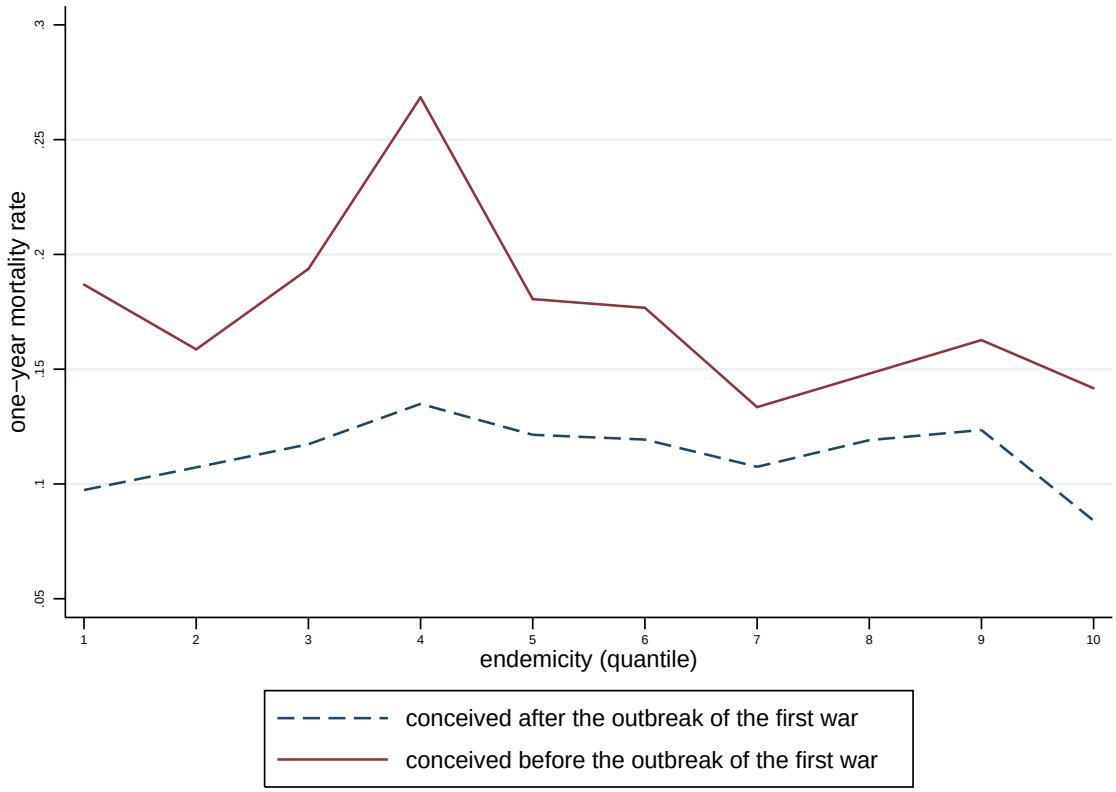

Figure 3: One-year mortality rate by levels of endemicity

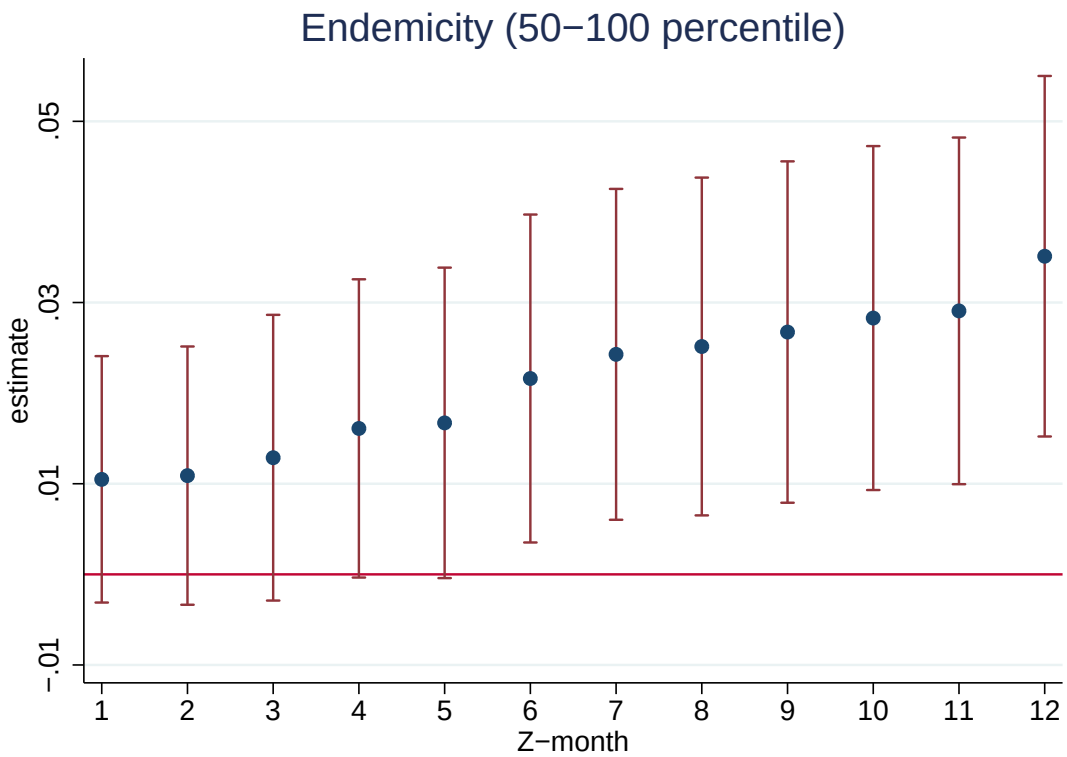

Figure 4: Impacts of wartime pregnancy in malaria endemic areas on Z-month infant mortality (OLS)

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.

Supplemental appendix

S.1 Literature review

S.1.1 Liberia and malaria suitability

Typified by the tropical heat and humidity, Liberia lies almost entirely within the rainforest zone. The landscape is characterized by swampy plains along the coast and rolling inland plateaus leading to low mountains. Most areas lie below an altitude of 500 m, thus making malaria transmission possible in almost all parts of the 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 higher than the minimum temperature (approximately 18 °C) required for development of *P.falciparum*, a major plasmodium species in the country (Patz and Olson, 2006). While the body's inflammatory response is a typical symptom of malaria, this most dangerous species also causes cytoadherence of erythrocytes to vascular walls; thus, infected blood cells are often trapped in small blood vessels, which can lead to end-organ damage or blood abnormalities. Furthermore, microcytic anemia in adults and folic acid deficiency also commonly occur due to the parasites' splenic sequestration (e.g., Schantz-Dunn and Nour, 2009).

S.1.2 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) had assumed 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 prioritized 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 a 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 Charles Taylor's leadership. This war ended 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 the country (e.g., diamonds, gold, iron ore, and timber), emerged and set the tone for the conflict that lasted 14 years. A second war commenced 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 this 14-year state of chaos and fear, more than 250,000 soldiers and civilians were killed, that is, more than 12% of the 2.1 million population in 1990 (Mama, 2014, p. 55).[30]

Due to the atrocious and indiscriminate nature of the fighting and human rights abuses (e.g., killings, looting, property destruction, rape, and child recruitment), approximately 500,000 people were estimated to have been internally displaced and another 780,000 sought refuge abroad.[31] 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 the following phases: hiding, internal displacement, and refuge in other countries. In response to the sound of gunfire, government warnings or assault rumors, 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 suffered from difficulties stemming from the displacement more disproportionately than 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 toward 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 the Accra Peace Agreement was signed in 2003, most IDPs relocated to formal camps near 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 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% of the refugees were residing in camps by 1996. As refugees began to outnumber their hosts, however, anti-refugee sentiments emerged, sometimes forcing

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

[31] 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.

