Weather and Infant Mortality in Africa*

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November 9, 2012

Abstract

We estimate how random weather fluctuations affected infant mortality across 28 African countries in the past, combining high-resolution data from retrospective fertility surveys (DHS) and climate-model reanalysis (ERA-40). We find that infants were much more likely to die when exposed in utero to much longer malaria spells than normal in epidemic malaria regions, and to droughts in arid areas, especially when born in the hungry season. Based on these estimates, we predict aggregate infant deaths in Africa, due to extreme weather events and to maternal malaria in epidemic areas for 1981-2000 and 2081-2100.

*Prepared for the conference on Climate and the Economy in Stockholm, September 5-8, 2012. We are grateful to Daron Acemoglu, Sandra Black, Robin Burgess, Angus Deaton, Colin Jones, Dean Karlan, Heiner Körnich, Ben Smith, Peter Svedberg, Jakob Svensson, John van Reenen, and participants in seminars at the IIES, Amsterdam, UPF, UCLA, UBC, Chicago, LSE, Princeton, Berkeley, Oslo, LSHTM, Edinburgh, Nottingham, Houston, Stockholm School of Economics, Gothenburg, Harvard/MIT, Bristol, Leicester, Umeå, Zurich, Toulouse, SEA Annual Meeting 2009, CIFAR Meeting, and 2010 EEA Annual Congress for helpful comments; to Heiner Körnich, Lars Eklundh, and Johannes Karlsson for assistance with data; to Pamela Campa for research assistance; and to Mistra, the ERC, and the Torsten and Ragnar Söcerberg Foundations for financial support.

1 Introduction

To think clearly about global policy responses to climate change, we need to know about the impact of weather on central socioeconomic outcomes, like health, at large scales and over long periods of time. While some impact assessments do exist,[1] many global estimates rely on bold extrapolation. Existing knowledge is particularly scant for developing regions, especially for Africa. As its climate is already harsh and its societies probably will remain vulnerable, Africa is likely to be hit the hardest by a changing climate.

We deal only marginally with climate change, but carefully evaluate the health impacts of weather shocks in Africa over some 40 years in the past. Specifically, we focus on infant mortality. Alongside HIV, infant death is Africa's largest health problem: still today, close to 10% of babies die before the age of one. But unlike HIV, weather variation is important for infant death, in particular through its effect on malaria and malnutrition. The fragmented information we have about such effects comes from clinical short-run studies in local settings, the imprecisely estimated number of drought victims, and cross-sectional regressions on cause-of-death surveys.

Differently from these studies, we exploit 50 nationally representative Demographic and Health Surveys, covering more than 17,500 locations in 28 African countries. Thus, we obtain data for nearly one million births in 1957-2002 – our period of study – including their month of birth and geographic (survey) coordinates. To gauge how the probability of infant death depends on local weather conditions, we use data from so-called re-analysis by a global atmospheric weather forecasting model, with a six-hour frequency on a 1.25×1.25 degree (139x139 kms, at the equator) earth grid.

To identify the causal effects of weather shocks, we use only temporary deviations in infant mortality and in weather outcomes from their respective monthly average pattern in each location. Constant geographic and seasonal differences are correlated with numerous other factors that also influence infant mortality. For example, coastal areas have different weather patterns than land-locked ones, but as economic opportunities are better, people have higher incomes and lower infant mortality. Similarly, long-run weather trends are highly spatially correlated, and thus potentially confounded with long-run infant-mortality trends tied to the evolution of national health-care systems or income. There is little hope to convincingly control for all relevant deter-

[1] See Parry et al. (2007) for an overview.

minants.

For these reasons, we rely on natural year-to-year variations of each month's weather, which should not correlate with any latent determinants of health. In effect, we are using a gigantic set of natural experiments to identify the effects on infant mortality. Therefore, it is logical to compare results from our large-scale study to those small-scale studies that rely on randomized control trials to generate variation in other determinants of malaria and malnutrition such as bednet use and food supplements.

We uncover statistically and quantitatively significant effects through both malaria and malnutrition. Infants born in areas with epidemic malaria, who in utero experience worse malarious conditions than the site-specific seasonal means face a higher risk of death, especially in areas with a low average exposure to malaria. A six-month malaria epidemic in a place with little average exposure to malaria raises infant mortality by more than 3.5 percentage points. Infants born in arid climate regions of Africa, who in utero experience droughts, face a risk of death 2.5 percentage points higher than other babies. This estimate doubles for infants born in the so-called hungry season around the start of the rains. We also find marked heterogeneities in the effects of rainfall and drought on infant mortality, depending on household occupation and education.

In aggregate, we estimate that 84,000 infants died in extreme malaria episodes between 1981 and 2000 in areas where malaria is epidemic in our sample. Extrapolating to all epidemic areas of Africa, we find that 106,000 infants died of maternal malaria over these twenty years. For the same period, we estimate that 8,700 infants died in droughts in the arid areas of our sample, and 11,000 in all arid areas of Africa. The latter number is comparable to a back-of-the envelope calculation of drought victims from the EM-DAT database of the Centre for Research on the Epidemiology of Disasters.

We also compute the share of all infant deaths caused by maternal malaria, in epidemic regions, relying on the estimated fall in infant mortality in randomly non-malarious weather conditions. This is in the spirit of comparing infants from non-malarious villages, due to randomly assigned medical treatment, with infants from non-treated villages. We find that infant mortality drops by 0.8 percentage points in non-malarious conditions – about 7% of our sample mean. By comparison, Steketee et al. (2001) – who survey results from randomized epidemiological studies – find that infant mortality falls by 3-8% (in endemic regions) when mothers are malaria-free due to randomized medical intervention.

We can use the same methodology to forecast future infant mortality risks due to malaria and droughts in the wake of climate change, even though these estimates are subject to qualifications such as our not taking account of different types of adaptation. Naturally, the estimates depend critically on the extent of climate change, but it may also depend on population growth. For example we find that – even as projected population change is taken into account – the number of infant deaths due to malaria epidemics are cut by more than half in a midrange mitigation scenario (from 84,000 to 41,000 in our sample), while it more than doubles in a high-emission scenario (to 202,000) in the high-emission scenario. The number of infants deaths from droughts goes up in both scenarios, but by *more* in the midrange scenario, reflecting the interactions between climate change and population growth.

While we are not aware of any studies with a similar scope and methodology for Africa, there exist a few related recent studies by economists. Deschenes and Greenstone (2007b) estimate the effect of weather shocks on overall mortality in the United States, but they rely on county-level rather than individual-level data and focus on cardiovascular disease. Barecca et al (2012) estimate the effect of climate change on mortality in the US, taking into account patterns of adaptation estimated from historical data. Burgess et al (2010) look at weather-induced mortality in India, but they too look at overall mortality and mostly rely on district-level data. References to other related works are given in context below.[2]

In the following, Section 2 of the paper gives general background on our data sources and how we put the data together. We also discuss the seasonality of African weather. Sections 3 and 4 go into details about our methods of analysis. These sections report the estimated effects of malaria and malnutrition, respectively, on infant mortality. Section 5 shows how our results can be used to estimate some aggregate effects on infant mortality and to pinpoint the areas most at risk in the past and in the future. We also discuss methodological differences between our estimates and those in the existing medical literature. Section 6 summarizes our findings and mentions possible extensions.

[2]Artadi (2006) estimates the impact of being born in rainy seasons and hungry seasons on infant mortality in African countries. But her interest is to measure the impact of average monthly weather patterns while our focus is to estimate the weather impact of deviations from the average seasonal pattern.

2 Data and Background

DHS surveys Demographic and Health Surveys (DHS) have been carried out in many developing countries since 1984 with financial support from USAID. Each survey collects information on life and health outcomes by interviews of a nationally representative sample of women in child-bearing age. See MEASURE DHS (2012) for details about the sampling procedure.

Thanks to their standardized survey format, data from different surveys can easily be combined. We use all 50 DHS surveys, from 28 African countries, available in 2011, where the geographic coordinate of each cluster – town district or village – is collected by a GPS receiver. These surveys comprise information from a total of 17,568 clusters covering all parts of the continent.

Women aged 15 to 49 in the sampled households are asked about the month and year of birth for each of their children, whether the child died after birth and, if so, the age at death in months. The surveys we exploit cover circa 1.2 million births by about 300,000 mothers in the period (1957-2002) covered by our weather data. Dropping observations with imputed birth dates leaves us with 962,471 births by 269,754 mothers.[3]

For each birth, we define the major dependent variable in the paper: a binary indicator of whether the child died as an infant – i.e., at the age of 12 months or less. This indicator varies quite a bit across time and place. For the full sample, the overall mean is 100.6 deaths per 1000 births, with a standard deviation across clusters of 69.3. But the mean masks a general decline from levels about 144 in 1970 to about 86 in 2002. Infant mortality also varies quite a bit from year to year, as well as across rural and urban areas within the same country.

While the birth and death of one's children are certainly life-defining events, we cannot rule out measurement error (perhaps more about the year than the month of birth or death), as the surveys are retrospective. However, our results do not change significantly when we drop all reported births more than 10 years before the survey. If events nearer in time are more easily

[3]One could argue that mortality of babies with missing birth date may be lower because mothers may remember the birth date of dead babies better. However, in the data babies with imputed birth dates die *more likely*, by about 96 deaths per 1000, than babies with non-imputed dates. This suggests that babies dropped from our sample are more vulnerable to weather shocks, and that our estimates give a lower bound of the true impact.

recollected, this suggests that measurement error owing to imperfect recall is not a major problem.

Another cause of concern is migration of mothers, such that locations at survey time may differ from locations at children's birth time. Dropping all births before migrating mothers (last) move to the survey location, however, does not materially affect the results. Thus, the prospective downward bias of using mis-located weather data appears to be small. In Sections 3 and 4, we discuss other possible sources of selection bias in context.

The DHS surveys also give basic information about each child's gender and birth-order, and – at the moment of survey – their mother's weight, stature, years of education, and occupation, her husband's years of education and occupation, the household's assets, etc. We exploit some of these variables to see if the impact of weather shocks on infant mortality is heterogeneous.

ERA-40 re-analysis To isolate exogenous variation in variables like income, development economists increasingly rely on weather shocks from different sources (see the Web Appendix for more detailed discussion of these). The bulk of this research relies on data from weather stations. The perhaps most well-known large data set based on station observations is the CRU data set from the Climate Research Unit at the University of East Anglia.[4] This data set indeed has monthly data at down to a 0.5×0.5 degree resolution for much of Africa. Since weather stations with uninterrupted time-series observations in most African countries are few and far in between, and their precise location is not even public information, the CRU data relies on extensive interpolation across time and space. This is not suitable for our purpose to exploit within-location variation.

To study African civil wars Miguel et al. (2004) use another popular data source: rainfall data from the Global Precipitation Climatology Project (GPCP), which relies on satellite images of cloud cover. But for our study the GPCP is unsatisfactory: its spatial resolution for rainfall data is a coarse 2.5×2.5 degrees, and we need consistent temperature data to predict malaria transmission risk.[5]

Instead, we rely on *re-analysis*.[6] Specifically, we use data archive ERA-40

[4]See the webpage at `www.cru.uea.ac.uk/cru/data/`

[5]Higher resolution data (1.0×1.0 degree) is available only after October 1996.

[6]See Auffhammer et al. (2011) for an account of re-analysis directed to economists.

from the European Centre for Medium-Term Weather Forecasting (ECMWF), with weather outcomes for every six hours, from September 1957 to August 2002, on a global grid of parallels and meridians at a 1.25×1.25 degree resolution (about 139×139 kms at the equator).[7]

The ERA-40 re-analysis begins with high-frequency historical observations from a variety of sources: weather stations, ships, aircraft, weather balloons, radiosondes, and – most importantly – satellites. These are fed into the ECMWF's large-scale atmospheric circulation model (IFS CY23r4) to predict the atmospheric state, such as humidity and temperature, six hours earlier. The predictions are corrected by observations whenever available. Given this estimated time series of the atmospheric state, the climate model forecasts precipitation at every six-hour interval.

Re-analysis data, rainfall in particular, is subject to measurement error. But the precipitation data in ERA-40 do *not* depend on rainfall gauge data, which is particularly coarse and low-quality in Africa, but instead on the climate model and the estimated state of the atmosphere. Due to the well-known difficulty of predicting the precise location of convective rainfall (i.e. thunderstorms), the forecast may fail to predict the exact amount of rainfall in a precise location in a particular six-hour period. Aggregation in time (to a month) and space (to 1.25×1.25 degrees), however, resolves much of this problem. A comparison of rainfall data from ERA-40 and gauge data (where available) suggests that the ERA-40 data have less bias in the arid and semi-arid areas of Africa, where annual departures from regular seasonal fluctuations – our source of identification – are the largest.[8]

We expect this data set to contain among the very best available weather data for Africa. The climate model makes observations from data-sparse regions more realistic and reliable, as weather follows physical laws almost linearly at time scales such as six hours. This advantage is larger from the time global satellite data became available: in 1973 and, at higher frequency, in 1979. About 88% of the births in our sample are from 1979 or later.

Armed with the two sources of data, we match each DHS cluster to the ERA-40 grid cell that contains it (by ArcGIS 9.3's Spatial Join tool). These matched grid cells and clusters are plotted in Figure 1. With 17,568 clusters and 743 grid cells, we have almost 24 clusters per average grid cell. For each

[7]See Uppala et al (2006) for an overview and details on the methodology behind the ERA-40 archive, as well as a (partial) validation of the data.

[8]Zhang, Körnich and Holmgren (Forthcoming) discuss the quality of re-analysis data – from different sources, including ERA-40 – for the Southern hemisphere in Africa.

cell, we extract six-hourly data on rainfall and temperature from ERA-40, and aggregate to monthly data. Effectively, this gives us a large, balanced panel data set of weather outcomes, with 743 cross-sectional units and 540 (12×45) monthly observations for each unit. Summary statistics, by clusters and various subgroups (defined below), are reported in Table 1.

Seasonal fluctuations in African weather The monthly frequency of our data is important, as the most salient aspect of African weather is its strong seasonal fluctuations, with large rainfall variations between dry and wet periods in a typical year – especially in arid and semi-arid areas. Continent-wide rainfall patterns are governed by the so-called Inter Tropical Convergence Zone, along which convectional thunderstorms form daily and dump large amounts of scattered afternoon rains. This zone moves north and south with the seasons, causing large rainfall variations between dry and wet periods in a typical year (for more details see the Web Appendix).

On top of this regular seasonal cycle, however, temperature and rainfall fluctuate considerably from year to year, especially in arid and semi-arid areas. The fluctuations partly reflect chaotic weather dynamics over horizons beyond a couple of weeks. But they are also due to poorly-understood, and thus hard-to-predict, medium-term fluctuations in air pressure associated with the Southern Oscillation.[9] The warming phase (El Nino) is generally associated with wetter-than-normal weather in East Africa during March-May, but less rainfall than normal in parts of South and Central Africa during December-February, with opposite patterns during the cooling phase (La Nina).

