Weather and Infant Mortality in Africa*

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Abstract

How have weather fluctuations affected infant mortality in Africa over the last half century? To answer, we combine individual level data, obtained from retrospective fertility surveys (DHS) for nearly a million births in 28 African countries, with data for weather outcomes, obtained from re-analysis with climate models (ERA-40). We find robust statistical evidence of quantitatively significant effects via malaria and malnutrition. Infants in areas with epidemic malaria that experience worse malarious conditions during the time in utero than the site-specific seasonal means face a higher risk of death, especially when malaria shocks hit low-exposure areas. Infants in arid areas who experience droughts when in utero face a higher risk of death, especially if born in the so-called hungry season. We also uncover heterogeneities in the infant mortality effects of growing season rainfall and drought shocks, depending on household occupation or education. Based on the estimates, the paper estimates the number of infant deaths due to extreme weather events and the total number of infant deaths due to maternal malaria in epidemic areas.

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1 Introduction

To think clearly about global policy responses to climate change, we need information 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 future climate change.

We deal only marginally with climate change. But we focus on carefully evaluating the health impacts of weather shocks in Africa over some 40 years in the past. Specifically, we focus on the effects on infant mortality. The reason is twofold. Alongside HIV, infant death is Africa's largest health problem: still today, close to 10% of babies born on the continent die before the age of one. But unlike the case of 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, imprecise estimates of e.g., the 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 around 17,500 cluster locations in 28 African countries. This way, 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 find out how the probability that these children survive their first year depends on local weather conditions, we use weather 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 (140x140km at the equator) earth grid.

To identify the causal effects of weather shocks, we use only the temporal deviation of weather from the normal monthly pattern within each given location. Constant geographic weather differences are correlated with numerous other factors, which also influence infant mortality. For example, coastal areas have different weather than land-locked ones, but as economic opportunities are better, people along the coast have higher incomes and lower infant mortality. There is little hope to convincingly control for all the relevant determinants. Similarly, long-run weather trends are highly spatially

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

correlated, and thus potentially confounded with long-run infant-mortality trends tied to the evolution of national health-care systems or income.

For these reasons, we rely on natural weather variations, which – arguably – are uncorrelated 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 the our results from our large-scale study with those small-scale studies that rely on randomized control trials to generate variation in other determinants of malaria or malnutrition, like bednet use or 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. Infants born in arid climate regions of Africa, who in utero experience droughts face a much higher risk of death than other babies, especially if 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.

The results are not only statistically robust, but also quantitatively important. For example, we estimate that a six-month malaria epidemic in a place with little average exposure to malaria raises infant mortality by more than 3.5 percentage points. The effect of a drought in an arid area is of similar magnitude and doubles for infants born in the so-called hungry season.

Because our estimates measure average effects across nationally representative samples, we can use them to estimate the average historical effects of these weather-induced phenomena. 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 twenty-year period, we estimate that 8,700 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 average number of all infant deaths that were caused by maternal malaria, in epidemic regions. Our calculation relies on the estimated fall in infant mortality in randomly non-malarious weather conditions. This is in the spirit of comparing infants from villages that were non-

malarious due to randomly assigned medical treatment with infants from non-treated villages. In terms of magnitude, we find that infant mortality drops by 0.8 percentage points in non-malarious conditions – about 7% of infant mortality. Since our estimates are based on representative samples, we can also estimate the total effect of malaria. We find that, out of 64 million births, 629,000 infant lives were lost in 1981-2000 due to malaria in epidemic regions. By comparison, Steketee et al. (2001) – who survey results from randomized epidemiological studies – find that infant mortality falls by 3-8% when mothers are malaria-free due to randomized medical intervention.

We quantify historical infant mortality risks due to weather-related malaria epidemics and droughts. How about forecasting the future development of these risks in the wake of climate change? In general, this is a hazardous exercise because of the genuine uncertainty about future changes in climate, and because of possible adaptation to a changing climate. While the first problem remains, some of our estimates can be used to overcome the latter problem. We hope to illustrate how this can be done for specific simulations of the future African climate.