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 had chosen to leave the country only after enduring several years of internal displacement and 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 in post-war periods (e.g., Wright and Savage, 2007). Therefore, the majority of IDPs required more than three years to return to their homes or settle elsewhere following the 2003 Peace Agreement.[32] Furthermore, 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).

S.1.3 Factors affecting infant mortality

Based on a recent estimate, approximately 44% of deaths in children under the age of five occur within the first four weeks following birth (Liu et al., 2015).[33] Public health research has reported the 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., $56\% = 100\% - 44\%$), respectively, of under-five mortality (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 under five years.

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

As these factors are closely interconnected, it is difficult to identify a single reason for each neonatal and infant

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

[33]The corresponding ratio was 37% in 1990; thus, an under-five deaths are now being increasingly 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 amongst major developing areas; furthermore, 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.

death. For example, preterm birth increases the newborn's difficulty in feeding and maintaining his or her body temperature and also the risk of contracting infectious diseases (Lawn et al., 2005; Lawn et al., 2010). Prematurity and in-utero growth failure or disruption also increase neonatal death risk, especially due to infection (Lawn et al., 2005). Many infants recorded as dying from infectious diseases are also premature (Lawn et al., 2010).

S.1.4 Maternal malaria infection

Among adults, it is well acknowledged that pregnant women are at a considerably high risk of contracting malaria.[34] 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), at least one in four women in malaria-endemic areas of Africa is estimated to be infected with the disease at the time of childbirth. While adult females may be asymptomatic owing to immunity acquired in childhood, their immune system still becomes weaker in pregnancy, particularly for primigravidae (Schantz-Dunn and Nour, 2009).

In sub-Saharan Africa, maternal anemia is one of the major complications of this infectious disease. Desai et al. (2007) estimated that approximately 26% of severe anemia among pregnant women can be attributed to malaria.[35] Pregnant women also suffer from a serious anemic burden due to the sequestration of the parasites in the placenta (placental malaria). According to one recent estimate, in the absence of 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 between malaria and 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; these percentages are substantially lower than the corresponding rates in eastern and southern African countries.

S.1.5 Influence of maternal malaria infection

S.1.5.1 Fetal growth disruption

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

[34] 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/`).

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

Uneke, 2007c). Among these, low birthweight (i.e., a birthweight less than 2,500 g) is one of the most important factors causing neonatal and infant deaths.[36] Based on Guyatt and Snow (2001, 2004), in malaria-endemic areas in Africa, babies born to mothers with 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 annual infant deaths in this region.[37]

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 more careful research is required to establish this theory (e.g., Guyatt and Snow, 2004; Menendez et al., 2000).

In contrast to the influence of malaria infection on prematurity, its relations with intrauterine growth retardation are better understood. Umbers et al. (2011) provided a sound pathological review on the underlying factors linking placental malaria with fetal growth restriction. For instance, placental infection is likely to impair placental development while prompting maternal hypertension and placental vascular dysfunction. Furthermore, placental inflammation attenuates the levels of those hormones that regulate placental functions. Of those hormones, some affect fetal cell proliferation, maternal appetite, metabolism, and fat accumulation. Moreover, a high parasite density in placental blood and the associated immune response may decrease nutrient levels (e.g., amino acids, lipids, and glucose) delivered to the fetus. Similarly, placental infection can also prevent the transplacental transfer of maternal antibodies and cellular immune responses to several infectious diseases (e.g., measles, tetanus) (e.g., Brair et al., 1994; de Moraes-Pinto et al., 1998). Placental changes such as the thickening of cytrophoblastic membranes may also hinder the effectiveness of nutrient transporters (e.g., Guyatt and Snow, 2004).

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).

S.1.5.2 Seasonality

This subsection discusses the seasonal influence of maternal malaria infection on fetal growth in detail; multiple factors could affect such influence, a possibility supported by the studies reviewed below.

[36] Congenital malaria (i.e., transplacental transmission of malaria to the fetus) is another possible factor linking maternal malaria infection with infant mortality. The materno-fetal circulation or direct penetration through the chorionic villi may cause congenital malaria. Previously, it was presumed that congenital transmission was rare, but more recently, an increasing number of cases have been reported (e.g., Menendez and Mayor, 2007; Uneke, 2007a). Moreover, malaria infection in pregnancy during wartime may increase maternal stress, thus further reducing the birthweight of neonates (Camacho, 2008; Mansour and Rees, 2012).

[37] Approximately 60—80% of newborn deaths are estimated to be cases of low-birthweight neonates (Lawn et al., 2005).

In Africa, birthweights are likely to exhibit seasonal fluctuations (e.g., Rayco-Solon et al., 2005). While maternal malaria infection may be only one factor underlying this observation, it could possibly exert a seasonal influence on fetal growth for three reasons. First, some gestational months may biologically reveal a higher risk of contracting the disease than 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 epidemiological literature review made by Desai et al. (2007) and others (e.g., Brabin, 1983; Rogerson et al., 2000; Walker et al., 2014; Zhou et al., 2002), infection risk appears to be 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).

In general, malaria transmission positively correlates with relatively predictable precipitation patterns, 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 vector-mediated transmission, this seasonal pattern seems to be followed even in this country. *Anopheles gambiae*, a principal anopheles species in Liberia (WHO, 2013, p. 149), is most abundant during the rainy seasons (Fahmy et al., 2015; Gelfand, 1955; Hogh et al., 1993; Somah, 2005).

Compared to the health consequences of malaria in pregnancy in general, the gestational period of infection that has the largest unfavorable effects is relatively unknown (e.g., Cottrell et al., 2007; Kalilani et al., 2010; Huynh et al., 2011). As Desai et al. (2007) asserted, 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. Although the adverse effects of infection in the first trimester are relatively unclear, it may also explain the decrease in newborns' birthweights (e.g., Huynh et al., 2011).

S.2 Parallel trend assumption

The DID approach's key identification assumption is a parallel trend of the outcomes. While Figure 2 (infant mortality) and (as explained in the supplemental appendix S.5) Figure S.3 (height-for-age) provide an informal assessment of and support for the assumption, this section statistically tests whether the pre-war trend of the outcomes significantly varied with malaria endemicity. To achieve this purpose, this study only focused on data pertaining to individuals 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

$$y_{ij} = \beta_1 + \sum_h \beta_2^h \cdot a_{ij}^h + \sum_h \beta_3^h \cdot a_{ij}^h \cdot m_j + v_j + u_{ij}, \tag{2}$$

where a_{ij}^h is an indicator equal to one if the person was conceived in year h and zero otherwise (the reference group included those conceived in and before 1979); v_j is community-level fixed effects; and u_{ij} is a stochastic error. The units of analysis are either children born to the DHS respondents or the DHS respondents themselves, depending upon the relevant outcomes. 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 as well as the respondents' height-for-age and height were presented in columns (a), (c), and (e), respectively, in Table S.2. In the estimations of the remaining columns in Table S.2, several controls used in Table 2 and (as explained in the supplemental appendix S.5) Table S.9 were included in regressors. As clarified from the table, these exercises provided no evidence undermining the parallel trends assumption in the pre-war periods.

S.3 Robustness checks

S.3.1 Non-linear empirical models

In Table S.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: children conceived before and after the outbreak of the conflict. It then independently estimated the influence of 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 S.3 (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 and 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) and included a community's geographical position (latitude, longitude) and elevation in meters in regressors.[38]

[38] A county corresponding to each community could not be identified from the DHS data alone. 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`). The communities were consequently categorized into 15 counties plus one group for which the ArcGIS failed to identify the corresponding county. The analysis in Table S.3 included the unidentified group as one county corresponding to 3% of the entire sample.