The fluctuations around the regular seasonal cycle play a crucial role in our analysis. In particular, malaria, as well as crop yield (in rain-fed agriculture) require a certain amount of rain. Thus, we expect local, annual fluctuations in the timing and amount of rainfall to map into fluctuations in the incidence of malaria and malnutrition. These, in turn, will show up in local infant mortality.

[9]The time series pattern of these fluctuations during the past century are analyzed and discussed e.g., in Zhang, Wallace and Battisti (1997).

3 Malaria

We start this section by a brief and selective overview of the epidemiology, immunology and pathology literatures on malaria and infant death. Drawing on this overview, we focus on malaria during pregnancy as the channel whereby weather affects the survival of infants.[10] Next, we describe our index to measure weather conditions suitable for malaria infection, and use it to classify our different DHS clusters into different zones of malaria risk. Then, we present and discuss our econometric methodology and some results for the full sample and different malaria zones. Our basic results clearly indicate that site-specific weather shocks to malarious conditions only have a significant effect on infant death in African areas with epidemic malaria. Thus, we look closer at subsets of these epidemic areas, allowing for a non-linear effect in malarious conditions.

Malaria and infant mortality Black et al. (2010) suggest that malaria caused about 16% of deaths of children under the age of five in Africa, while Snow et al. (1999) estimate that about 75% of the estimated malaria death toll in sub-Saharan Africa in 1995 is made up of children less than five. However, infants are known to have a reduced sensitivity to malaria during the first few months of life (Maegraith 1984).

Malaria in pregnancy[11] is known to raise the likelihood of low birthweight, a major risk factor for infant death (McCormick 1985). Guyatt and Snow (2001) show that the risk of low birthweight doubles if a baby's mother is infected with malaria during pregnancy, and that 5.7% of infant deaths in Africa might be attributed to low birthweight induced by maternal malaria.[12] The exact mechanism remains unclear, although insufficiency of a malaria-infected placenta is thought to lead to intrauterine growth retardation and premature delivery (Brabin et al. 2004). Placental infection by malaria parasites in African pregnant women is quite frequent.[13]

[10] By malaria, we mean the infection in humans caused by Plasmodium falciparum, the most deadly species of malaria parasites, which is the most prevalent in Africa.

[11] See Desai et al. (2007) for a recent and extensive review of the medical literature on malaria in pregnancy.

[12] Studies reviewed by Steketee et al. (2001) attribute 3 to 8 % of infant mortality to maternal malaria.

[13] For African areas with endemic malaria, the median infection rate in the studies reviewed by Gyatt and Snow (2004) is 26%, with a range of 5 to 52%. Desai et al. (2007)

Given the immunity of infants to malaria during the first few months of life, malaria in pregnancy may have a more profound effect on infant survival than infants' own infection after birth.[14] Thus, we focus on weather-induced variation in malaria incidence while the child is in utero on the subsequent risk of infant death, although we briefly discuss exploratory estimates of malarious weather conditions on mortality during the first year of life.

The medical literature suggests several factors that may raise the risk of infant death due to maternal malaria. One such factor is the annual prevalence of malaria. Where malaria is endemic, adult women develop partial immunity after repeated infections since childhood and thus avoid symptoms such as fever and anemia during pregnancy. Where malaria is seasonal or epidemic-prone, however, adult women lack in immunity. As a result, once infected with malaria, pregnant women get sicker; e.g., they get fever, which is known to increase the chance of premature delivery and of infant death (Luxemburger et al. 2001). Also, malaria mortality in general is known to be much higher in epidemic areas (Kiszewski and Teklehaimanot 2004). For these reasons, we strongly expect the impact of variations in malarious conditions on infant mortality to be larger in areas where malaria transmission is low. Based on the medical literature (see the Web Appendix), we also investigate whether the risk of infant death due to malarious conditions vary by certain individual characteristics of the mother.

Measuring malarious weather conditions The incidence and prevalence of malaria in a given area and time depend on a host of factors, including climatic, biological, geographic, and socioeconomic conditions. Researchers have tried to combine clinical measures of malaria prevalence into estimates of the spatial distribution of malaria in so-called malaria maps (e.g., Kiezowski et al. 2004, Hay et al. 2009). In this study, we exploit the weather-induced variability of malaria-prone conditions over time within each area.

review studies conducted in low-malaria transmission areas and report a median prevalence of placental infection of 6.7%.

On top of a higher likelihood of low birthweight, babies born to mothers with an infected placenta are reported to be more likely to develop a malaria infection during the first year of life (Le Hesran et al. 1997). They may also become susceptible to measles earlier than other babies due to reduced placental transfer of maternal antibody (Owens et al., 2006). Measles is estimated to account for about 12% of deaths of children under the age of five in sub-Saharan Africa in 1990 (Murray and Lopez, 1996, Appendix Table 6f)

[14]Snow et al. (2004) argue that looking only at the direct cause of death would significantly underestimate the impact of malaria on child death.

A necessary condition for malaria to spread is the growth and survival of parasites causing the disease and vectors (a certain mosquito species) transmitting the parasites. Growth and survival are known to heavily depend on temperature and rainfall. To capture these conditions in a parametric way, we follow Tanser, Sharpe, and le Seuer (2003), who propose a parsimonious weather-based index for malarious conditions for Africa. This index builds on the comparison of long-term (1920-80) *mean* monthly rainfall and temperature with monthly profiles of malaria transmission intensity in 15 locations that differ in malaria prevalence rates, as well as on published biological ranges affecting both vector and parasite development. Their monthly predictions of malaria transmission are empirically validated against the malaria occurrence data from about 3800 parasite surveys across Africa. The index correctly predicts 63% of malaria transmission incidents and 96% of the absence of malaria transmission.[15]

Following Tanser et al. (2003), we postulate:

Definition 1 *If and only if all of the following four conditions are satisfied, we set our binary malaria index for month τ in grid cell g, $Z_{g,\tau} = 1$:*

(a) *Average monthly rainfall during the past 3 months ($\tau-2, \tau-1, \tau$) is at least 60mm.*

(b) *Rainfall in at least one of the past 3 months is at least 80 mm.*

(c) *No month in the past 12 months ($\tau-11$ to τ) has an average temperature below $5°C$.*

(d) *The average temperature in the past 3 months ($\tau-2, \tau-1, \tau$) exceeds $19.5°C$+SD(monthly temperature in the past 12 months),*

If any one of conditions (a)-(d) fails, we set $Z_{g,\tau} = 0$.

Conditions (a) and (b) ensure the availability of breeding sites for the vector and sufficient soil moisture for the vector to survive; (c) is required for the

[15] A high probability of correctly predicting the disease absence is remarkable given that these parasite survey sites were chosen because of their potential for transmission. A modest probability of correctly predicting the incidence of malaria is presumably due to socio-economic factors that prevent malaria transmission despite the suitable weather conditions.

survival of the vector, as it quickly dies off at lower temperatures; and (d) allows the parasite to become infectious inside the vector's body before the vector dies.[16] The required threshold of temperature is higher, the higher the standard deviation of monthly temperature, because, after a cold winter, the populations of parasites and vectors need to be quickly regenerated to the level sufficient for malaria transmission.[17]

Climatological conditions thus influence malaria prevalence. In some areas, malaria is *endemic*, with high risk of malaria at least a good part of every year. In *epidemic* areas, malaria spells are more short lived because transmission is seasonal (recurs in particular months due to stable variations in rainfall and temperature) or unstable (present in some years but not in others). Finally, in *non-malarious* areas, the climate is too dry or too cold for malaria to be present or infectious at all.

As mentioned, we expect a larger effect on infant mortality of seasonal weather shocks in epidemic areas, due to lower immunity and more severe malaria infections. To test this hypothesis empirically, we divide the ERA-40 grid cells (and thus DHS clusters) into three different malaria zones. Non-malarious zones have no single malarious month, defined by malaria index $Z_{g,\tau}$, over the entire sample period; epidemic areas have strictly positive average malaria exposure between 0 and 4 months; while endemic areas have higher exposure rates.[18]

Our classification is illustrated in Figure 2. Non-malarious areas, in green, entail about 20% of the births in our sample and are found in the very North and South of Africa, in high mountain tracts (which are too cold), and in desert or near-desert regions (which are too dry). The remaining 80% of births are split almost equally into the other two categories. Epidemic areas, in yellow, are mainly found in the Sahel, in higher East African terrain, and

[16]The vector obtains a parasite by biting a malaria-infected person. But it takes time for the parasite to become infectious and thus for the vector to transmit malaria by biting another person. Higher temperature both shortens the time required for the parasite to become infectious and helps the vector survive long enough.

[17]The definition for our binary malaria index is slightly different from that in Tanser et al (2003). Even if a month fails to satisfy all the four conditions, they treat it as malarious if it is sandwiched by two malarious months. A priori such a sandwich condition may make sense in their cross-sectional context, but it makes less sense in our time-series context. When we implement the index exactly as Tanser et al (2003), the results are weaker presumably due to less time variation. Dropping separately each of the four conditions, we find conditions (a) and (d) to be the most relevant in predicting infant death.

[18]We have also set the epidemic-endemic split at 6 months with similar results.

in dry areas of the South. Endemic areas, in red, are found in tropical Africa with stable warm and humid conditions throughout the year. These three zones, based on weather conditions alone, correspond reasonably well to the distribution of actual cases of parasite infection in malaria maps, based on cross-sectional clinical observations (see e.g., Hay et al. 2009).

For each birth in our sample, we then create a measure of maternal malaria exposure during the 12 months up to the birth month. Specifically, for children born in a cluster within ERA-40 grid cell g and in running month t, we define

$$z_{g,t} = \sum_{\tau=t-11}^{t} Z_{g,\tau} \ . \tag{1}$$

In words, we ask for how many months in the year before birth a mother was exposed to malarious weather conditions. This varies substantially across areas and time. Mothers in endemic areas are on average exposed to 7.9 months of malarious conditions, with a standard deviation of 1.0 months. In epidemic areas, the corresponding numbers are 1.8 and 1.0 months. Mean-adjusted variability is thus much higher in epidemic areas. (See Table 1, Panel B for summary statistics on $z_{g,t}$).

Econometric specification We start the econometric analysis by estimating linear panel regressions of the following type:

$$m_{i,c,x,t} = \beta z_{g,t} + \alpha_{c,s} + \alpha_{x,y} + \varepsilon_{i,c,x,t} \tag{2}$$

The dependent variable, $m_{i,c,x,t}$, is the binary indicator, indicating death at the age of 12 months or less, for child i, born in cluster c, in grid cell g in country x, and running month t (calendar month s of year y).[19] We multiply this indicator by 1000 so that our results square with the conventional way of measuring infant mortality.

On the right-hand side, our parameter of interest β measures how many more infants per 1,000 die of one more month suitable for malaria transmission in year before birth. Further, $\alpha_{c,s}$ is a *fixed effect* for cluster c and

[19]The standard definition of infant mortality is death *before* turning the age of 12 months. The distribution of age at death in the DHS data, however, has a peak at 12 months, suggesting some of the babies who died before turning 12 months old are reported to die exactly at the age of 12 months. Using the standard definition of infant mortality, we find somewhat smaller impacts of weather fluctuations.

calendar month $s = 1, .., 12$. That is, in the full sample we control for 12 monthly means in each of our 17,568 DHS clusters, making for over 210,000 fixed effects. This way, we are identifying β from the deviation within each cluster from its site-specific monthly mean. To non-parametrically allow for country-specific declining trends of infant mortality, in line with actual observations, $\alpha_{x,y}$ is a fixed effect for calendar year $y = 1957, ..., 2002$ in country x. This adds another set of 1260 (45×28) fixed effects. That is, we allow (non-parametric) trends in national health systems, policies, or economic conditions, which could conceivably be related to local weather realizations.[20] Finally, we cluster standard errors at the grid cell level.

Basic results The full-sample results are displayed in Columns (1)-(3) of Panel A in Table 2. Column (1) is the result of a "standard" panel regression, with fixed effects for clusters and years. Column (2) replaces the cluster fixed effects with cluster-by-month fixed effects, whereas Column (3) replaces year fixed effects with country-by-year fixed effects.

The point estimates of β are all positive, as expected. Evidently, taking the very local seasonal patterns of infant mortality and weather into account in Column (2) raises the point estimate at only a minor loss of precision. But the more general specification in Column (3) cuts the point estimate and renders it statistically insignificant. This specification absorbs all country-by-year malaria shocks in the fixed effect. The lower coefficient makes sense, as country-wide malaria shocks may have more severe consequences than purely local shocks, e.g., because infections might spread between neighboring areas.

However, this basic specification assumes a homogenous malaria effect across all of Africa – a very strong assumption. To test our prior of a larger effect in epidemic areas, we consider the endemic and epidemic subsamples, dropping non-malarious areas (which have a constant malaria index of 0). Estimates for endemic areas are shown in columns (4)-(6) of panel A. The point estimates for temporary malaria shocks are never statistically significant. This does *not* mean that malaria is not a large risk factor for infant death in endemic areas. Our identification of the effect hinges on the deviation from the average seasonal pattern of malaria transmission. As year-to-year variation in seasonal malaria transmission for endemic areas is not very

[20] For example, Kudamatsu (forthcoming) finds democratization has reduced infant mortality in sub-Saharan Africa while Bruckner and Ciccone (2011) find negative rainfall shocks led to democratization in Africa.

large (see Table 1 Panel B), most malaria-induced infant deaths are likely absorbed by the cluster or cluster-month fixed effects.

Panel B shows the results in epidemic areas. The estimated coefficients in Columns (1)-(3) follow the same pattern as in Panel A, but now the coefficient in the most general specification with national non-parametric trends is just below one and significantly different from zero at the 10% level. Mothers in an average epidemic cluster, who face three months above-normal malaria exposure face a raised infant-mortality risk of just below 3 per thousand (close to Sweden's total infant mortality rate).

The three remaining specifications in Panel B show that these results for epidemic areas are robust. Clustering standard errors at the grid-cell level, as in Column (3), allows for arbitrary serial correlation of infant mortality and weather in each grid cell. But weather and infant death may also be correlated across grid cells and between a certain cell in a certain month and its neighboring cells in following months. To allow for simultaneous spatial and temporal correlations, we cluster by 5 year-period and average malaria exposure (see the Notes of the table), giving a total of 36 clusters. As Column (4) shows, this yields standard errors for β slightly lower than those with grid-level clustering.

Our maternal malaria exposure index, $z_{g,t}$ in equation (**??**), is by construction highly non-linear in temperature and precipitation. The estimated β may therefore pick up some other non-linear effects of weather on infant mortality. In Column (5), we check for this possibility by controlling for cubic polynomials in rainfall and in temperature during the 12 months preceding each specific birth (see Web Appendix for details). The resulting estimate of β is a bit above 1 with a slightly higher standard error than in Column (3).

While the specification allows for (non-parametric) trends in infant mortality at the country level, it is conceivable that some confounding variation is more local. Thus, column (6) further controls for (linear) trends at the level of each grid cell to the specification in column (5). The resulting point estimate only drops a little bit and has a marginally higher standard error.