While we are not aware of any studies with a similar scope and methodology for Africa, there exist a few recent reminiscent 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 work are given in context below.[2]

In the following, Section 2 of the paper gives general background on our data. Sections 3 and 4 go more into details about our methods of analysis and 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 pinpointing 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

[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.

6 summarizes our findings and discusses a few possible extensions.

2 Data and Background

The most important data for our study come from two sources. We exploit individual data on health and demography outcomes assembled from DHS surveys, and spatially disaggregated data on weather outcomes obtained from ERA-40 re-analysis. In this section, we give some background on these data and how we put them together – we also comment on the crucial seasonality of African weather patterns.

DHS surveys Demographic and Health Surveys (DHS) have been carried out in many developing countries since 1984 with financial support from US-AID. Each survey is carried out to collect information on life and health outcomes by interviews of a nationally representative sample of women in child-bearing age. Thanks to their standardized survey format, data from different surveys can easily be combined. DHS data have been used in a growing number of microeconomic papers on various topics in economic development.[3]

Each DHS survey employs a two-stage sampling, first selecting so-called clusters – i.e., villages and town districts – and then selecting households within each cluster. In this study, we use a total of 50 DHS surveys, from 28 African countries – all the surveys available in 2011 in which the geographic coordinate of each cluster is collected by a GPS receiver. These 50 surveys comprise information from a total of 17,568 clusters, located in both rural and urban settings. Figure 1 plots these clusters on a map of Africa. The data cover all parts of the continent.

In the retrospective fertility module of any DHS survey, 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. If either the month or the year of birth is not reported or inconsistent, the date of birth is imputed from auxiliary information. The surveys we exploit contain information about circa 1.2 million births by about 300,000 mothers that occurred at least 12 months before the survey date, in

[3]Detailed information on the DHS surveys and the underlying methodology can be obtained from the website:
`www.measuredhs.com`

the period (1957-2002) covered by our weather data. Dropping all the births with an imputed birth date leaves us with 962,471 births by 269,754 mothers.[4]

For each of these births, we construct a binary variable indicating whether the child died as an infant – i.e., at the age of 12 months or less.[5] This is our major dependent variable in the paper. Infant mortality varies quite a bit both across time and place. For the full sample of births, 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 of mortality about 144 in 1970 to about 86 in 2002. Inspection of the data shows that infant mortality also varies quite a bit from year to year in addition to generally declining trends, as well as across groups of clusters (e.g., rural and urban areas) within the same country.

The fact that the surveys are retrospective gives us some causes of concern. 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). However, our results do not change significantly when we drop all reported births more than 10 years before the survey. Assuming that events nearer in time are more easily recollected, this is encouraging, and suggests that measurement error owing to imperfect recall is not a major problem in practice.

Another cause of concern is that mothers might migrate, so the mother's location at the time of the survey may not coincide with her location when her children were born. Using weather information pertaining to the surveyed cluster may thus attribute incorrect weather conditions to the time around birth. The surveys allow us to drop all births taking place before migrating mothers moved to the survey location, and this robustness check does not materially affect the results. Thus, the prospective downward bias of using weather data from the wrong place appears to be small. In Sections 3 and 4, we discuss other possible sources of selection bias in context.

[4]It is conceivable that the mortality of the babies with a missing birth data is lower if the death of babies make their mothers more likely to remember their birth date. To assess this possible source of bias, we compare the mortality rates of the babies with and without an imputed birth date. We find that babies with their birth date imputed are *more* likely to die by about 96 deaths per 1000. If this result suggests that those babies dropped from our sample are more vulnerable to weather shocks, then our estimates will be a lower bound of the impact.

[5]The results are robust to excluding death at the age of 12 months from the definition of infant death.