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

By employing a Cox proportional hazard model, this study also analyzed the duration (months) from birth to death in columns (h) and (p) in Table S.3, whereby the estimated hazard ratio is reported and a ratio greater (smaller) than one indicates that the variable induces (prevents) mortality.[39] Owing to the proportional hazard assumption, the ratio should be interpreted as the hazard ratio at any point in time between two individuals that only varies by one unit of covariates. Schoenfeld residual (p-values) reported at the bottom of Table S.3 failed to reject the assumption of proportional hazards. Again, the results highlight that malaria infection risk in pregnancy or infancy only decreases the survival likelihood of children conceived after the outbreak of the war (with 5% statistical significance).

S.3.2 Alternative measures of malaria infection risk

This study collected annual estimates of endemicity levels of *P.falciparum* between 2000 and 2010 from another study of the Malaria Atlas Project, Bhatt et al. (2015).[40] Despite the similar Bayesian geostatistical model and the map resolution exploited by Gething et al. (2011) and Bhatt et al. (2015), the 2010 estimates of the two studies varied. 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). This difference is partly attributed to differences in the geographical scope of these two studies and 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 conducted 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 the substantial difficulty faced when estimating endemicity in general, this study avoids a hasty judgment about which of these (or other possible) studies provides more precise estimates of endemicity.[41] Instead, these alternative estimates were used to check the robustness of the main findings. Therefore, this study estimated the specification (1) based on endemicity estimates provided by Bhatt et al. (2015) and re-

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

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

[41] Similar to the endemicity estimates of Gething et al. (2011), those based on Bhatt et al. (2015) should also be viewed as only another proxy for the pre-war levels of endemicity in the present study.

ported the estimated α_2 in Table S.4. In each panel from (A) to (K), endemicity levels sourced from different years were utilized 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. Moreover, the estimates are almost identical across panels. This finding supports the discussion in subsection 3.1 that there has been no marked change in the endemicity levels of *P.falciparum* between 2000 and 2010 in Liberia despite a global malaria control effort. 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.

S.3.3 Mortality effects trend

In column (a) in Table S.5, this study interacted the previously used indicator for areas of high malaria endemicity (50—100 percentiles) with four different periods of conception - 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, people are expected to have lower infection risk in the post-war periods. However, the results indicate significant mortality effects even after the war. This finding remained unaffected even after utilizing a continuous measure of malaria endemicity in column (b).

The ITT interpretation of mortality consequences includes the influence of limited access to pre- and post-natal care and thus, makes this finding possible. While the 2007 National Health Policy has contributed toward progress in providing basic health services, Kruk et al. (2010) noted that the government's ambitious target was unlikely to be achieved by 2010 based on their study that explored the availability of essential health services in 2008. They attempted to survey a "typical" rural population and demonstrated that only one-quarter of their respondents had access to basic emergency obstetric care and even fewer obtained the integrated management service of childhood illness.[42] The overall service quality is also low, with long travel times required to reach 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 expanded 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 owing to this increase or not, the mortality effect shows a declining tendency starting with conceptions occurring in 2009, as seen in Figure S.2. This figure further disaggregated periods following the outbreak of the first war and

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

reported the estimated α_2 (with 95% confidence intervals) specific to each year of conception.[43]

S.4 Heterogeneity

S.4.1 Environmental risk

Several environmental factors might increase malaria infection risk while further discouraging child survival. Three relevant perspectives were examined in Table S.6: battle intensity, types of residential areas, and seasons of conception. Analyses made in panels (A)—(C) in the table used an indicator for the upper 50% quantile of malaria endemicity as compared with the remaining panels (D)—(F), which used the continuous value of the index. The estimations in this table (and Table S.7) 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 that were more frequently affected by the armed conflict. Therefore, exercises conducted in the first two columns in Table S.6 split the sample in accordance with the number of battle events that occurred within a 25-km radius from each DHS community and independently estimated the specification (1) for both sub-samples. The sample median number of fighting incidents was used as the criterion for sample separation. As explained in Section 4, information regarding the battle events was sourced from the ACLED. As indicated by the estimation results in the first two columns, 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.

Considering the presumption that the mortality effect is more significant in rural rather than urban areas due to possible 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 S.6. The results supported the expectation.

Malaria transmission, in general, positively correlates with relatively predictable precipitation patterns (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 vector-mediated transmission, this picture still seems true. *Anopheles gambiae*, a principal anopheles species in Liberia (WHO, 2013, p. 149), is most abundant during the rainy season (e.g., Fahmy et al., 2015; Gelfand, 1955; Hogh et al., 1993; Somah, 2005). Based on these arguments, this study estimated the

[43] 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.

specification (1) for both sub-samples of children conceived in rainy (May to October) and dry seasons (November to April) in the fifth and sixth columns in Table S.6, respectively. The results indicated that the negative health consequences of wartime pregnancy in malaria-endemic areas were identified more clearly for children conceived in the rainy reason than for those conceived in the remaining periods. However, interpretation of the estimates may not be so straightforward, provided that malaria infection during pregnancy primarily explains the mortality effects. This is because the seasonal influence of maternal malaria infection varies by three factors: gestational timing of the highest infection risk, overlap between gestation periods and malaria-prone seasons, and gestational timing that yields the worst malaria-related birth outcomes (see the supplemental appendix S.1.5.2 for details). Nevertheless, this finding may still reflect the influence of a higher infection risk affecting newborns in the rainy season.

S.4.2 Human biology

In Table S.7, this study also investigated whether the influence of wartime pregnancy in malaria-endemic areas varied by biological factors from two perspectives: maternal immunity and child genetics. Similar to Table S.6, the indicator and continuous measure of malaria endemicity were utilized 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.

Maternal immunity may serve a function, provided that the aforementioned mortality consequences were largely driven by malaria infection in pregnancy. First, women acquire parity-dependent immunity; therefore, it is observed that first- and second-time mothers are at greater risk of contracting malaria than multigravidae (e.g., Desai et al., 2007; Schantz-Dunn and Nour, 2009; Uneke, 2007b). Young maternal age also independently increases the risk of infection due to the acquisition of age-associated immunity.[44] Therefore, this study separated the sample into children born to paucigravidae and the others in the first two columns in Table S.7. Those born to young (lower 50% quantile of mothers' age at birth) and elderly mothers (upper 50% quantile) were in the next two columns.

The results only confirm the mortality effect's age-dependent heterogeneity (although a significance of this heterogeneity may also emerge from inappropriate pre- and post-natal care given to their babies by young inexperienced mothers). 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 had not

[44]In general, HIV/AIDS possibly eliminates this gravidity- or age-specific patterns of malaria risk by transferring the burden to all pregnant women (Desai et al., 2007); however, the prevalence of HIV/AIDS in Liberia was estimated at 1.5% and 1.9% based on the 2007 and 2013 DHS reports, percentages that are substantially less than the corresponding rates in eastern and southern African countries.

experienced normal deliveries and/or who had died early in infancy and were thus 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.[45]

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 the malaria-related mortality effect varied across gender due to this male vulnerability, this study separately estimated the specification (1) for boys and girls; the results, reported in the final two columns in Table S.7, indicate that 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 of Akresh et al. (2011). They showed that in Rwanda, the armed conflict reduced the height-for-age of boys and girls equally while highlighting the indiscriminate nature of the violence (compared to a peacetime crop failure, which significantly affected 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 may be attributed to the failure of this study to consider the mortality effect on boys who were not a result of normal pregnancy and/or who died shortly after birth and therefore were not recorded in the DHS data.[46]

S.5 Mothers' health and its long-term selection on the war

This section investigates whether in the long term, the immediate mortality effect may enable only those who are 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 and/or postnatal exposure to malaria infection risk and present health status. To achieve this purpose, this study utilized the 2013 DHS data. The data set includes extensive information regarding 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 the units of analysis (rather than children born to those respondents, as in the mortality equation) and by replacing the outcome of specification (1) with the respondents' height-for-age (z-scores) at the time of the survey, similar DID estimations were performed in Table S.9. The height-for-age measure is frequently utilized to

[45] Based on Rogerson et al. (2003) (p. 1372), for example, the parity-specific pattern was more evident for microscopically, rather than histologically, detectable *P.falciparum* infection.