Non-linear effects All specifications in Table 2 assume that the impact on infant mortality is linear in months of malaria exposure. Since infant mortality is an extreme outcome, however, perhaps it is more closely related to extreme weather events. Table 3 shows estimates that relax the functional-form assumptions. We first disaggregate the epidemic area into

two subgroups, above and below 2 months of average yearly exposure, as illustrated in Figure 3. The immunity argument may suggest that weather increasing the susceptibility to malaria has its most pronounced effect where malaria occurs the most rarely. Columns (1) and (2) of Table 3 display the results when the linear specification in equation (**??**) is estimated on these two separate epidemic subsamples. However, the linear model does not produce very different estimates in the two samples.

To get further, we allow a non-linear response within each subsample to five different bins of malaria exposure, setting the omitted bin at average exposure. Columns (3)-(5) look at the 0-2 month exposure sample. In those places, the cream-colored regions in Figure 3, the distribution of malaria exposure is highly skewed to the left. Over 60% of the births have no malaria exposure at all. But about 1% of births are exposed to five or more months of malaria.

The sign and size pattern of the point estimates is exactly what one might expect: exposures above average are associated with positive and increasing point estimates, even though these are quite noisy. The most striking finding is the comparison of those pregnancies that have more than 6 vs. 1-2 (or 0) months of malaria exposure. A randomly long malaria season, i.e., a potential long epidemic, raises infant mortality by about 38 per 1000, compared to a malaria season with little or no exposure. This huge effect (recall that average infant mortality rate in the sample is about 100 per 1000) is consistent with mothers in these areas having little or no immunity at all. Columns (4) and (5) show that the result is robust to including cubic polynomials in rainfall and temperature, and ERA-40 cell-specific linear trends. As shown by the F-test, the polynomial terms are not statistically significant.

Columns (6)-(8) show analogous estimates for DHS clusters with 2-4 months average exposure. In this subsample, the orange regions in Figure 3, nearly 10 percent of the pregnancies have no exposure to malaria while 1% are exposed to 7 or more malaria months. The general sign pattern is similar to that in Column (3). That is, zero or very little exposure is associated with much lower infant mortality rates than above 6 months exposure. But now the difference depends on the specification. As the polynomial terms are now significant, the point estimates in columns (7) and (8) are the most appropriate. According to these, random elimination of malaria in a given year, saves about 10 infants in 1000, i.e., it brings down infant mortality by one percentage point. On the other hand, a rise in malaria exposure from the

average to above 6 months increases infant mortality by about 20 in 1000.[21]

Below we will refer to these malaria exposure episodes that have a significant impact on infant death as *malaria epidemics* (i.e. 5 or more malaria months in the 0-2 month area; 7 or more in the 2-4 month area). Out of 63,317 months in which we observe at least one birth in the epidemic areas, malaria epidemics hit 844 months, suggesting that our results are not driven by very few observations. Figure 4 shows the spatial distribution of the number of such epidemic months across epidemic areas. These events occurred in various places in Africa, but with a certain concentration to East Africa, especially the mountainous regions around the Great Rift Valley.

These findings are potentially very important for the consequences of a changing climate. In Section 5, we use them to estimate historical death tolls due to malaria, and to discuss the implications of climate change.

Other specifications Following the medical literature summarized in the Web Appendix, we have investigated if the impact of maternal malaria exposure is heterogeneous across different types of babies, mothers, or households. We have also investigated whether malaria shocks after birth contribute to death before the age of one. As further discussed in the Web Appendix, there is no robust evidence of neither heterogeneity nor in-life shocks playing a significant role.

4 Malnutrition

In this section, we explore how past weather events impact on infant death through malnutrition. We first construct indices capturing how weather affects food availability and nutrition relevant for infant mortality. These indices are validated against local crop prices. We then investigate how weather induced variation in nutrition affects infant mortality, using a similar specification as in the malaria case. Finally, we test whether the estimated effects are heterogeneous across birth seasons and household characteristics.

[21] We have also tried to distinguish areas with seasonal and unstable malaria, based on the standard deviation of the number of annual malaria months, within the epidemic sample. But this does not produce any stark differences in the estimated effect of malaria shocks.

Infant mortality and malnutrition Maternal malnutrition poses a major risk for infant health.[22] A lack of food during pregnancy diminishes the intake of calories and important micro-nutrients, which negatively affects the growth of the fetus in utero. This way, maternal malnutrition increases the risk of low birth weight, which in turn raises the risk of infant death through birth asphyxia and infections (McCormick 1985, Black et al. 2008). Most African infants are breast-fed, and thus have lower mortality risk than those who obtain non-breast milk liquid or solid food during the first six months of life (see e.g., Black et al. 2008, Table 4).[23] Consequently – and in analogy to the malaria section – we do not focus on variations in food supply after birth, but rather on weather-induced variations in the risk of maternal malnutrition during pregnancy and their subsequent effects on infant survival.

Maternal nutrition intake in Africa depends on local crop yields. Most African countries are agricultural economies – in 2004, some 55% of people on the continent were employed in agriculture (Frenken 2005, Table 2), and many more depend on agriculture in other indirect ways. In addition, transportation infrastructure in Africa is poorly developed.[24] Most people are thus largely dependent on the local yields of subsistence crops for nutrition (or on cash crops for income to buy food).

Crop yields in the non-tropical areas of Africa are crucially dependent on the seasonal rains falling in the *growing season* – i.e., the rainy period of the year discussed in Section 2. Irrigation of land plays a minor role, especially in Sub-Saharan Africa – only 6.4% of cultivated land was irrigated in 2004 (Frenken 2005, Table 12). We therefore use the total amount of rainfall during the growing season as a proxy for crop yield.[25]

Indices for weather impact on malnutrition We use measures of plant growth from satellite data to determine the growing season in each ERA-40

[22]For a recent review see Black et al., 2008

[23]One might think that food availability after the birth of a child is important for his or her mother to produce breast milk. However, as long as it is not very severe, maternal malnutrition is known to have little impact on the volume and composition of breast milk (see Brown and Dewey, 1992 for a review).

[24]Herbst (2000, Table 5.3) reports that the road density for the median African country around the year of 1997 is merely 0.07 kilometers per square kilometers of land.

[25]Lobell et al. (2008) use the growing season rainfall (and temperature) to predict crop yields in developing countries under the future climate change scenarios. Deschenes and Greenstone (2007a) also use growing season rainfall to predict agricultural profits in the United States.

grid cell. Plant growth is observable from a long distance, because growing plants reflect light at the infrared part of the spectrum and absorb light at the near-red part of the spectrum. We use such data made available through the Global Inventory of Modeling and Mapping Studies or GIMMS (Tucker et al. 2005), namely the so-called normalized difference vegetation index (NDVI). The NDVI index is globally available as bi-weekly series from 1982 and onwards on a resolution of 8×8 kilometers (see the Web Appendix for further information)

To obtain the growing season from this time-series NDVI data, we use the TIMESAT program (Jonsson and Eklundh, 2004).[26] Following the common practice among ecologists (e.g. Heumann et al. 2007), this program extracts the start and the end of the growing season, defined as the time period in between 20% above one trough of the (smoothed) NDVI index to 20% above the next trough. To deal with the potential endogeneity of the observed annual growing seasons, we average the start and end dates over the 25 years available for each location, and use the calendar months between these two average dates as our measure of the *fixed* growing season.[27]

Next, we develop a simple index of how rainfall during the growing season affects each mother's nutritional intake for the 12 months before her child is born. The relevant growing season(s) of an individual birth depends on its timing relative to local harvest time. As an example, suppose a child is born in September 2000, two months after the last harvest in this location (July 2000). In the last year before giving birth, the mother has consumed food for two month from that harvest and for ten months from last year's harvest. In general, the mother's nutritional intake during the year before giving birth depends on the two last harvests. We weight these by the number of months the mother was able to consume from each harvest. In the example, our index weights rainfall during the growing seasons of 2000 and 1999 by weights 2/12 and 10/12, respectively.

To be more precise, we define the *rainfall exposure index*, our proxy for nutritional intake during the 12-month period up to birth, as follows.

Definition 2 *Consider babies born in location g in running month t. Let $r_1^{g,t}$ and $r_2^{g,t}$ be the total rainfall during the last and second-to-last (respectively)*

[26] We are grateful to Lars Eklundh, Department of Earth and Ecosystem Sciences, Lund University for his assistance with this program and the data.

[27] In areas where there are two growing seasons per year, we use every odd growing season in our calculation of the fixed growing season.

completed growing seasons preceding date t for location g. Further, let $h^{g,t}$ be the running month of the last harvest preceding date t in location g. We define the rainfall exposure index for location g in month t as

$$r_{g,t} = \omega_{g,t} r_1^{g,t} + (1 - \omega_{g,t})\, r_2^{g,t} \ , \tag{3}$$

where weight $\omega_{g,t}$ is given by $\omega_{g,t} = \frac{t - h^{g,t}}{12}$.

Underlying this index are three simplifying assumptions. First, all crop yields in location g become available at the final month of the growing season, and this harvest month, $h^{g,t}$, is the same calendar month every year. Second, yields harvested in months $h^{g,t}$ and $h^{g,t} - 12$ depend directly only on the cumulated rainfalls during the growing seasons that ended in those months ($r_1^{g,t}$ and $r_2^{g,t}$, respectively). Third, the marginal effect of weather variation on nutritional intake is constant across the year of exposure. The mean of the rainfall exposure index in the full sample is 70.0 centimeters (cm), while the average grid-level standard deviation is 19.2 cm.

Below we compare the performance of our rainfall exposure index with a simpler measure – the past 12-month rainfall – to provide suggestive evidence for the validity of the first two assumptions. We also relax the third assumption and investigate whether the marginal impact of harvested crop yields on children's health is larger during part of the year – the hungry season.

Since infant death is an extreme health outcome, it is plausible that it is closely related to extreme precipitation events, such as droughts or floods – in analogy with the malaria epidemics discussed in Section 3. We construct a drought index as follows.

Definition 3 *In each grid cell, we first compute the average value of our rainfall exposure index, $\overline{r}^g$, as well as its standard deviation, $\sigma^{r,g}$, using the full 45 years of ERA-40 data from 1957 to 2002 (irrespective of whether we observe child births or not). We then define a binary drought indicator variable for babies born in location g and running month t as*

$$d_{g,t} = I[r_{g,t} < \overline{r}^g - 2\sigma^{r,g}] \ . \tag{4}$$

That is, the birth is associated with a drought indicator of unity if its rainfall exposure index falls two standard deviations below the local mean. For

convenience, we define a flood symmetrically, as an extreme event in the opposite direction.[28]

Finally, we partially allow each unit of growing season rainfall to translate into different amounts of nutrition by dividing the sample according to the climate, to which people adapt by crop choice (e.g. drought-resistant crops such as millet and sorghum in arid areas). For this purpose, we rely on the well-known Köppen climate classification. Based on monthly mean temperature and precipitation as well as the latitude of the location, it distinguishes between several climate types that reflect vegetation differences (see, e.g., Peel et al. 2007 for more details). Using the Köppen classification criteria and our ERA-40 weather data, we subdivide all the DHS clusters in our sample into two climate zones: *rainy* areas (rainforest, monsoon, savannah and temperate climates), and *arid* areas (steppe and desert climates). The resulting classification of our DHS clusters is shown in Figure 5. Note that the arid climate zones largely overlap with the epidemic malaria zones in Figure 3.

The mean and standard deviation of the rainfall exposure index are 122.7 and 28.5 for the rainy climate zone, and 17.3 and 5.9 for the arid climate zone. Mean-adjusted variability is clearly larger for the arid climate zone. This sample split turns out to give us almost exactly as many births in the rainy and arid climate subsamples (see Table 1, Panel C).

Validation of the indices Before estimating their effects on infant death, we validate our rainfall and drought indices against local crop prices. If these indices measure local crop scarcity, then higher growing season rainfall should be correlated with lower local crop prices, and drought incidents should be correlated with higher crop prices. We exploit monthly crop price data between 1970 and 2002 for six major African crops in 424 local markets located in eight of the countries where we measure infant mortality. These data are obtained from USGS (2009).

We then regress these local crop prices on rainfall and drought indices. We exploit only the within-market monthly deviations from the local seasonal

[28]Our drought measure is similar to the Standardized Precipitation Index (McKee et al., 1993), but is based on our rainfall exposure index rather than just average rainfall. For a discussion of drought indices and their application to Africa, see Ntale and Gan (2003). We also interacted our drought index with the indicator of vulnerability to drought and flood from Dilley et al. (2005). However, this interaction variable did not significantly affect infant mortality.

mean, using a specification that is fully analogous to that used in our infant-mortality analysis. See the Web Appendix for more on the data construction and the econometric specification.

Table 4 reports the estimation results. Column (1) shows that in rainy areas the crop price drops significantly by 2.1% with a one standard deviation increase in rainfall during the previous completed growing season, i.e., $r_1^{g,t}$ in equation (**??**). A drought incident significantly increases the crop price by 6.7%. Column (2) shows that in arid areas the linear impact of growing-season rainfall on crop prices is insignificant, but a drought incident significantly raises crop prices by 9.5 percent. These results support our assumption that local rainfall affects the availability of food due to the lack of irrigation and transportation infrastructure.

Basic results We now investigate the effects on infant mortality of rainfall during the growing season. Table 5 reports the estimates from running panel regressions like equation (**??**) in Section 3, except that we replace the malaria exposure index $z_{g,t}$ with the rainfall exposure index $r_{g,t}$. Columns (1)-(3) show the estimates of the coefficient of interest in the full sample, with only cluster fixed effects or cluster-by-month fixed effects included, and with different treatment of trends. The point estimates always have the expected negative sign – i.e., more rainfall in the growing season(s) before birth cuts the risk of infant mortality. In the most conservative specification with country-specific non-parametric trends in Column (3), the coefficient is the highest in absolute value and is significantly different from zero at the 10% level.

Column (4) shows that the point estimate is lower in absolute value and not significantly different from 0, if we replace $r_{g,t}$ with the cumulated rainfall over the 12 months preceding birth, with no allowance for the location-specific growing seasons. This indicates that our rainfall exposure index captures the mothers' nutritional intake better than 12-month average rainfall.

Columns (5) and (6) report corresponding estimates when the same specification is estimated on the rainy and arid climate-zone subsamples. In both areas, the point estimates have the same negative sign as in the full sample, but are too noisy to be statistically significant.

Next, we investigate the effects of extreme precipitation events, captured by our drought and flood indices. Table 6 displays the results from adding the drought and flood indexes defined above to the econometric specification

used in Column (3) of Table 5. The full-sample estimates in Column (1) – the most conservative specification with cluster-month plus country-year fixed effects – show a positive, albeit statistically insignificant, point estimate for drought and an insignificant, negative estimate for floods. The results for rainy areas in Column (2) are similar.

In the arid-area subsample, the results are different. While the rainfall coefficient in Column (3) is insignificant, as in Table 5, the estimated coefficient on drought is positive and precisely estimated. The effect of a drought is now estimated to be quite powerful: it raises infant mortality by 23.1 per 1000, an amount equal to nearly a quarter of the sample mean. But we do not find any effect of extreme positive amounts of rainfall.