[46] As seen from Table S.1 (see footnote 16 for exposition of this table), children in malaria-endemic areas tend to be girls due to the war, although the estimate is statistically insignificant. On one hand, unfavorable conditions in wartime might have increased the likelihood of females being 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). On the other hand, this finding may also be consistent with the under-reporting problem of male births and their immediate deaths.

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 regarding height-for-age was collected only for that particular sample. Furthermore, these estimations included similar controls to those used in the fertility analysis performed in subsection 5.3.1 (i.e., Table S.8).

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

As before, the DID approach needs to assume that high and low malaria endemicity areas experienced a similar trend in health outcomes. Figure S.3 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 mention. First, the figure reveals a similar trend of adult health between the two areas observed while supporting the identification assumption (see also the supplemental appendix S.2 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 high endemic ones before the war,[47] the pattern was reversed once the war commenced. 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 enabled only those who were healthy in childhood to grow into adulthood (while discouraging the present survival of weak infants).

Table S.9 reports the DID estimation results of the respondents' height-for-age and height (cm) in columns (a)—(e) and (f)—(j), respectively. For each outcome, the exercise in the first column utilizes 10 categorical variables indicating malaria intensity in contrast to the indicator for the upper 50% quantile and the continuous measure used in the second and third columns, respectively. In the fourth and fifth columns, community-specific linear time-trends (i.e., years of conception multiplied by dummies for each community) were added to regressors used in the estimations performed in the second and third columns. According to the results, wartime pregnancy in malaria-endemic areas is positively correlated with health-related outcomes pertaining to adult women. This finding is robust to the inclusion of numerous geographic and climate controls (the relevant estimation results are

[47]This observation is consistent with the finding in Figure 2, which illustrated that the mortality rate before the war was higher in areas of low malaria endemicity than in areas of high malaria endemicity. Due to the great likelihood of infant mortality, only healthy children are expected to survive in low endemic areas until the present. Thus, the height-for-age in adulthood possibly becomes higher in low endemic areas than in the disease-prone areas.

available from the author upon request) as well as to exploitation of alternative endemicity estimates provided by Bhatt et al. (2015) (see Table S.4).

Notably, because the negative health influence of malaria infection in pregnancy and/or infancy may have accumulating consequences for adult health, the DID approach only identifies net effects of this (negative) and the aforementioned (positive) selection effects. Given these two possibly conflicting forces, the apparent long-term health improvement may highlight the significance of the immediate mortality effects. Moreover, it is also difficult to interpret that 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 in malaria-prone rural areas is lower than that in urban cities. Based on a 2008 nationwide household survey, for example, more than two-thirds of rural households require more than an hour to reach the nearest health care facility, compared to the 41% country-wide average (Lee et al., 2011).

S.6 Geo-coded variables

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

S.6.1 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/`.

S.6.2 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.

S.6.3 Administrative areas

Both the county and district corresponding to each DHS community were 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).

S.6.4 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

S.6.5 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 (≈ 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 (≈ 1 km^2).

S.6.6 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 (≈ 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")

(For the supplemental appendix)

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Table S.1: 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)	-0.414***	(0.151)	0.011	52849
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 S.2: 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)	
Unit of obs.	children		respondents		respondents	
	(a)	(b)	(c)	(d)	(e)	(f)
Conceived in 1989	0.017	0.071**	-0.155	-0.162	-0.926	-0.954
	(0.028)	(0.028)	(0.106)	(0.106)	(0.635)	(0.628)
Conceived in 1988	-0.014	0.044	0.008	0.026	0.047	0.192
	(0.027)	(0.028)	(0.176)	(0.177)	(1.052)	(1.054)
Conceived in 1987	-0.039	0.014	-0.149	-0.154	-0.887	-0.906
	(0.029)	(0.029)	(0.140)	(0.141)	(0.834)	(0.840)
Conceived in 1986	-0.036	0.011	0.014	0.012	0.086	0.077
	(0.028)	(0.028)	(0.139)	(0.142)	(0.830)	(0.845)
Conceived in 1985	-0.061**	-0.012	-0.084	-0.083	-0.502	-0.514
	(0.028)	(0.027)	(0.168)	(0.167)	(1.001)	(0.989)
Conceived in 1984	-0.039	-0.003	0.016	0.005	0.096	0.031
	(0.030)	(0.030)	(0.129)	(0.133)	(0.766)	(0.792)
Conceived in 1983	-0.069**	-0.041	-0.031	-0.038	-0.184	-0.240
	(0.029)	(0.028)	(0.130)	(0.130)	(0.777)	(0.778)
Conceived in 1982	-0.068**	-0.034	0.055	0.042	0.328	0.218
	(0.029)	(0.028)	(0.164)	(0.167)	(0.976)	(1.005)
Conceived in 1981	-0.019	0.007	-0.174	-0.167	-1.035	-0.999
	(0.036)	(0.035)	(0.129)	(0.128)	(0.766)	(0.762)
Conceived in 1980	-0.018	-0.003	-0.084	-0.096	-0.506	-0.575
	(0.038)	(0.036)	(0.167)	(0.169)	(0.999)	(1.005)
Malaria endemicity (50-100 percentile)						
× Conceived in 1989	-0.033	-0.034	-0.003	0.015	0.027	0.155
	(0.038)	(0.036)	(0.149)	(0.150)	(0.890)	(0.895)
× Conceived in 1988	0.026	0.015	0.013	-0.013	0.132	-0.085
	(0.039)	(0.037)	(0.235)	(0.235)	(1.402)	(1.399)
× Conceived in 1987	0.028	0.017	0.034	0.061	0.243	0.447
	(0.039)	(0.038)	(0.172)	(0.174)	(1.025)	(1.042)
× Conceived in 1986	0.025	0.022	-0.070	-0.090	-0.371	-0.535
	(0.038)	(0.036)	(0.215)	(0.217)	(1.287)	(1.300)
× Conceived in 1985	0.058	0.047	-0.096	-0.087	-0.467	-0.372
	(0.039)	(0.038)	(0.216)	(0.216)	(1.293)	(1.294)
× Conceived in 1984	-0.027	-0.031	-0.088	-0.070	-0.288	-0.170
	(0.041)	(0.039)	(0.178)	(0.181)	(1.076)	(1.097)
× Conceived in 1983	0.062	0.066*	-0.053	-0.042	-0.125	-0.047
	(0.040)	(0.038)	(0.177)	(0.179)	(1.075)	(1.092)
× Conceived in 1982	0.045	0.032	-0.205	-0.186	-0.951	-0.820
	(0.042)	(0.040)	(0.216)	(0.220)	(1.352)	(1.375)
× Conceived in 1981	0.035	0.018	0.061	0.063	0.481	0.488
	(0.049)	(0.047)	(0.172)	(0.173)	(1.034)	(1.041)
× Conceived in 1980	0.001	0.014	0.042	0.029	-0.089	-0.214
	(0.050)	(0.048)	(0.244)	(0.245)	(1.605)	(1.581)
Individual controls	No	Yes	No	Yes	No	Yes
Mother characteristics	No	Yes	NA	NA	NA	NA
Month-of-conception FE	No	Yes	No	Yes	No	Yes
Round FE	Yes	Yes	NA	NA	NA	NA
Community FE	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.122	0.158	0.150	0.155	0.147	0.155
No. of obs.	9342	9259	3135	3125	3138	3128

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 in columns (a)—(b) include a gender dummy, birth order, a single-birth dummy, and mothers' age at birth. The individual controls in columns (c)—(f) include birth order, no. of alive siblings at age 10, no. of late siblings at age 10, and religion dummies. (4) The mother characteristics include mothers' education (years) and religion dummies.