The remaining three columns in Table 6 check the robustness of the result in Column (3) in an analogous way to Columns (4)-(6) in Panel B of Table 2. Column (4) shows that the estimates are robust to clustering the standard errors at climate zones by 5-year periods.[29] In Column (5), we add cubic polynomials in the past 12-month temperature and rainfall to the regression, and obtain a similar point estimate for droughts. Finally, Column (6) adds ERA-40 cell specific linear trends and obtains a point estimate almost identical to that in Column (3).

Out of 69,303 months in which we observe at least one birth in the arid areas, droughts occurred in 181 months, suggesting that our results are not driven by very few observations in absolute terms. Figure 6 shows the spatial distribution of the number of such drought months across arid areas, analogous to Figure 4 for malaria epidemics. As seen from the figure, the droughts from which we identify our estimates are quite evenly spread over the various African regions that make up the arid climate zone.

Taken together, our results suggest that in arid areas extreme shortfalls of rain have large effects, while more piecemeal variations in precipitation do not have any measurable effects on infant mortality. These results are consistent with Susser (1991), who reviews studies on the relationship between maternal nutrition and birth weight and concludes that nutritional intake by mothers significantly affects birth weight only in famine conditions.

In Section 5, we use these results to estimate historical death tolls due to drought, and discuss implications of projected climate change.

[29]The arid areas are divided into 4 zones by northern versus southern hemispheres and by steppe versus desert climate zones, as defined by the Köppen climate classification.

Heterogeneous effects We now ask if the weather effects are heterogeneous across household characteristics and the seasons of birth.

First, do the effects depend on what time of year the infant was born? Given the earlier results, we focus on the effects of droughts in arid areas. The literature on agriculture and rural poverty in sub-Saharan Africa and other developing regions stresses the concept of the "hungry season", the period just after the start of the annual rains, when food stocks from the previous harvest are on the decline at the same time as the calorie expenditures are peaking due to extensive agricultural work (see e.g., the contributions in Chambers, Longhurst and Pacey 1981, and Sahn 1989). Indeed, low birth weights have been found to occur more often during rainy seasons than during dry seasons (Bantje 1983 and Kinabo 1993 for Tanzania, Fallis and Hilditch 1989 for Zaire). This suggests that annual fluctuations in weather-induced nutritional availability may have heterogeneous impacts on infant survival, depending on the season of birth.

To measure birth seasons, we construct four dummy variables indicating the number of quarters since the beginning of the growing season. For example, the dummy for quarter 1 equals one if the infant's birth month falls into the first quarter since the beginning of the growing season. The quarter immediately before the growing season is labelled 0 (instead of four). The average length of the growing season in arid areas is less than six months in our sample. Therefore, food is the least available in quarters 0 and 1. On top of that, quarter 1 is the time when energy expenditure of people – including pregnant women – reaches its peak over the year owing to the need for clearing the land and planting the seeds.

In order to estimate effects by season, we then regress infant mortality on our rainfall and drought indices, interacted with these four birth quarter dummies (see Web Appendix for details). Figure 7 plots estimated coefficients of the interaction terms with the drought index and their 95% confidence intervals. The vertical line in the figure indicates the beginning of the growing season. The babies born in the quarters around the beginning of the growing season, marked 0 and 1 in Figure 7, fare much worse in the wake of a drought shock than the babies born closer to the harvest. The estimated hike in death rates for babies born at the beginning of the growing season – on the order of 60 per 1000 births – is a stunning number indeed.

It is natural to compare our estimated effects of random variations in rainfall with the results from randomized controlled trials which vary nutritional intakes in other ways. In one study, dietary supplements were given to

a treatment group of randomly selected pregnant women in Gambia (Ceesay et al., 1997). The estimated effect of these supplements on the incidence of low birthweight and early infant death were both significantly larger for babies born in the hungry season. Our empirical findings suggest that these results from particular Gambian villages may likely be generalized to a much greater arid area of Africa.

Second, do the malnutrition effects vary by household characteristics? We focus on two specific sources of heterogeneity, which appear important a priori and reasonably measurable in the DHS data. One is occupation: we call an infant's household *agricultural*, when the parents earn a living only from agriculture at the time of the survey. In the full sample, about 42% of all children, excluding those with missing information on their parents' occupation, are born in agricultural households. Measurement error in the classification of agricultural households is inevitable: parents may have changed the job since the baby's birth, and the definition of agriculture in the DHS data also includes forestry and fishery. These factors, however, would bias our results against finding heterogeneous effects of weather fluctuations.

We also consider education, and define a child's household as *well-educated* if both the mother and her husband (if relevant) have more than eight years of education. Eight years is chosen as the cutoff, because we see a marked drop in the cross-sectional distribution of infant mortality above this level of education. In the DHS sample, only slightly more than 8% of the babies are born to well-educated households. The retrospective nature of the survey is unlikely to be a major source of mismeasurment when it comes to education. See Table 1, Panel A for summary statistics by subgroup.

To test for heterogeneous effects by household type, we then regress infant mortality on our rainfall and drought indices, interacted with the indicator of household type, agricultural or well-educated (see the Web Appendix for details). Similar to Table 5, column (3), we include cluster-by-month and country-by-year fixed effects, but now we also interact these fixed effects by household type.

Table 7 reports the estimated coefficients. Column (1) shows the estimates for the occupational breakdown in the rainy sample. In contrast to the results in Tables 5 and 6, the results suggest that rainfall exerts a significant negative linear effect on infant mortality for agricultural households. The sum of the main and interacted coefficients on the rainfall exposure index is statistically significant at the 5% level. The non-interacted coefficient shows that the effect is statistically insignificant and close to zero for non-

agricultural households. To the contrary, droughts have no effect on infant mortality in either groups in rainy areas.

In arid areas, the results in Column (2) show something close to the opposite. There is no linear effect of rainfall on infant mortality in either group. But a drought has a large effect in non-agricultural households, whereas it has no significant effect in agricultural households (the sum of main and interacted coefficients is not significantly different from zero).

A reasonable interpretation of these results is that in rainy areas a normal shortfall of rain tends to make agricultural households nutritionally worse off. On the other hand, in arid areas such households have better access to scarce crop yield at the time of drought, so the main burden is instead borne by non-agricultural households.

Columns (3) and (4) repeat the same exercise for the breakdown of household type by education. The main result here is two-fold. It is primarily the non-educated that benefit from more rainfall in rainy areas. And the well-educated appear to be protected from drought shocks in arid areas, perhaps as a result of higher purchasing power or better opportunities.

Discussion We have investigated separately two channels whereby local seasonal weather shocks affect infant mortality rates in Africa. While we have carefully defined these weather shocks according to the mechanism under investigation, all shock measures emanate from the same weather data. For example, more rainfall can potentially have two opposite effects on infant mortality: more rain may be good through increased nutrition but bad through increased malaria. For the babies in our sample, the malaria exposure index $z_{g,t}$ is indeed positively correlated with the rainfall exposure index $r_{g,t}$ with a correlation coefficient in the full sample of 0.76.

It is thus legitimate to ask if the results hold up when we allow both types of shocks to occur simultaneously. The brief answer is yes. As shown in a previous version of the paper, a few of the point estimates for the linear effect of growing season rainfall and the number of malaria months increase in absolute value. But all the important estimates of nonlinear effects – of malaria epidemics and droughts – are not affected.

5 Aggregate Effects

What can our results say about aggregate effects of weather and climate on infant mortality? We begin this section by discussing existing estimates in the medical literature and the methodology of the underlying studies. Next, we use our results in Sections 3 and 4 and the distribution of historical weather to estimate how many infant deaths during (part of) our sample period were caused by extreme malaria epidemic and drought episodes. We also estimate how many infants, in total, died from maternal malaria in Africa's epidemic regions. Finally, we pinpoint areas at risk for high infant mortality at the end of this century, now using our earlier results together with distributions of future weather from climate-model simulations fed by continued emissions of carbon dioxide.

Existing studies of infant deaths from maternal malaria Mortality estimates due to disease ideally come from national vital registration systems that categorize all deaths in the country by cause. However, such systems do not exist for most African countries. Existing medical studies therefore rely on two types of alternative data sources and methodologies. We now briefly describe these data and methodologies and compare them to the ones we will use.

The first type of study builds on epidemiological surveys, including randomized control trials (RCT), on the impact of medical treatment of malaria infection.[30] The paper with a scope most comparable to our own is probably Steketee et al (2001) – see the Web Appendix for a more detailed account. These authors rely on two clinical studies, which estimate the effect of malaria medicine on *birthweight* – *not* infant mortality, as the sample is too small – by using an RCT. Then, they combine this estimate with the correlation coefficient between low birth weight and infant mortality based on observational data. Finally, they scale the effects by regional infant mortality rates. This way, the paper finds that, globally, 75,000 to 200,000 infant deaths in each year might be attributable to malaria infection during pregnancy in

[30]There also exist some RCTs on the prevention of malaria. Insecticide-treated bed nets are, for example, estimated to reduce infant mortality by 5.5 deaths per 1000 births on average, according to five studies in endemic areas of sub-Saharan Africa (Lengeler, 2004). Preventive measures against malaria such as bed nets, however, only partially reduce malaria incidence and for this reason provide a lower bound on the effect of eliminating malaria.

endemic areas. This amounts to 3-8% of infant mortality.

A number of assumptions are necessary to draw these conclusions. To consistently estimate the effects of malaria on infant mortality in a studied population, one has to assume that: (E1) no factor correlated with birthweight directly affects infant mortality and (E2) malaria affects infant mortality only through birth weight. To correctly extrapolate to other areas and times, one requires: (E3) effects are proportional to the average infant mortality rate in the region and (E4) the level of malaria exposure in the local regions and specific times of the underlying RCT are representative of all regions and times.

In comparison, the estimates we will present here rely on random year-to-year weather shocks to identify effects on infant mortality. Consequently, our random assignment depends on weather fluctuation rather than explicit randomization (or assignment by day as in Stekeete et al. 1996). Because of the very large size of our sample (nearly a million births), we can estimate the effect on infant mortality directly and do not have to make assumptions such as (E1) and (E2) to draw conclusions. Our sample is also a lot more representative of all of Africa during a long sample period, and we measure the average effect across this sample. For this reason, we do not have to make assumptions (E3) and (E4) to extrapolate our results to other regions and times.

The second type of study relies on verbal autopsy reports, which attempt to assign cause of death based on signs, symptoms and events leading up to the death, as reported by caretakers of the deceased and sometimes supported by medical documentation. One example are estimates of malaria's contribution to mortality of African children below 5 (*not* infants) in the WHO's World Malaria Report or in Black et al. (2010). These are based on results from meta-regression studies – see the Web Appendix for more details. First, the share of deaths of different causes – including malaria – in a number of verbal autopsy studies is regressed on a number of observable factors, such as the endemicity of malaria. Then, the resulting regression coefficients are used to predict the distributions of under-5 deaths by cause for all of Africa. This way, Black et al. (2010) attribute 16% of under-5 deaths to malaria.

While such cross-sectional regression studies may predict the relative importance of mortality causes, they are not necessarily able to accurately identify causes of deaths. Three obvious concerns are: (V1) cross-sectional correlations may reflect confounding factors – factors that affect infant mor-

tality, like crop choice, may correlate with malaria endemicity; (V2) errors in the verbal autopsy reports may correlate with the meta regressors – if reports poorly differentiate malaria from other diseases causing fever (e.g., septicaemia, viral encephalitis or pneumonia), fever may more likely be seen as a sign of malaria in endemic areas than in non-endemic areas; (V3) children may die of multiple illnesses – extrapolating to the total effects of eliminating malaria is not straightforward, as the methodology focuses on the relative importance of mortality causes, taking the total number of deaths as given.

In comparison to the meta-regressions based on verbal autopsy reports, we use random time series variation within the same locality to identify effects. Confounding cross-sectional variation is thus absorbed by our cluster-by-month fixed effects to avoid concern (V1), a key motivation for our choice of methodology. Also, we do not rely on verbal autopsy reports and capture the direct effect on total infant mortality (as reported), avoiding concerns (V2) and (V3).

Existing studies of malnutrition and infant mortality How about the relation between malnutrition, droughts and infant mortality? Some estimates of the effects of malnutrition on *general* mortality are available in data bases such as EM-DAT of the Centre for Research on the Epidemiology of Disasters. Over the time period for which we will provide our estimates, namely 1981-2000, this data records 195 droughts in Africa 1981-2000, involving a total of 554,756 killed. Let us assume that infants make up 3% of the population (from the WDI crude birth rate), and are hit by droughts uniformly with all other ages (probably an overestimate, given the effects of breast-feeding discussed at the outset of Section 4). Then, this would imply an excess of 16,600 infant deaths due to drought over the twenty-year period. It is noteworthy that vast majority (99%) of the deaths recorded by EM-DAT are associated with a mere three droughts in the mid 1980s (Ethiopia, Sudan and Mozambique).

There is also a body of existing studies that provide estimates of the relation between malnutrition and *infant* mortality. One common approach is to use meta-regressions of (the log of) average infant mortality rates by weight-for-age categories on weight for age. The distribution of infant weight for age found in observational data is then used to compute the excess mortality relative to the ideal weight for age (see Pelletier 1994 and Black et al. 2008 for examples). A typical estimate is that one half to one third of

infant mortality is attributable to malnutrition and that mortality increases monotonically as weight falls, at least in a region from one standard deviation below the mean. Using this approach to predict infant mortality may run into problems, however, because social and disease related factors may affect both malnutrition and infant mortality. Thus, we cannot readily use results from such studies to estimate the causal effect, through nutrition, of weather variations or climate change on infant mortality.

Yet another approach is to rely on RCTs to assess the effect of nutrition on infant mortality. Kramer and Kakuma (2003) review the evidence on energy and protein intake in pregnancy and conclude that balanced energy/protein supplements improve fetal growth and may reduce the risk of fetal and neonatal death. The experiment in Ceesay et al. (1997), mentioned in Section 4, find large effects on fetal growth and mortality. To exploit results like these to estimate the effect of weather variation on infant mortality, one needs to know how the distribution of nutrient consumption across Africa changes with weather variations, as well as the average response of infant mortality to these changes.

Aggregate effects of malaria epidemics and droughts What is the excess infant mortality due to malaria epidemics across all epidemic regions of Africa? To answer this question, we begin by estimating the number of births for each year and ERA-40 cell in the epidemic regions of Africa from 1981 to 2000, a period for which we have reliable gridded population data (see the Web Appendix for details). We then multiply the estimated number of births with the increase in the probability of infant death in events with three or more malaria months above the average, implied by the estimates in Columns (5) and (8) of Table 3. This yields the excess infant mortality caused by each such malaria episode. Finally, we sum the excess infant mortality across all these malarious episodes.

If we restrict attention to the ERA-40 cells in our sample, the excess deaths during 1981-2000 is 84,000 out of 139 million births. Extrapolating to all malaria epidemic areas of Africa, we estimate that 106,000 infants died in these malaria episodes out of 179 million births in the same twenty years. This is 0.64 children in 1000 births, accounting for a bit less than 1% of infant mortality. We know of no existing study that is directly comparable to this estimate. The map on the left in Figure 8 shows that the epidemic areas stricken by malaria epidemics lie in the East (Ethiopia, Somalia, and

Kenya), the South (Zambia and Angola), and, in particular, the area around Rwanda and Burundi.

Analogously, we estimate the excess number of infants dying of droughts in Africa's arid climate zones. The calculation now relies on the estimate in Column (6) of Table 6. In the sample areas, we estimate an 8,700 extra infant deaths from these droughts, out of 141 million births. Extrapolating to all arid areas of Africa (the map on the right in Figure 8), we estimate 11,000 extra infant deaths, relative to 206 million births. This is only 0.05 children in 1000, accounting for 0.05% of infant mortality.

Our mortality toll from droughts is much more dispersed in time and space than the aforementioned estimates of infant deaths based on the EM-DAT data base. Our estimates capture a different phenomenon: extreme deviations from local rainfall conditions in arid areas that typically do not attract the attention of the large aid donors. Interestingly, the estimated aggregate infant mortality from these unnoticed disasters is of the same magnitude as that of the major disasters entering in EM-DAT: 11,000 vs. 16,600 excess infant deaths.

Total number of infant deaths from maternal malaria We can also estimate the total number of infant deaths caused by malaria in epidemic regions. Our estimate draws on the fall in infant mortality with zero malarious months in the year before birth relative to a typical year. This estimate is in the same spirit as comparing infants of mothers randomly assigned to malaria medicine treatment (and therefore non-malarious) with infants of mothers not given treatment. In our study, because of random weather fluctuations, some mothers were non-malarious and our estimate compares their infants to infants born under normal malarious conditions in this area.

In areas with 2-4 average malaria months, our estimates (column (8) in Table 3) imply that infant mortality drops by 8.1 deaths per 1000 with zero malarious months in the year before birth. Since the average infant mortality rate in these epidemic areas is 108.9 per 1000, maternal malaria is estimated to account for 7.4% of infant mortality (the 95% confidence interval is 4-11%). Interestingly, this is about the same size as the Steketee et al (2001) estimate of 3-8% for endemic regions.

Multiplying our estimate by the total number of births in a year, we get the reduction in infant mortality from eliminating malaria. We find that 629,000 infant lives out of 64 million births were lost due to malaria in these

epidemic regions in 1981-2000. In areas with 0-2 average malarious months, our point estimate of a drop to zero malaria is not significantly different from zero.

Areas at risk in the future We have identified African areas vulnerable (in terms of infant survival) to weather shocks, namely epidemic malaria zones and arid climate zones. We now analyze areas at risk when the climate changes. This analysis mirrors that of the aggregate effects of malaria and droughts we have just discussed, but substitutes historical weather and population distributions in 1981-2000 with projected distributions for 2081-2100 (see the Web Appendix for detail). The weather predictions rely on data from the EC-EARTH model, the climate model closest to the one used in the ERA-40 reanalysis. The data are based on two emission scenarios under the Climate Model Intercomparison Project phase 5 (CMIP5): a high-emissions scenario (RCP8.5) and a midrange-mitigation emissions scenario (RCP4.5).[31] For population, we rely on United Nations Population Division (2011).

We do this to demonstrate how our methodology can say something about the expected impact of climate change. An important caveat is that our estimates do not take into account future economic growth or e.g. developments of new medical treatments that may alter weather impacts. We also only use two weather scenarios, and relegate to future work taking into account uncertainty, generated by different scenarios in different climate models.

Table 8 summarizes our estimates of the aggregate number of infant deaths. For comparison, the first column reports our estimates of the historical death toll.[32] To better isolate the effects of climate and population change, we report (for each climate scenario) the estimates when population is held fixed at the 1981-2000 levels (columns 2 and 4) as well as when it is changed to the projected 2081-2100 levels (columns 3 and 5).

Panel A shows the estimated number of infants killed in malaria epidemics and droughts. The number of infant deaths in malaria epidemics is estimated at 84,299 in 1981-2000. Under the midrange-emission scenario, this number falls in 2081-2100: only 16,380 with population held constant, and 41,474

[31] See Taylor et al. (2012) for the detail on the CMIP5.

[32] Exact numbers differ from what we report in the previous subsections because the spatial resolution is slightly different from the one for the ERA-40 data, and the numbers in Table 8 are derived by matching the CMIP5 grid cells with the ERA-40 weather outcomes.

with projected population. Under the high-emission scenario, on the other hand, the death toll goes up: weather changes alone raise the number to 101,272, and population changes raise it further to 202,173.

The top two maps in Figure 9 show the areas at risk of infant death from (maternal) malaria epidemics in 2081-2100. Comparing these maps to the corresponding one for historical epidemics in Figure 8 helps explain the very different outcomes between the two emission scenarios. With high future emissions, a long east-west stretch of the Sahel suffers from epidemics in addition to the areas already hit during the late 20th century, namely the Horn of Africa and the vicinity of Lake Victoria, central Angola, and the borderlands of Zambia and the Congo – areas which all have high projected population growth. But with midrange future emissions, epidemics mainly fall in currently less populous regions of Africa (the Sahel), rather than the more populous areas stricken in the high-emission scenario.

The next row in Panel A shows the responses of drought-related deaths to climate change. They display quite a different pattern. In both emission scenarios, the number of infant deaths by droughts in 2081-2100 go up from the 1981-2000 level of 11,453. But in a seeming paradox, the high-emission scenario does not raise the death toll any more than the midrange-emission scenario: 19,976 compared to 18,513 with population held constant, and 36,601 vs. 52,869 with population projected.

The bottom two maps in Figure 9 show areas at risk of droughts in 2081-2100, holding in mind that the precise locations of these rare weather events are difficult to predict for existing climate models. Comparing these maps with Figure 8 reveals why the infant death toll by droughts goes up under both emission scenarios. First, larger areas are hit by droughts in 2081-2100 than in 1981-2000. Second, the most populated areas of Africa, around Lake Victoria, are affected in 2081-2100 while they were not in 1981-2000. The explanation for the seeming paradox above is that the midrange scenario leaves southern Somalia and eastern Kenya at risk, but the high scenario instead attributes droughts to areas along the border between Namibia and Botswana. According the UN population forecast, births in southern Somalia increase a great deal, while births in Namibia and Botswana actually go down by the end of the 21st century, as illustrated in Figure A5 (see Web Appendix).

Finally, we turn to the change in the total number of infant deaths due to maternal malaria in epidemic regions, which we can estimate for regions that remain non-malarious or epidemic under both climate change scenar-

ios (shown in Figure 10).[33] The results are displayed in Panel B of Table 8. With midrange emissions, climate change alone reduces the number of infant deaths by around 100,000. Population change, however, more than doubles this number to 268,180, higher than the 1981-2000 level of 214,429. The high emission scenario entails a different picture. Climate change alone doubles the number of infant deaths, and population change further triples it, resulting in a nearly six-fold death toll (1,273,329 compared to 214,429). Evaluated at the projected future populations, the effect of going from the midrange emissions to the high emissions scenario is one million additional infant deaths.

Figure 10 shows the geographical distribution of these infant deaths in maternal malaria: the left map for midrange emissions, the right for high emissions. The main difference between the two scenarios is Tanzania and Zambia, which stay in the 2-4 month epidemic areas in the high-emission scenario while they do not in the midrange scenario. These areas are densely populated and this accounts for the large difference in death tolls. Substantial increases in the projected number of births (Figure A5, Web Appendix) in these areas further magnifies this effect.

6 Final Remarks

We believe this paper makes substantive as well as methodological contributions. In terms of substance, we uncover two main channels whereby weather shocks impact infant mortality in African countries. Weather shocks that raise malaria exposure of pregnant mothers have a large impact on infant death, especially when they sow the seeds of a malaria epidemic in areas where malaria is rare. Drought shocks have a pronounced effect on infant

[33]Currently epidemic areas that will become endemic in 2081-2100 are mostly located on the southern edge of the Sahel region, southern Mozambique, and the coastal area of Angola under both emission scenarios. Very few areas transit from the endemic zone to the epidemic zone in the high scenario, while the southern edge of Congo-Kinshasa and some inland areas of Tanzania and Mozambique become epidemic in the midrange scenario.

Infant mortality in malaria may not be much higher in endemic than the 2-4 malarious regions. Existing RCT estimates of infant deaths in malaria in endemic areas (discussed above) are similar to ours in the 2-4 malaria-month region. We also do not find that infant deaths rise above average under temporary malaria shocks in endemic regions, and resposes to permanent shocks are probably even weaker because of adaptation. Thus, we are estimating the effects of changing malaria incidence were it is likely the largest.

death in arid areas, especially for babies whose parents are not well educated, not dependent on agriculture, and for babies who are born in the hungry season. The malaria epidemic and drought effects we estimate are statistically robust and quite large.

The results paint a bleak picture for the parts of Africa that receive little rainfall on average. Due to the erratic movements of the Intertropical Convergence Zone, these areas also face large weather variations. When it rains a lot this may cause a malaria epidemic, when it rains a little this may cause a drought – whichever way with bad outcomes for infant mortality.

In terms of methodology, we have outlined a possible research design for future impact research, showing how one may combine very different data sources for large-scale statistical work, when conventional data sources are absent or poor. We have also shown how the continental scale and scope of our study allows us to draw reasonable conclusions about the aggregate impacts for all of Africa in the past few decades. A similar approach and statistical methodology may be used to study other outcomes of interest in Africa or other regions. For example, further research on Africa could use DHS data to look at the weather dependence of other outcomes, such as fertility, child mortality, child health, or even generational spillovers, whereby girls with negative weather shocks in early life become physically or cognitively impaired adults facing a larger risk of bad outcomes when giving birth.

There is certainly scope for improvement on the natural-science side of our measurement. For example, one could use re-analysis from *regional* rather than *global* climate models to obtain more recent and fine-gridded weather data which better pick up the spatial distribution of rainfall, or use structural crop-yield models to get a better handle on the interplay between temperature and rainfall in producing local crop yields.

Finally, we have shown how some of our results can, in principle, be used to identify areas at risk for large health impacts in the wake of future climate change. On this front, much more work is needed to come up with robust conclusions – not the least to various forms of adaptation. All these tasks are left for future research.

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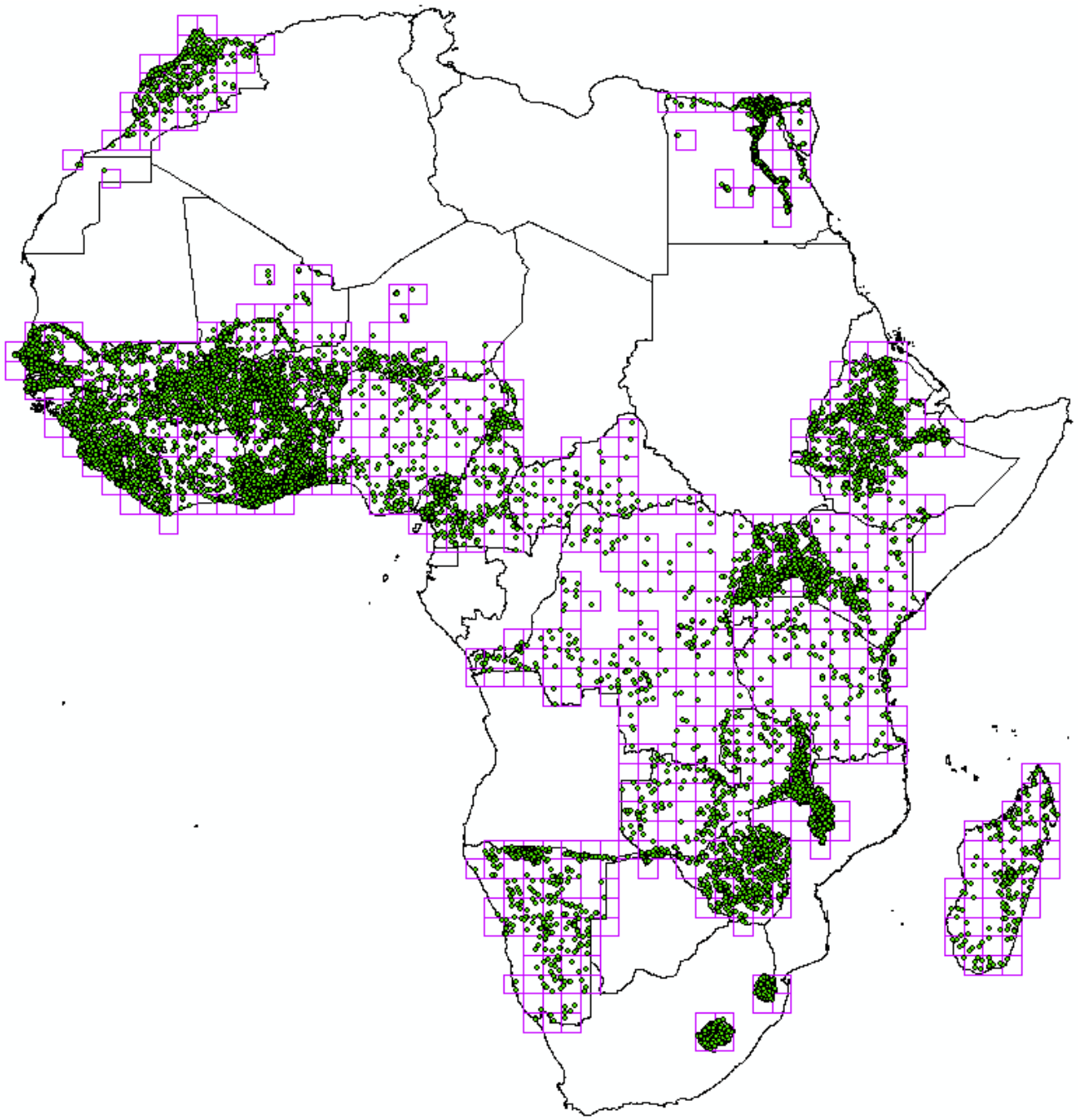

Figure 1 – ERA-40 Grid and DHS Clusters in the Sample

Notes: Purple squares indicate ERA-40 grid cells; green circles indicate DHS clusters.