Table S.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	-0.005	0.001	-0.000	-0.004	-0.002	0.945
(50-100 percentile)	(0.009)	(0.015)	(0.018)	(0.020)	(0.020)	(0.021)	(0.021)	(0.097)
Male (dummy)	0.008*	0.017**	0.020**	0.025***	0.026***	0.027***	0.028***	1.132***
	(0.005)	(0.007)	(0.008)	(0.009)	(0.009)	(0.009)	(0.009)	(0.051)
Birth order	0.008***	0.018***	0.024***	0.026***	0.028***	0.028***	0.029***	1.123***
	(0.002)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.024)
Single birth (dummy)	-0.210***	-0.277***	-0.295***	-0.311***	-0.332***	-0.336***	-0.328***	0.435***
	(0.035)	(0.038)	(0.039)	(0.039)	(0.037)	(0.037)	(0.037)	(0.055)
Mother's age at birth	-0.004***	-0.009***	-0.012***	-0.014***	-0.014***	-0.014***	-0.015***	0.936***
	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.009)
Mother's education	-0.002**	-0.007***	-0.008***	-0.009***	-0.010***	-0.010***	-0.011***	0.944***
(years)	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.008)
Longitude	0.004	0.003	-0.022	-0.026	-0.020	-0.024	-0.024	0.944
	(0.012)	(0.017)	(0.020)	(0.023)	(0.024)	(0.024)	(0.026)	(0.135)
Latitude	0.022	0.032	0.033	0.038	0.065*	0.064*	0.067*	1.537**
	(0.022)	(0.029)	(0.034)	(0.037)	(0.037)	(0.038)	(0.039)	(0.296)
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.)	-	-	-	-	-	-	-	0.284
R-squared	0.070	0.077	0.063	0.059	0.059	0.059	0.058	-
No. of obs.	9070	9112	9112	9112	9112	9112	9112	8593
	(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	0.017*	0.018*	0.021*	0.025**	1.194**
(50-100 percentile)	(0.004)	(0.007)	(0.009)	(0.010)	(0.011)	(0.012)	(0.012)	(0.098)
Male (dummy)	0.010***	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***	0.016***	0.019***	0.021***	0.023***	0.023***	1.133***
	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.013)
Single birth (dummy)	-0.142***	-0.195***	-0.216***	-0.230***	-0.236***	-0.232***	-0.236***	0.423***
	(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*	-0.002***	-0.002***	-0.002***	-0.003***	-0.003***	-0.004***	0.980***
(years)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.005)
Longitude	0.004	0.001	-0.011	-0.021*	-0.024*	-0.023*	-0.024*	0.872
	(0.005)	(0.008)	(0.010)	(0.011)	(0.012)	(0.013)	(0.013)	(0.075)
Latitude	0.004	0.012	0.003	-0.008	-0.006	-0.011	-0.015	0.993
	(0.008)	(0.011)	(0.016)	(0.018)	(0.019)	(0.021)	(0.022)	(0.138)
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.)	-	-	-	-	-	-	-	0.229
R-squared	0.062	0.067	0.065	0.060	0.059	0.056	0.054	-
No. of obs.	42314	41081	39596	37079	34491	31769	29353	41182
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
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 S.4: 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)	
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): Endemicity in 2000						
Conceived after the outbreak of the war	0.019*	0.073**	0.146**	0.701**	0.848**	4.861**
× Malaria endemicity	(0.010)	(0.037)	(0.067)	(0.275)	(0.427)	(1.886)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)
Panel (B): Endemicity in 2001						
Conceived after the outbreak of the war	0.022**	0.079**	0.123*	0.701***	0.807*	4.828***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.267)	(0.431)	(1.827)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)
Panel (C): Endemicity in 2002						
Conceived after the outbreak of the war	0.025**	0.082**	0.110	0.733***	0.734*	5.013***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.266)	(0.432)	(1.818)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)
Panel (D): Endemicity in 2003						
Conceived after the outbreak of the war	0.025**	0.083**	0.118*	0.734***	0.778*	5.121***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.259)	(0.431)	(1.838)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(5a)	(5b)	(5c)	(5d)	(5e)	(5f)
Panel (E): Endemicity in 2004						
Conceived after the outbreak of the war	0.024**	0.077**	0.124*	0.744***	0.820*	5.151***
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.261)	(0.432)	(1.855)
R-squared	0.085	0.085	0.133	0.134	0.140	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(6a)	(6b)	(6c)	(6d)	(6e)	(6f)
Panel (F): Endemicity in 2005						
Conceived after the outbreak of the war	0.013	0.069*	0.150**	0.723***	0.990**	4.931***
× Malaria endemicity	(0.010)	(0.036)	(0.068)	(0.265)	(0.435)	(1.849)
R-squared	0.085	0.085	0.133	0.134	0.141	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(7a)	(7b)	(7c)	(7d)	(7e)	(7f)
Panel (G): Endemicity in 2006						
Conceived after the outbreak of the war	0.010	0.065*	0.167**	0.712***	1.088**	4.754**
× Malaria endemicity	(0.010)	(0.037)	(0.068)	(0.269)	(0.436)	(1.840)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(8a)	(8b)	(8c)	(8d)	(8e)	(8f)
Panel (H): Endemicity in 2007						
Conceived after the outbreak of the war	0.015	0.064*	0.225***	0.696***	1.413***	4.573**
× Malaria endemicity	(0.010)	(0.037)	(0.067)	(0.265)	(0.433)	(1.776)
R-squared	0.085	0.085	0.135	0.134	0.142	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(9a)	(9b)	(9c)	(9d)	(9e)	(9f)
Panel (I): Endemicity in 2008						
Conceived after the outbreak of the war	0.021**	0.066*	0.206***	0.670***	1.308***	4.302**
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.257)	(0.432)	(1.686)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(10a)	(10b)	(10c)	(10d)	(10e)	(10f)
Panel (J): Endemicity in 2009						
Conceived after the outbreak of the war	0.017*	0.072**	0.195***	0.665***	1.247***	4.237**
× Malaria endemicity	(0.010)	(0.036)	(0.067)	(0.255)	(0.430)	(1.646)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
	(11a)	(11b)	(11c)	(11d)	(11e)	(11f)
Panel (K): Endemicity in 2010						
Conceived after the outbreak of the war	0.016	0.078**	0.178***	0.663**	1.139***	4.163**
× Malaria endemicity	(0.010)	(0.037)	(0.067)	(0.265)	(0.430)	(1.685)
R-squared	0.085	0.085	0.134	0.134	0.141	0.141
No. of obs.	49121	49121	4576	4576	4582	4582
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

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 S.5: Mortality effects trend (OLS)