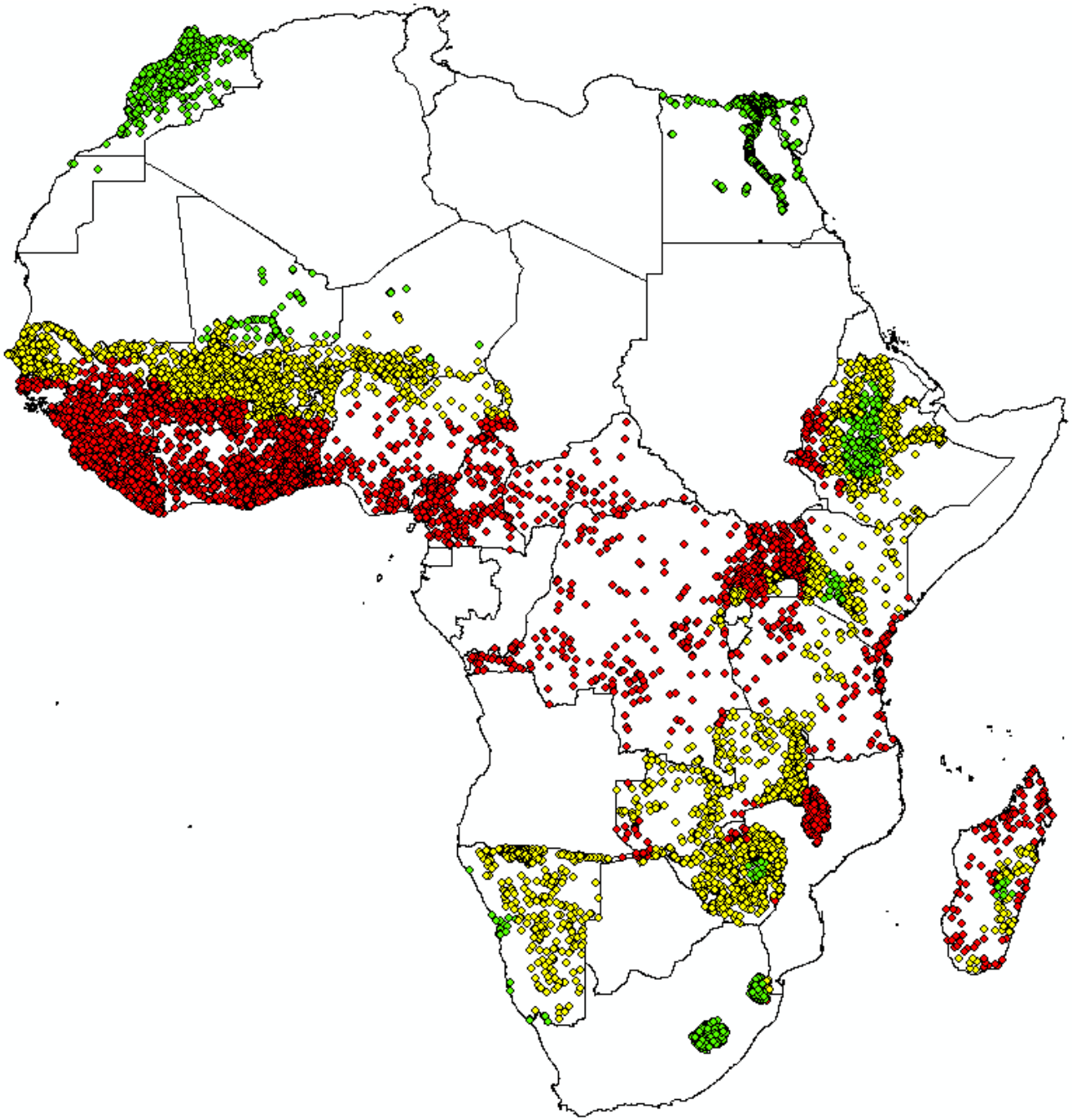

Figure 2 – Malaria Exposure Zones in Africa

Notes: Red, yellow, and green circles indicate DHS clusters in endemic, epidemic, and non-malarious areas, respectively.

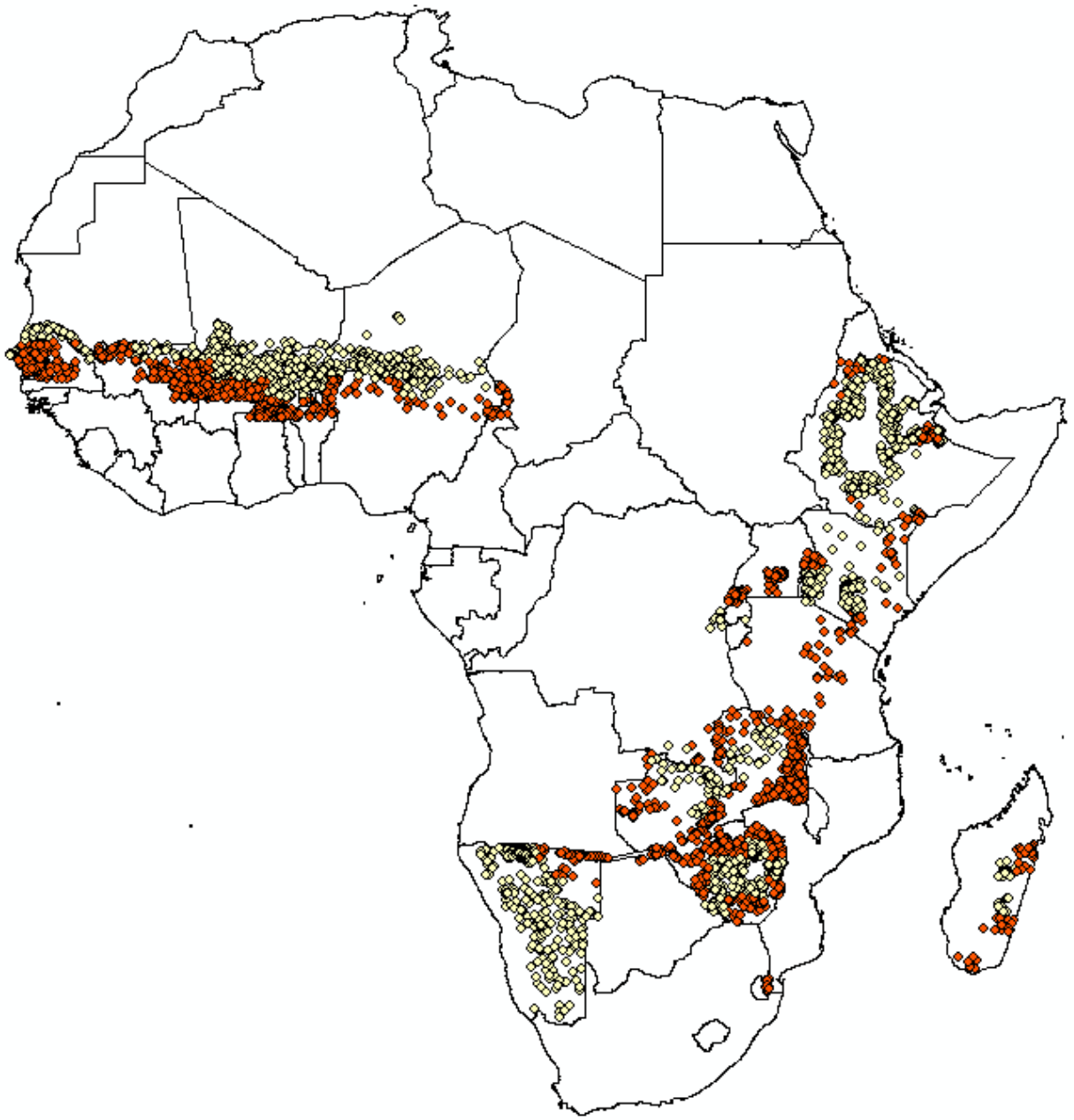

Figure 3 – Low and High Epidemic Malaria Exposure

Notes: Cream- and Orange-colored circles indicate DHS clusters with the average number of malarious months being 0-2 and 2-4 months per year, respectively.

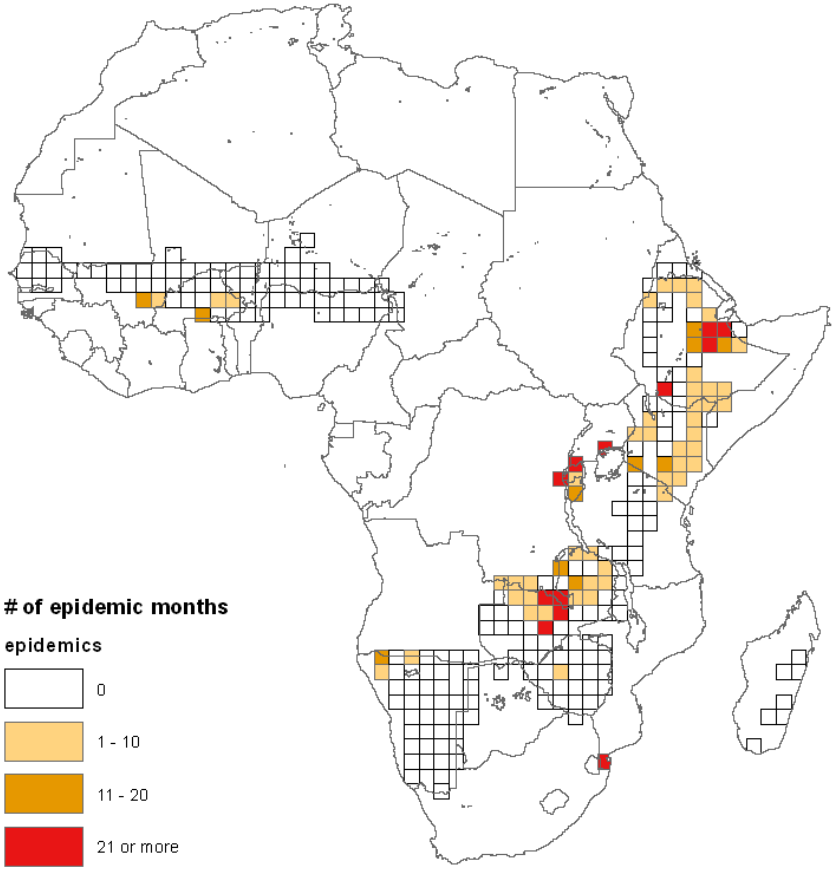

Figure 4 – Number of Malaria Epidemic Months in the Sample

Notes: Square cells indicate epidemic areas in the sample. The color scheme indicates the number of months in which we observe at least one birth *and* a malaria epidemic occurs in the sample.

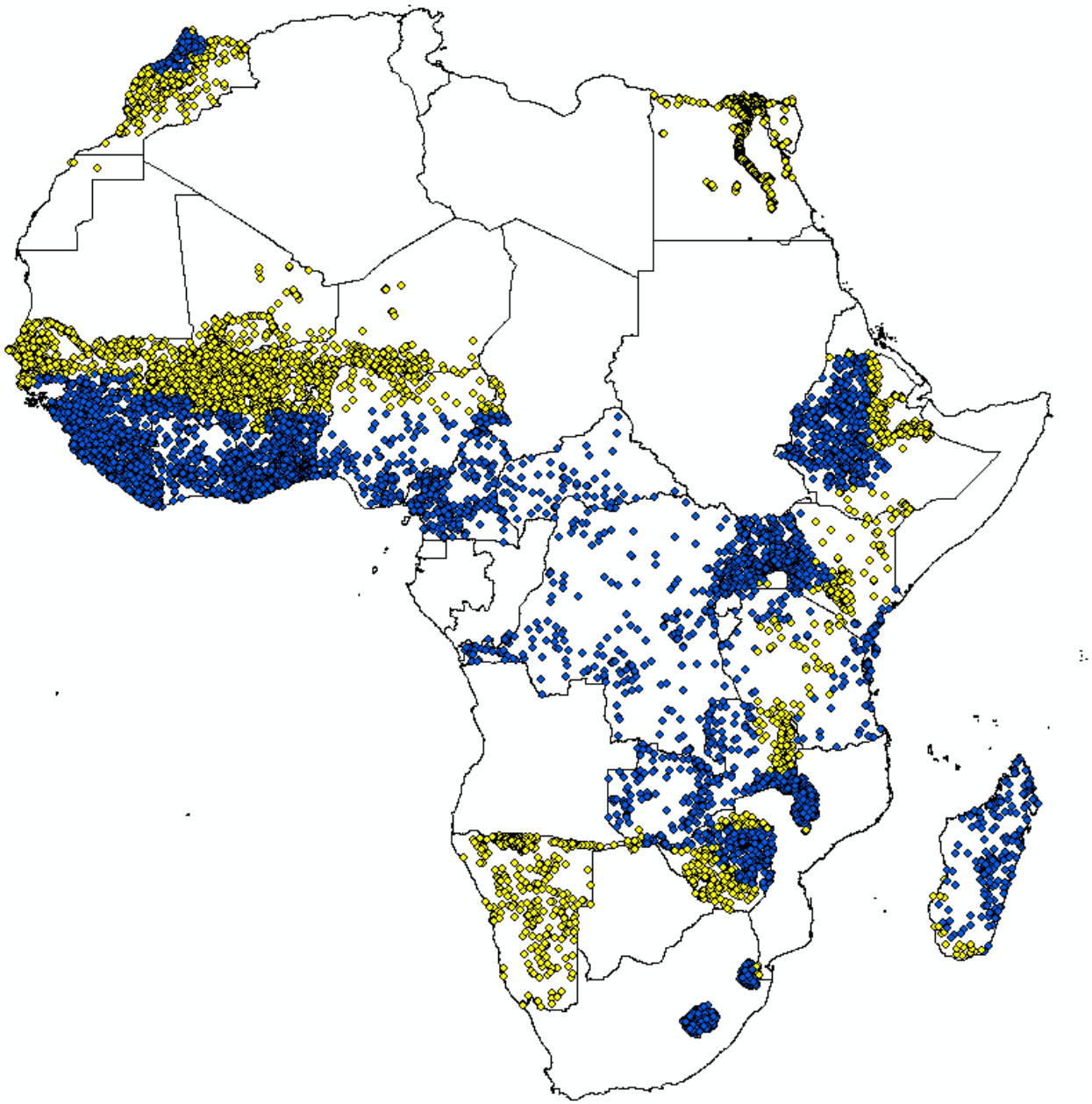

Figure 5 – Arid and Rainy Climate Zones in Africa

Notes: Blue circles indicate DHS clusters in rainy climate zones (Af, Am, Aw, Cs, Cw, and Cf in Koppen climate classification); yellow circles indicate those in arid climate zones (BS and BW). These climate zones are based on the average monthly temperature and total rainfall calculated from ERA-40 for the period 1957-2002.