Dependent variable:	One if die within within 12 months after the birth	
Malaria endemicity:	50-100 percentile	continuous measure
	(a)	(b)
Malaria endemicity × Conceived:		
during the 1st war	0.036***	0.129***
(Dec 1989 to July 1997)	(0.011)	(0.049)
during the ceasefire	0.031**	0.098*
(Aug 1997 to Mar 1999)	(0.014)	(0.052)
during the 2nd war	0.035***	0.097*
(Apr 1999 to Aug 2003)	(0.012)	(0.053)
during the post-war	0.036***	0.104*
(After Sep 2003)	(0.012)	(0.054)
Male (dummy)	0.017***	0.017***
	(0.003)	(0.003)
Birth order	0.013***	0.013***
	(0.001)	(0.001)
Single birth (dummy)	-0.239***	-0.239***
	(0.014)	(0.014)
Mother's age at birth	-0.006***	-0.006***
	(0.000)	(0.000)
Mother's education	-0.003***	-0.003***
(years)	(0.001)	(0.001)
Mother's religion FE	Yes	Yes
Month-of-conception FE	Yes	Yes
Year-of-conception FE	Yes	Yes
Round FE	Yes	Yes
Community FE	Yes	Yes
R-squared	0.085	0.085
No. of obs.	49121	49121

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 S.6: 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 seasons	Dry season
	(1a)	(1b)	(1c)	(1d)	(1e)	(1f)
Panel (A): $Z = 12$						
Malaria endemicity (50-100 percentile)	0.037**	0.030**	0.042***	0.025	0.040***	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	0.078	0.095	0.104
No. of obs.	22995	26126	32532	16589	26632	22489
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)
Panel (B): $Z = 36$						
Malaria endemicity (50-100 percentile)	0.040**	0.019	0.035**	0.028	0.040***	0.025*
× Conceived after	(0.018)	(0.016)	(0.015)	(0.020)	(0.015)	(0.015)
the outbreak of the war						
R-squared	0.096	0.090	0.097	0.083	0.102	0.112
No. of obs.	20657	23328	29136	14849	23923	20062
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)
Panel (C): $Z = 60$						
Malaria endemicity (50-100 percentile)	0.037*	0.024	0.038**	0.029	0.046***	0.025
× Conceived after	(0.019)	(0.018)	(0.016)	(0.020)	(0.016)	(0.016)
the outbreak of the war						
R-squared	0.097	0.092	0.097	0.086	0.105	0.115
No. of obs.	18354	20453	25610	13197	21070	17737
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)
Panel (D): $Z = 12$						
Malaria endemicity (continuous)	0.118*	0.069	0.149*	0.098	0.177***	0.086
× Conceived after	(0.071)	(0.069)	(0.078)	(0.063)	(0.067)	(0.060)
the outbreak of the war						
R-squared	0.087	0.087	0.091	0.078	0.095	0.103
No. of obs.	22995	26126	32532	16589	26632	22489
	(5a)	(5b)	(5c)	(5d)	(5e)	(5f)
Panel (E): $Z = 36$						
Malaria endemicity (continuous)	0.136*	-0.003	0.129	0.074	0.169**	0.070
× Conceived after	(0.076)	(0.078)	(0.083)	(0.073)	(0.077)	(0.066)
the outbreak of the war						
R-squared	0.096	0.090	0.097	0.083	0.102	0.112
No. of obs.	20657	23328	29136	14849	23923	20062
	(6a)	(6b)	(6c)	(6d)	(6e)	(6f)
Panel (F): $Z = 60$						
Malaria endemicity (continuous)	0.127	0.010	0.145*	0.067	0.197**	0.065
× Conceived after	(0.078)	(0.082)	(0.085)	(0.072)	(0.079)	(0.068)
the outbreak of the war						
R-squared	0.097	0.091	0.097	0.085	0.105	0.115
No. of obs.	18354	20453	25610	13197	21070	17737
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 S.7: 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**	0.035***	0.017	0.032***	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	0.099	0.103	0.100	0.097	0.097
No. of obs.	26848	22273	23731	25390	25141	23980
	(2a)	(2b)	(2c)	(2d)	(2e)	(2f)
Panel (B): $Z = 36$						
Malaria endemicity (50-100 percentile)	0.037**	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	0.109	0.106	0.107	0.104	0.104
No. of obs.	23707	20278	20638	23347	22498	21487
	(3a)	(3b)	(3c)	(3d)	(3e)	(3f)
Panel (C): $Z = 60$						
Malaria endemicity (50-100 percentile)	0.036**	0.034**	0.002	0.037***	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	0.113	0.111	0.108	0.105	0.108
No. of obs.	20495	18312	17521	21286	19886	18921
	(4a)	(4b)	(4c)	(4d)	(4e)	(4f)
Panel (D): $Z = 12$						
Malaria endemicity (continuous)	0.112*	0.117**	0.039	0.106*	0.136**	0.101*
× Conceived after	(0.067)	(0.057)	(0.078)	(0.057)	(0.066)	(0.060)
the outbreak of the war						
R-squared	0.105	0.098	0.103	0.100	0.096	0.097
No. of obs.	26848	22273	23731	25390	25141	23980
	(5a)	(5b)	(5c)	(5d)	(5e)	(5f)
Panel (E): $Z = 36$						
Malaria endemicity (continuous)	0.148**	0.087	0.023	0.106*	0.120	0.098
× Conceived after	(0.073)	(0.064)	(0.089)	(0.060)	(0.076)	(0.064)
the outbreak of the war						
R-squared	0.109	0.108	0.103	0.107	0.104	0.103
No. of obs.	26848	22273	23731	25390	22498	21487
	(6a)	(6b)	(6c)	(6d)	(6e)	(6f)
Panel (F): $Z = 60$						
Malaria endemicity (continuous)	0.137*	0.099	-0.018	0.133**	0.132*	0.097
× Conceived after	(0.075)	(0.068)	(0.094)	(0.063)	(0.078)	(0.065)
the outbreak of the war						
R-squared	0.113	0.113	0.111	0.108	0.105	0.108
No. of obs.	20495	18312	17521	21286	19886	18921
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 S.8: Fertility trend (ordered probit model)

Dependent variables:	No. of children ever delivered		No. of sons ever delivered		No. of daughters ever delivered			
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)
Born in 1966-1970	-0.427***	-	-0.286***	-	-0.300***	-	0.016	-
	(0.073)		(0.066)		(0.069)		(0.068)	
Born in 1971-1975	-0.909***	-	-0.629***	-	-0.650***	-	0.020	-
	(0.073)		(0.067)		(0.064)		(0.064)	
Born in 1976-1980	-1.379***	-	-1.015***	-	-0.932***	-	0.099	-
	(0.074)		(0.064)		(0.067)		(0.060)	
Born in 1981-1985	-2.211***	-	-1.588***	-	-1.567***	-	-0.037	-
	(0.081)		(0.069)		(0.070)		(0.063)	
Born in 1986-1990	-3.146***	-	-2.251***	-	-2.289***	-	-0.356***	-
	(0.093)		(0.076)		(0.076)		(0.065)	
Born in 1991-1995	-3.948***	-	-2.932***	-	-2.820***	-	-0.480***	-
	(0.093)		(0.082)		(0.078)		(0.073)	
Born in 1996-1998	-5.484***	-	-4.075***	-	-4.225***	-	-1.549***	-
	(0.107)		(0.102)		(0.111)		(0.104)	
Malaria endemicity (50-100 percentile)								
× Born in 1966-1970	-0.099	-0.093	-0.100	-0.094	-0.048	-0.045	0.028	0.025
	(0.100)	(0.098)	(0.090)	(0.088)	(0.092)	(0.091)	(0.091)	(0.091)
× Born in 1971-1975	0.020	0.048	0.031	0.050	-0.001	0.017	0.009	0.011
	(0.094)	(0.091)	(0.088)	(0.085)	(0.084)	(0.084)	(0.086)	(0.086)
× Born in 1976-1980	-0.113	-0.084	-0.070	-0.050	-0.081	-0.061	0.060	0.062
	(0.093)	(0.089)	(0.085)	(0.081)	(0.086)	(0.085)	(0.083)	(0.083)
× Born in 1981-1985	-0.043	-0.028	-0.063	-0.055	0.015	0.024	0.160*	0.158*
	(0.101)	(0.096)	(0.090)	(0.085)	(0.091)	(0.089)	(0.083)	(0.083)
× Born in 1986-1990	-0.026	-0.002	-0.086	-0.075	0.100	0.113	0.297***	0.293***
	(0.112)	(0.105)	(0.097)	(0.091)	(0.097)	(0.094)	(0.083)	(0.083)
× Born in 1991-1995	-0.180*	-0.165	-0.079	-0.066	-0.130	-0.122	0.123	0.119
	(0.109)	(0.104)	(0.103)	(0.100)	(0.099)	(0.097)	(0.091)	(0.091)
× Born in 1996-1998	-0.118	-0.161	-0.060	-0.120	-0.010	-0.023	0.280*	0.249*
	(0.132)	(0.131)	(0.141)	(0.141)	(0.151)	(0.154)	(0.144)	(0.147)
Birth order	0.004	0.007	0.008	0.011*	-0.003	-0.001	-0.006	-0.006
	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
No. of alive siblings	0.009	0.009	0.004	0.003	0.008	0.008	0.006	0.006
at age 10	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)	(0.007)
No. of late siblings	0.014	0.015	0.014	0.014	0.005	0.006	-0.005	-0.004
at age 10	(0.011)	(0.011)	(0.011)	(0.011)	(0.012)	(0.012)	(0.012)	(0.012)
No. of children ever	-	-	-	-	-	-	0.516***	0.511***
delivered							(0.008)	(0.008)
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-birth FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-birth FE	No	Yes	No	Yes	No	Yes	No	Yes
Round FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
All interactions = 0	0.109	0.060	0.707	0.595	0.079	0.071	0.000	0.001
(p-values)								
No. of obs.	16191	16191	16191	16191	16191	16191	16191	16191

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 S.9: Mothers' health and its long-term selection on the war: 2013 DHS only (OLS)

Dependent variables:	Height for age (z-score)					Height (cm)				
	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)	(i)	(j)
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*	-	0.256**	-	-	0.766*	-	1.490**	-
(50-100 percentile)		(0.067)		(0.115)			(0.434)		(0.711)	
× Malaria endemicity	-	-	0.802***	-	1.285***	-	-	5.099**	-	7.418**
(continuous measure)			(0.306)		(0.463)			(2.003)		(2.879)
Birth order	0.009	0.009	0.009	0.007	0.006	0.049	0.050	0.048	0.042	0.038
	(0.012)	(0.012)	(0.012)	(0.013)	(0.013)	(0.072)	(0.072)	(0.072)	(0.077)	(0.077)
No. of alive siblings	0.006	0.006	0.006	0.009	0.009	0.049	0.050	0.051	0.058	0.063
at age 10	(0.011)	(0.011)	(0.011)	(0.012)	(0.012)	(0.067)	(0.067)	(0.067)	(0.072)	(0.073)
No. of late siblings	-0.019	-0.017	-0.017	-0.013	-0.012	-0.159	-0.155	-0.157	-0.126	-0.124
at age 10	(0.019)	(0.019)	(0.018)	(0.021)	(0.021)	(0.133)	(0.133)	(0.133)	(0.149)	(0.148)
Religion FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-of-conception FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Community-specific trend	No	No	No	Yes	Yes	No	No	No	Yes	Yes
R-squared	0.135	0.133	0.134	0.189	0.190	0.142	0.140	0.141	0.198	0.199
No. of obs.	4576	4576	4576	4576	4576	4582	4582	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 S.10: Checking on selected resettlement (OLS)

Dependent variables:	Age (years)	Education (years)	Christianity (proportion)	Married (proportion)	No. of children ever delivered	Household size
	(a)	(b)	(c)	(d)	(e)	(f)
Malaria endemicity	1.677***	-1.548***	0.001	0.093***	0.372***	1.428***
(50-100 percentile)	(0.406)	(0.298)	(0.042)	(0.022)	(0.136)	(0.364)
Malaria endemicity	-0.781*	0.226	0.046	-0.001	0.221	-1.218***
× One if 2007 or 2013 DHS	(0.450)	(0.343)	(0.045)	(0.026)	(0.155)	(0.386)
One if 2007 DHS	1.625***	0.877***	0.260***	-0.029	-0.214*	-1.315***
	(0.332)	(0.295)	(0.031)	(0.020)	(0.110)	(0.267)
One if 2013 DHS	1.453***	0.956***	0.241***	-0.046**	-0.048	-0.980***
	(0.317)	(0.291)	(0.032)	(0.019)	(0.110)	(0.267)
R-squared	0.081	0.133	0.197	0.093	0.094	0.173
No. of communities	776	776	776	776	776	776

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

Table S.11: Impacts of wartime pregnancy in malaria endemic areas: 2SLS and discontinuity sample

	2SLS				Discontinuity sample (OLS)	
Malaria endemicity:	50-100 percentile		Continuous measure		50-100 percentile	Continuous measure
	1st stage	2nd stage	1st stage	2nd stage		
Dependent variables:	Conceived after the outbreak of the war × Malaria endemicity	One if die within 12 months after the birth	Conceived after the outbreak of the war × Malaria endemicity	One if die within 12 months after the birth	One if die within 12 months after the birth	One if die within 12 months after the birth
	(a)	(b)	(c)	(d)	(e)	(f)
Conceived after the outbreak of the war						
× Malaria endemicity	-	0.034***	-	0.121***	0.062*	0.225
		(0.012)		(0.045)	(0.037)	(0.142)
× Malaria suitability (temperature)	1.618***	-	0.324***	-	-	-
	(0.035)		(0.007)			
× Malaria suitability (precipitation)	1.236***	-	0.359***	-	-	-
	(0.012)		(0.002)			
Male (dummy)	0.001	0.017***	0.000**	0.017***	0.029	0.028
	(0.001)	(0.003)	(0.000)	(0.003)	(0.019)	(0.019)
Birth order	0.001*	0.013***	0.000**	0.013***	0.021***	0.021***
	(0.000)	(0.001)	(0.000)	(0.001)	(0.008)	(0.008)
Single birth (dummy)	0.008***	-0.239***	0.001	-0.239***	-0.398***	-0.398***
	(0.003)	(0.011)	(0.000)	(0.011)	(0.068)	(0.068)
Mother's age at birth	-0.000	-0.006***	-0.000	-0.006***	-0.014***	-0.014***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.003)	(0.003)
Mother's education	-0.000	-0.003***	-0.000	-0.003***	-0.005*	-0.005*
(years)	(0.000)	(0.000)	(0.000)	(0.000)	(0.003)	(0.003)
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	Yes	Yes	NA	NA
Community FE	Yes	Yes	Yes	Yes	Yes	Yes
1st-stage F-statistics	8095.33	-	12842.37	-	-	-
Hansen (p-values)	-	0.468	-	0.266	-	-
R-squared	0.922	0.085	0.988	0.085	0.273	0.273
No. of obs.	49121	49121	49121	49121	2552	2552

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)-(d), whereas they are robust to heteroskedasticity and clustered residuals within each community in columns (e)-(f).