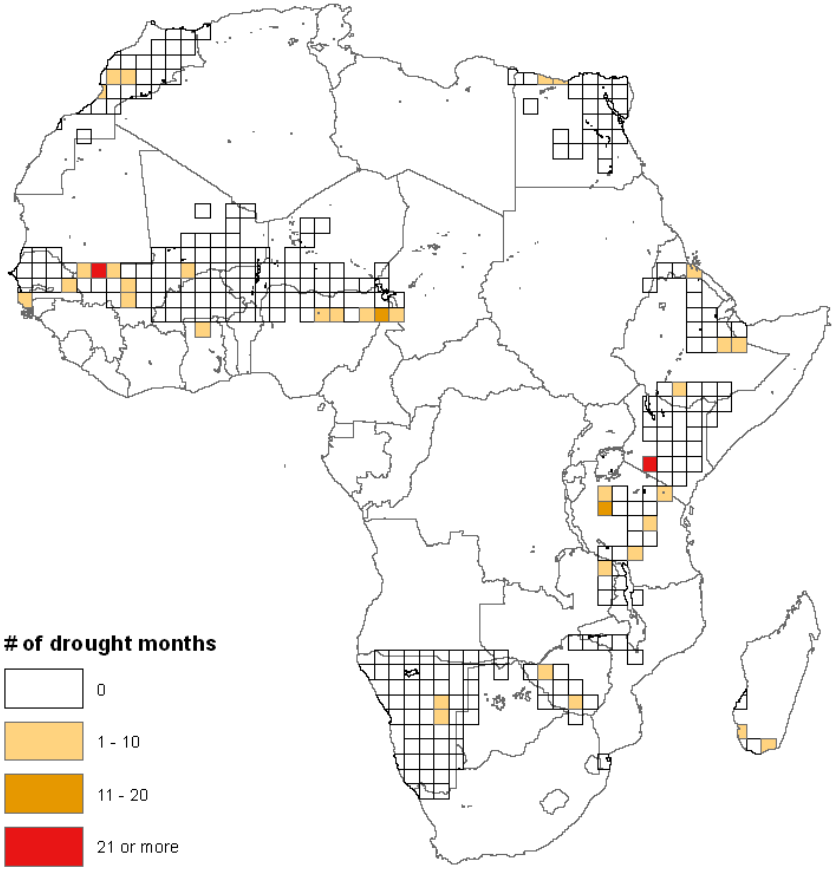

Figure 6 – Number of Drought Months in the Sample

Notes: Square cells indicate arid areas in the sample. The color scheme indicates the number of months in which we observe at least one birth *and* a drought occurs in the sample.

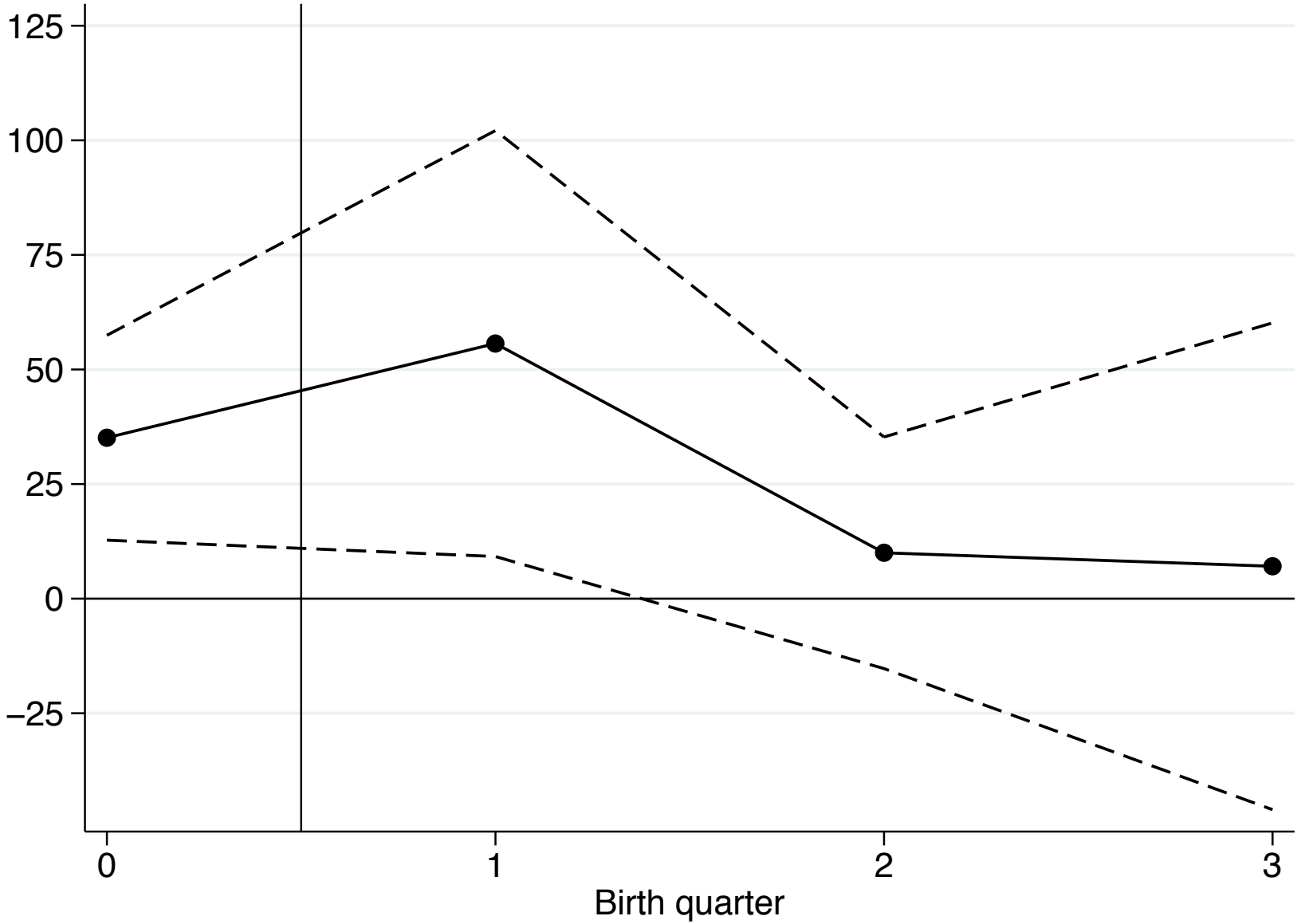

Figure 7 – Infant Death and Drought by Birth Quarter relative to the Beginning of the Growing Season

Notes: Plotted are the estimated coefficients of γ_k's in equation (6) in the text. Dashed lines indicate the 95% confidence intervals where standard errors are clustered at the ERA-40 grid cell level. The vertical line indicates the beginning of the growing season.

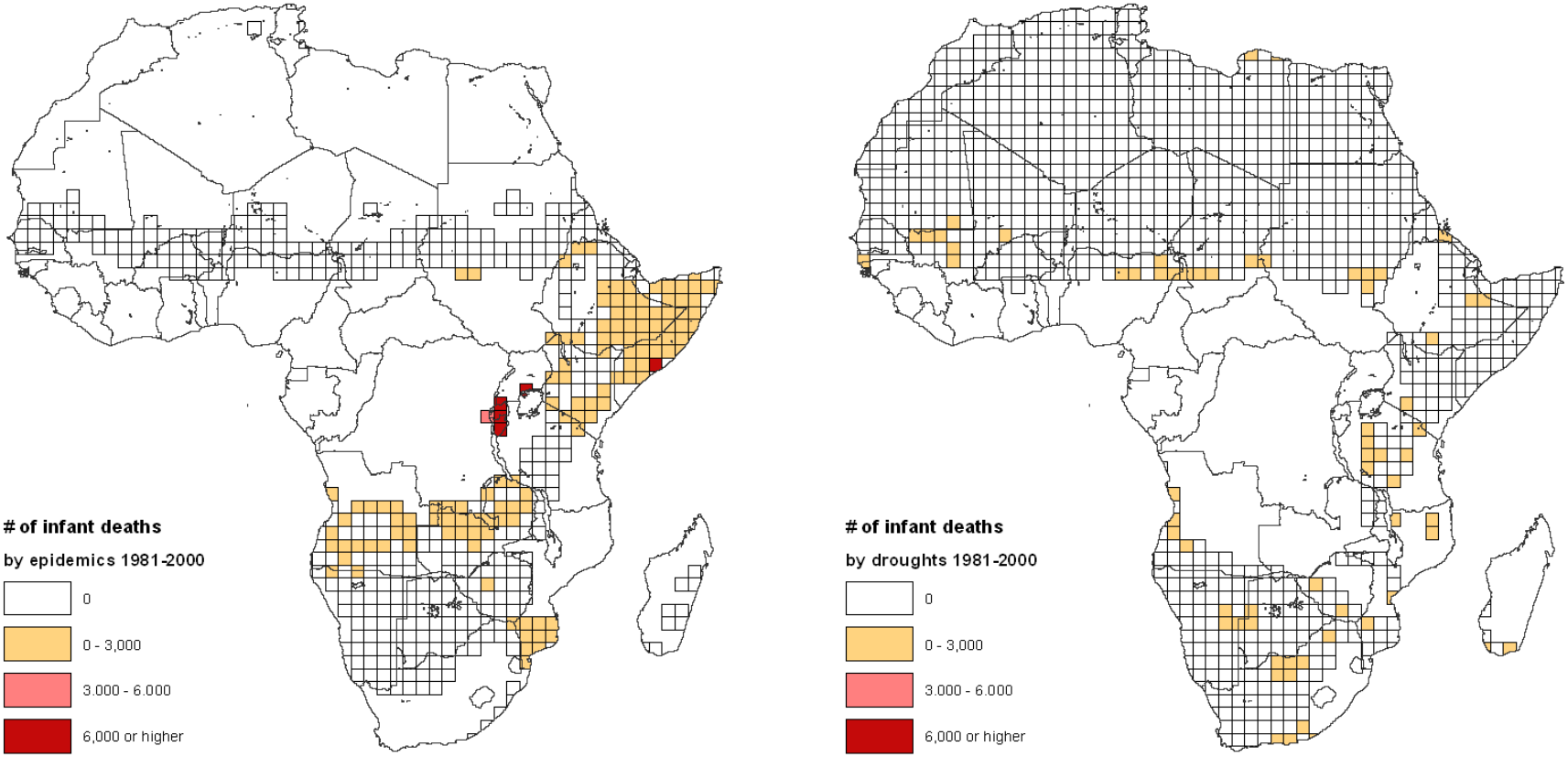

Figure 8 – Number of Infant Deaths by Weather Shocks during 1981-2000

Notes: Square cells represent epidemic areas in the left map and arid areas in the right one according to the 1957-2002 climate. The color scheme indicates the estimated number of infant deaths due to malaria epidemics (left) or droughts (right) between 1981 and 2000.

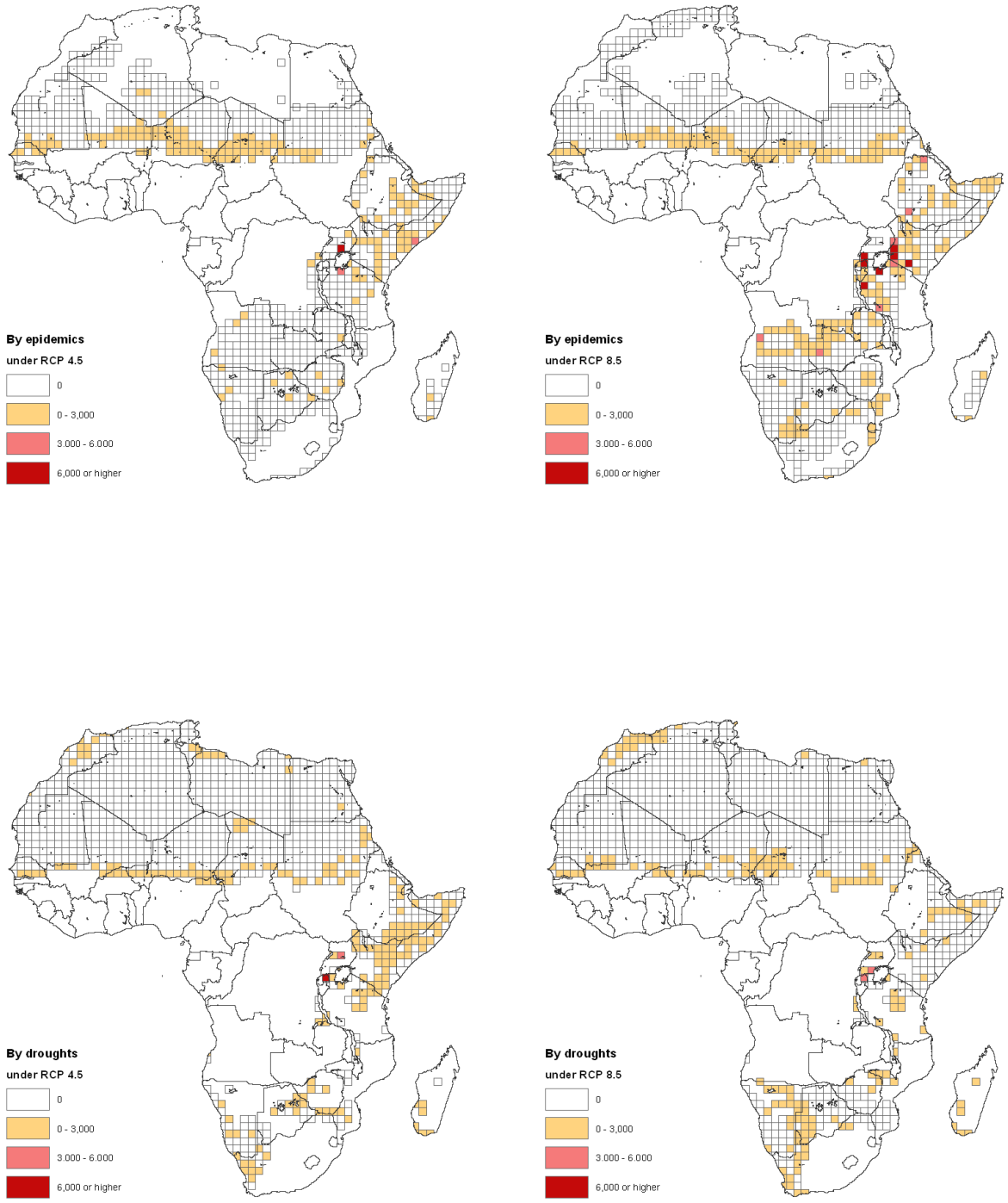

Figure 9 – Number of Infant Deaths by Weather Shocks during 2081-2100

Notes: Square cells represent epidemic areas in the top two maps and arid areas in the bottom two according to the 2051-2100 climate. The color scheme (the same as in Figure 8) indicates the estimated number of infant deaths due to malaria epidemics (top) or droughts (bottom) between 2081 and 2100 under the midrange emissions (left) and the high emissions (right) scenarios.