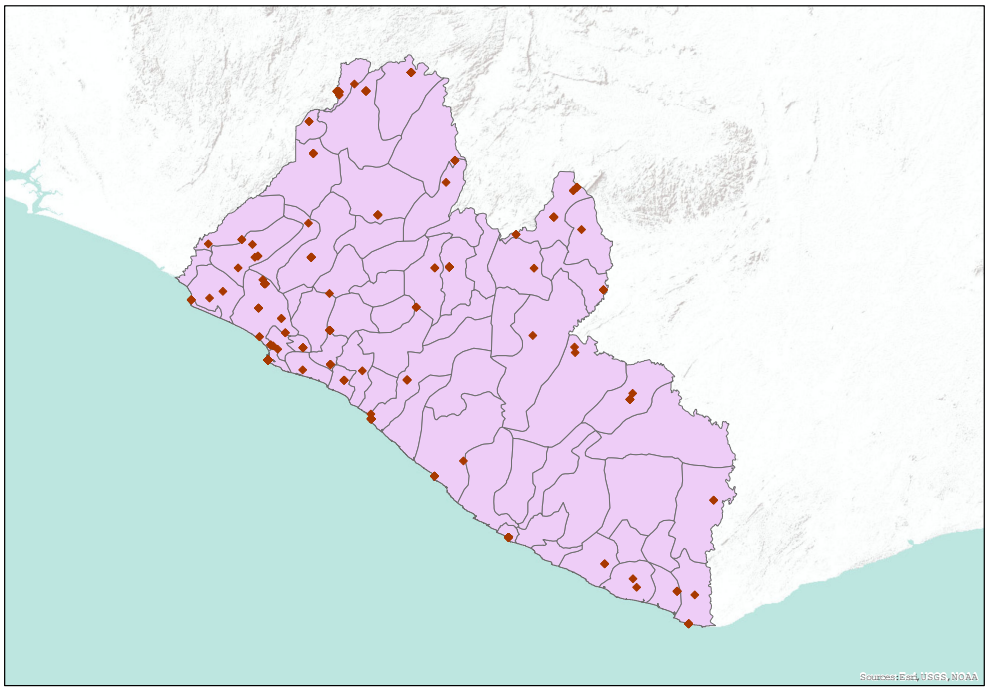

Figure S.1: Spatial distribution of battle events with district boundaries

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`).

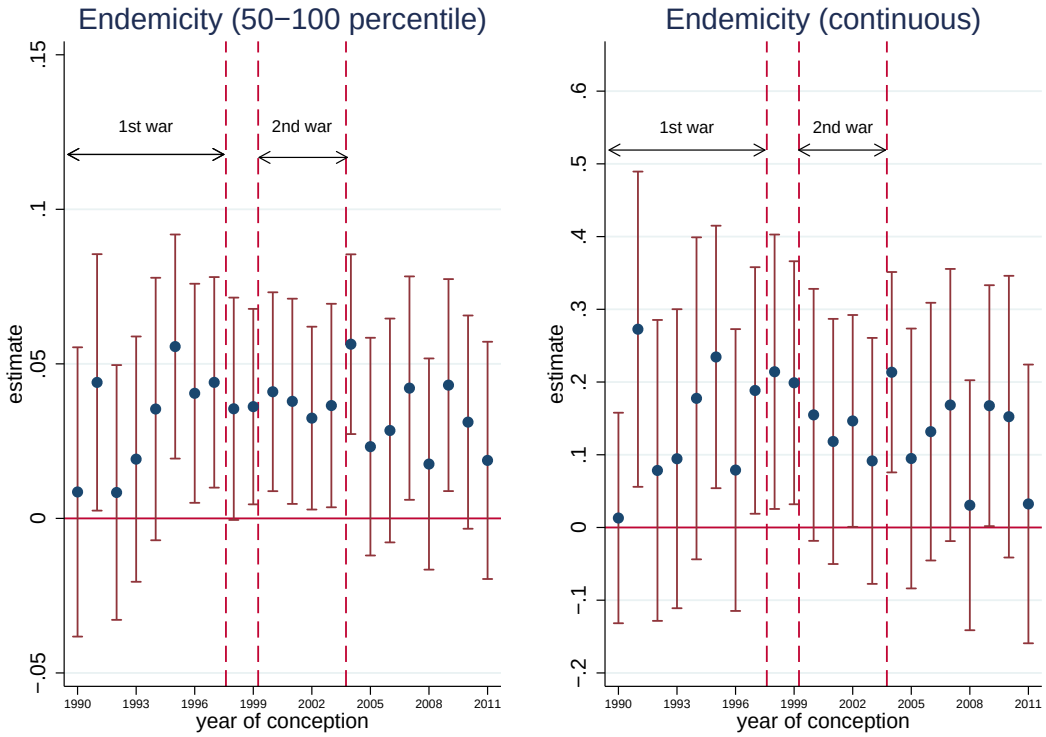

Figure S.2: Impacts of wartime pregnancy in malaria endemic areas on one-year mortality by the year of conception (LPM)

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.

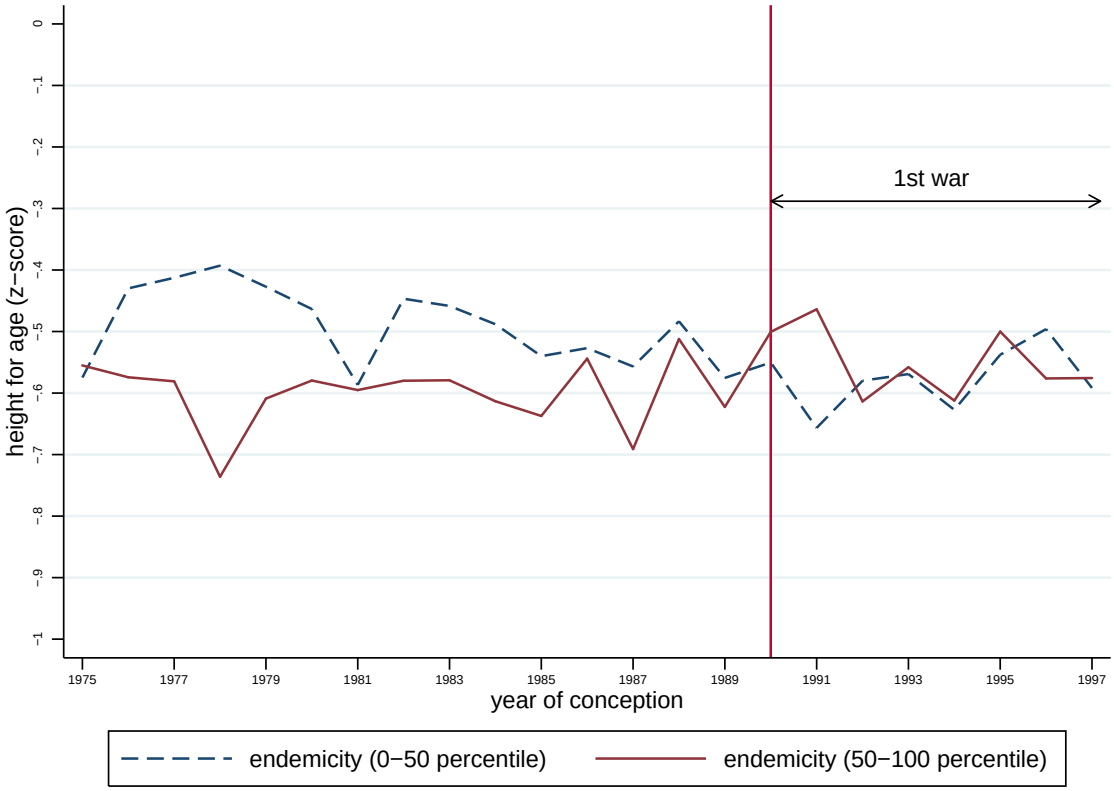

Figure S.3: Height-for-age (z-score) by the timing of conception (2013 DHS only)