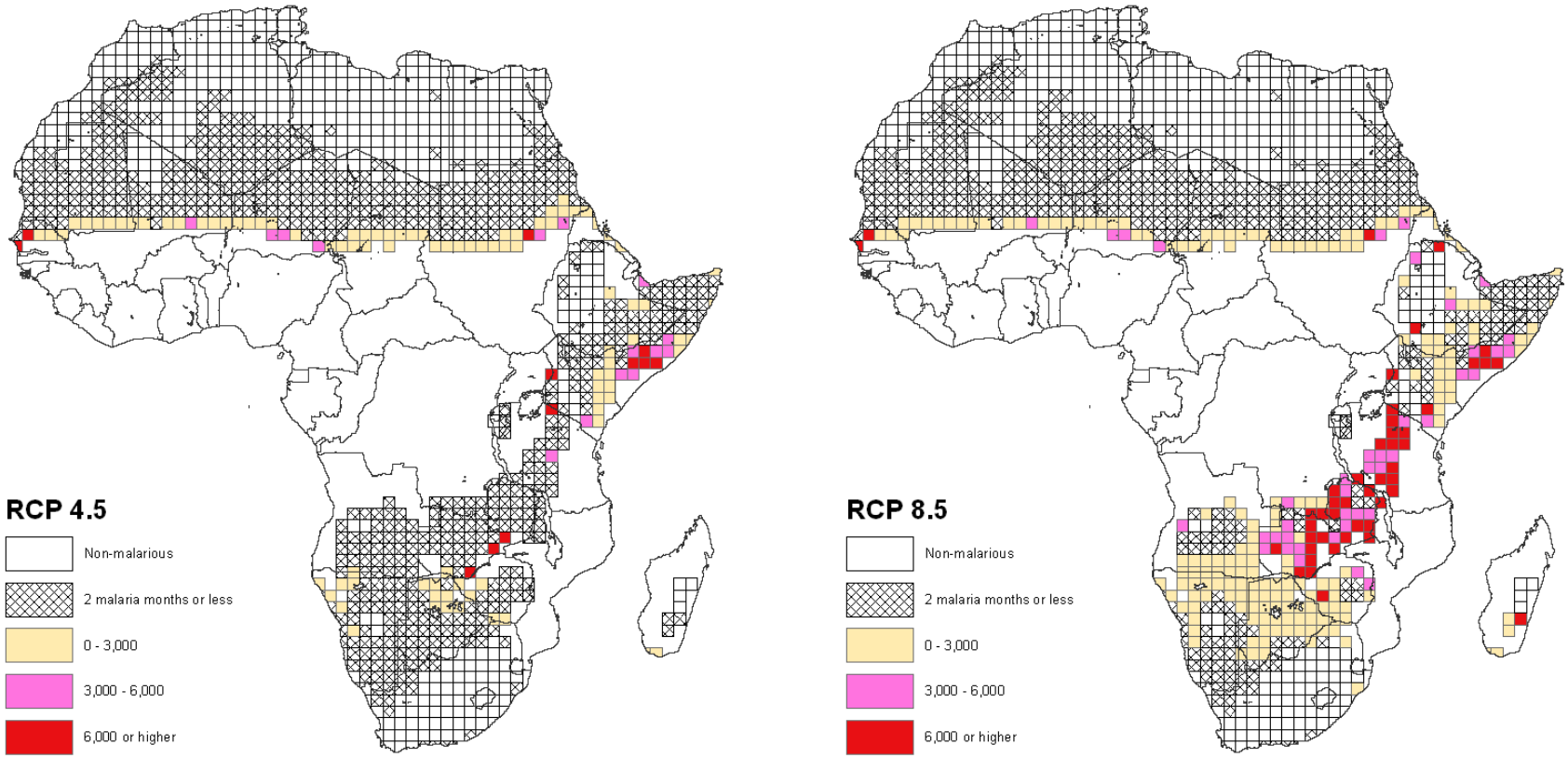

Figure 10 – Number of Infant Deaths by Malaria in the 2-4 month epidemic areas in 2081-2100

Notes: Square cells represent the areas that are never endemic in 1981-2000 or in 2081-2100 under both emission scenarios. Blank cells are non-malarious in 2081-2100. Hatched ones are the 0-2 month epidemic areas in 2081-2100. The rest in the 2-4 month epidemic area, and the cell color indicates the number of infant deaths by malaria.

Table 1 – Summary Statistics

	Sample mean	S.D. cluster-level means	Number of clusters	Number of observations
Panel A: Infant Mortality per 1000 live births				
Full sample	100.6	69.3	17568	962471
By area				
Endemic	108.5	73.6	7376	389116
Epidemic	107.1	68.8	5999	377361
Non-malarious	72.5	56.1	4193	195994
Rainy	103.0	71.6	9443	481018
Arid	98.2	66.1	8125	481453
By HH type				
Agricultural	119.6	111.2	12141	387605
Non-agricultural	85.6	92.2	16823	532866
Highly educated	46.2	119.0	8973	78103
Not highly educated	105.6	74.3	17420	880532

	Sample mean	Mean S.D. within-cell	Number of grid cells	Number of observations
Panel B: Malaria Exposure Index (months)				
Endemic	7.9	1.0	365	389116
Epidemic	1.8	1.0	275	377361

	Sample mean	Mean S.D. within-cell	Number of grid cells	Number of observations
Panel C: Nutrition Exposure Index (cm of rainfall)				
Rainy	122.7	28.5	439	481018
Arid	17.3	5.9	304	481453

Table 2 – Infant Mortality and Malaria: Basic Results

Dependent Variable: Infant death indicator (multiplied by 1000)

Panel A

	(1)	(2)	(3)	(4)	(5)	(6)
Sample	Full	Full	Full	Endemic	Endemic	Endemic
Malaria index in	0.53*	0.80**	0.33	0.06	0.18	-0.16
year before birth	(0.32)	(0.34)	(0.38)	(0.43)	(0.44)	(0.53)
Fixed effects	Cluster, Year	Cluster-month, Year	Cluster-month, Country-Year	Cluster, Year	Cluster-month, Year	Cluster-month, Country-Year
S.E. clustered at	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells
# of S.E. clusters	743	743	743	365	365	365
# of obs.	962471	962471	962471	389116	389116	389116

Panel B

	(1)	(2)	(3)	(4)	(5)	(6)
Sample	Epidemic	Epidemic	Epidemic	Epidemic	Epidemic	Epidemic
Malaria index in	1.18**	1.57***	0.94*	0.94**	1.24*	1.00
year before birth	(0.49)	(0.53)	(0.54)	(0.43)	(0.67)	(0.69)
Fixed effects	Cluster, Year	Cluster-month, Year	Cluster-month, Country-Year	Cluster-month, Country-Year	Cluster-month, Country-Year	Cluster-month, Country-Year
Polynomials	No	No	No	No	Yes	Yes
Cell linear trends	No	No	No	No	No	Yes
S.E. clustered at	ERA-40 cells	ERA-40 cells	ERA-40 cells	5-year by exposure	ERA-40 cells	ERA-40 cells
# of S.E. clusters	275	275	275	36	275	275
# of obs.	377361	377361	377361	377361	377361	377361

Notes: Robust standard errors in parentheses, clustered as indicated. * significant at the 10 percent level, ** 5 percent, *** 1 percent. Fixed effects included as indicated. The row "Polynomials" in Panel B indicates whether the cubic polynomials in the average monthly temperature and total precipitation over the 12-month period up to the birth month are included. The row "Cell linear trends" in Panel B indicates whether ERA-40 cell specific linear trends are controlled for. In Panel B column (4), "exposure" refers to four areas with above or below 2 malaria months per year, north and south of the equator, respectively.

Table 3 – Infant Mortality and Epidemic Malaria: Non-linear Effects

Dependent Variable: Infant death indicator (multiplied by 1000)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Sample	0-2 months avg. malaria	2-4 months avg. malaria	0-2 months avg. malaria	0-2 months avg. malaria	0-2 months avg. malaria	2-4 months avg. malaria	2-4 months avg. malaria	2-4 months avg. malaria
Malaria months in year before birth	0.42 (1.14)	0.93 (0.60)						
0 malaria months			0.30	0.93	1.40	-3.40	-7.52**	-8.14**
			(2.76)	(2.85)	(2.82)	(2.97)	(3.43)	(3.60)
1-2 malaria months						-7.15***	-8.86***	-9.30***
						(2.23)	(2.36)	(2.44)
3-4 malaria months			1.31	1.20	-0.24			
			(3.80)	(3.87)	(3.93)			
5-6 malaria months			15.62	14.14	11.55	-4.92	-3.55	-3.99
			(11.89)	(11.49)	(11.85)	(3.67)	(3.83)	(3.86)
>6 malaria months			38.44**	36.56**	35.95**	15.67**	20.79**	22.22**
			(15.62)	(15.74)	(15.17)	(7.70)	(8.21)	(8.95)
F-test (polynomials)				1.48 [0.190]	1.13 [0.346]		2.67 [0.018]	4.80 [0.000]
Polynomials	No	No	No	Yes	Yes	No	Yes	Yes
Cell linear trends	No	No	No	No	Yes	No	No	Yes
S.E. clustered at	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells
# of S.E. clusters	150	125	150	150	150	125	125	125
# of obs.	187858	189503	187858	187858	187858	189503	189503	189503

Notes: Robust standard errors in parentheses, clustered as indicated. * significant at the 10 percent level, ** 5 percent, *** 1 percent. Fixed effects for cluster-month and country-year are controlled for in all columns. The row "Polynomials" indicates whether the cubic polynomials in the average monthly temperature and total precipitation over the 12-month period up to the birth month are included. The row "Cell linear trends" in Panel B indicates whether ERA-40 cell specific linear trends are controlled for. The null for F-test(polynomials) is that the coefficients on polynomial terms are all zero.

Table 4 – Crop Price and Growing-season Rainfall

The Dependent Variable: Log Crop Price

	(1)	(2)
Sample	Rainy	Arid
Rainfall in previous completed growing season (centimeters)	-0.00046***	0.00005
	(0.00015)	(0.00036)
Indicator for rainfall in previous completed growing season < Mean - 2 SD	0.067***	0.095***
	(0.022)	(0.021)
Mean and SD of rainfall in previous completed growing season (centimeters)	85.5	25.2
	(45.7)	(18.4)
# of ERA-40 cells	85	75
# of obs.	109124	74631

Notes: Robust standard errors in parentheses, clustered at the ERA-40 cell level. * significant at the 10 percent level, ** 5 percent, *** 1 percent. Fixed effects for crop-by-market-by-month and for crop-by-country-by-year are included in both regressions.

Table 5 – Infant Mortality and Nutrition: Linear Effects

Dependent Variable: Infant death indicator (multiplied by 1000)

	(1)	(2)	(3)	(4)	(5)	(6)
Sample	Full	Full	Full	Full	Rainy	Arid
Rainfall (centimeters) in growing seasons associated with birth	-0.008	-0.012	-0.037*		-0.026	-0.070
	(0.018)	(0.019)	(0.022)		(0.024)	(0.104)
Rainfall (centimeters) in last 12 months				-0.011		
				(0.013)		
Fixed effects	Cluster, Year	Cluster-month, Year	Cluster-month, Country-year	Cluster-month, Country-year	Cluster-month, Country-year	Cluster-month, Country-year
S.E. clustered at	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells	ERA-40 cells
# of S.E. clusters	743	743	743	743	439	304
# of obs.	962471	962471	962471	962471	481018	481453

Notes: Robust standard errors in parentheses, clustered as indicated. * significant at the 10 percent level, ** 5 percent, *** 1 percent. Fixed effects included as indicated.

Table 6 – Infant Mortality and Nutrition: Nonlinear Effects

Dependent Variable: Infant death indicator (multiplied by 1000)

	(1)	(2)	(3)	(4)	(5)	(6)
Sample	Full	Rainy	Arid	Arid	Arid	Arid
Rainfall (centimeters) in growing season	-0.033	-0.025	-0.039	-0.039	-0.054	0.036
	(0.023)	(0.025)	(0.110)	(0.066)	(0.100)	(0.098)
Drought (0,1) in growing season	8.41	-2.64	23.1***	23.1***	22.3***	23.1**
	(8.19)	(12.2)	(8.49)	(7.69)	(8.53)	(9.32)
Flood (0,1) in growing season	-1.26	-0.99	-1.75	-1.75	-1.76	-3.66
	(2.69)	(3.94)	(3.79)	(3.37)	(3.74)	(3.62)
F-test (polynomials)					0.73	0.96
					[0.625]	[0.455]
Fixed effects	Cluster-month Country-Year	Cluster-month, Country-Year	Cluster-month, Country-Year	Cluster-month, Country-Year	Cluster-month, Country-Year	Cluster-mor Country-Ye
Polynomials	No	No	No	No	Yes	Yes
Cell linear trends	No	No	No	No	No	Yes
S.E. clustered at	ERA-40 cell	ERA-40 cell	ERA-40 cell	5-year by climate zone	ERA-40 cells	ERA-40 cel
# of S.E. clusters	743	439	304	35	304	304
# of obs.	962471	481018	481453	481453	481453	481453

Notes: Robust standard errors in parentheses, clustered as indicated. * significant at the 10 percent level, ** 5 percent, *** 1 percent. Fixed effects included as indicated. The row "Polynomials" indicates whether the cubic polynomials in the average monthly temperature and total precipitation over the 12-month period up to the birth month are included. The row "Cell linear trends" in Panel B indicates whether ERA-40 cell specific linear trends are controlled for. In column (4), "climate zone" refers to "steppe" and "desert" climates types, north and south of the equator, respectively. The null for F-test(polynomial) is that the coefficients on polynomial terms are all zero.

Table 7 – Infant Mortality and Nutrition: Heterogeneous Effects

Dependent Variable: Infant death indicator (multiplied by 1000)

	(1)	(2)	(3)	(4)
Sample	Rainy	Arid	Rainy	Arid
Household type	Agriculture	Agriculture	Educated	Educated
Rainfall (centimeters)	0.022	0.012	-0.041	-0.053
in growing season	(0.031)	(0.175)	(0.026)	(0.102)
Rainfall (centimeters) x	-0.121**	-0.194	0.188**	0.399
Household type	(0.049)	(0.245)	(0.073)	(0.395)
Drought (0,1) in	-0.982	33.761**	-2.057	26.901***
growing season	(16.454)	(13.377)	(13.270)	(9.345)
Drought(0,1) x	0.160	-23.628	-62.977	-62.115***
Household type	(17.195)	(22.469)	(88.381)	(19.008)
F-test (rainfall)	5.60	1.47	4.34	0.82
	[0.018]	[0.226]	[0.038]	[0.365]
F-test (drought)	0.00	0.31	0.60	4.81
	[0.954]	[0.581]	[0.440]	[0.029]
# of ERA-40 cells	439	304	439	304
# of obs.	457615	462856	478880	479755

Notes: Robust standard errors in parentheses, clustered at the ERA-40 cell level. * significant at the 10 percent level, ** 5 percent, *** 1 percent. Fixed effects for cluster-month and country-year interacted with household type indicators are included (see equation (5) in the text for the exact speficiation). The nulls for F-test (rainfall) and F-test (drought) are that the sum of rainfall and drought coefficients, respectively, is equal to zero.

Table 8 – Estimated Impacts of Climate Change

	(1)	(2)	(3)	(4)	(5)
Weather Period	1981-2000	2081-2100			
Emission scenario		Midrange		High	
Population		fixed	projected	fixed	projected
Panel A: Number of infant deaths by weather shocks					
by epidemics	84,299	16,380	41,474	101,272	202,173
by droughts	11,453	18,513	52,869	19,976	36,601
Panel B: Number of infant deaths due to malaria in those areas never endemic in 1981-2000 and in 2081-2100 under both scenarios					
	214,429	112,532	268,180	429,949	1,273,329

Notes: In columns (2) and (4), the number of births from 2081 to 2100 is assumed to be the same as from 1981 to 2000. In columns (3) and (5), the number of births from 2081 to 2100 is obtained by combining the population counts in 2000 from CIESIN and CIAT (2005) with the projected population growth and crude birth rates from United Nations Population Division (2011). Malaria zones and climate zones are derived from weather outcomes during 1957-2002 in column (1) and during 2051-2100 in columns (2)-(5).