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

ERA-40 re-analysis Development economics research has increasingly relied on shocks to weather, such as rainfall, as a way of isolating exogenous variation in variables like income. The bulk of this research relies on data from weather stations together with various interpolations to fill out the missing data.

A well-known data set based on weather station observations is supplied by the Climate Research Unit (CRU) at the University of East Anglia.[6] The CRU data set indeed includes data at a high temporal and spatial resolution (monthly data at down to 0.5×0.5 degree resolution) for much of Africa. But its interpolation method is problematic for exploiting variation within location over time.[7] Since weather stations with consistent 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 is not suitable for our purpose.

In their study of African civil wars, Miguel et al. (2004) use rainfall data from the Global Precipitation Climatology Project (GPCP), which relies on satellite images of cloud cover to estimate rainfall. However, for our study the GPCP is unsatisfactory: the spatial resolution of the rainfall data is rather coarse at 2.5×2.5 degrees, and we need consistent temperature data to predict malaria transmission risk (see the next section).[8]

Instead, we rely on weather data produced by what meteorologists call re-analysis.[9] Specifically, we use a data archive known as ERA-40 supplied by

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

[7] First, changes in the weather outcomes in a given location may be due to the availability of nearby weather station data over time. Second, if the closest weather station with available data is too far, a long-term average value is used. See Climate Research Unit (undated) for details.

[8] The higher resolution data of the GPCP (1.0×1.0 degree) is available only after October 1996.

[9] For an account of reanalysis to economists, see Auffhammer et al. (2011).

the European Centre for Medium-Term Weather Forecasting (ECMWF).[10] The archive provides weather outcomes for every six hours, over the period from September 1957 through August 2002, on a global grid of quadrilateral cells defined by parallels and meridians at a resolution of 1.25×1.25 degrees (about 139×139 kilometers around the equator).[11]

The ERA-40 re-analysis begins with historical data from a variety of sources: weather stations, ships, aircraft, weather balloons, radiosondes, and most importantly – from the 1970s – satellites orbiting the Earth.[12] Such observations at each point in time are fed into the ECMWF's large-scale atmospheric circulation model (known as IFS CY23r4) to predict the state of the atmosphere six hours earlier. These predictions are then corrected by using observations wherever available (the process known as "data assimilation") to produce the most consistent representation of the atmospheric state such as humidity and temperature. . Finally, given this estimated time series of the atmospheric state, the climate model forecasts precipitation (and other weather outcomes) in every six-hour interval. The resulting data are used in our analysis.

The re-analysis data – like other prospective data – is, of course, also subject to measurement error. The quality of the generated rainfall data is of particular concern, given the well-known difficulty of accurately predicting the precise location of convective rainfall and thunderstorms. It is noteworthy that precipitation data in the ERA-40 do *not* depend on rainfall gauge data, which is particularly coarse and likely of low quality in Africa. Instead, they rely entirely on the climate model and the estimated state of the atmosphere including humidity, temperature and winds. While the resulting forecasts may often fail to accurately predict the amount of rainfall in a precise location for the next six hours, the aggregation in time (to a month) and space (to 1.25×1.25 degrees) resolves much of this problem. A comparison of the ERA-40 rainfall data to rainfall gauge data also suggests that the bias in the ERA-40 rainfall data is less important for the arid and semi-arid areas of Africa, where the annual departures from the regular seasonal fluctuations

[10]The data were downloaded from ECMWF's Meteorological Archival and Retrieval System. We are grateful for Heiner Körnich for help in this process.

[11]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.

[12]The satellite data mainly provide temperature and humidity. These variables are highly spatially correlated, and thus the spatial resolution of satellite data, which is coarse for the earlier periods, does not hugely affect the data quality of re-analysis.

in rainfall are the largest.[13]

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

Matching the data sets Each DHS cluster is matched to the ERA-40 grid cell that contains it, by using ArcGIS 9.3's Spatial Join tool. These matched ERA-40 cells and DHS clusters are plotted in Figure 2. With 17,568 clusters and 743 grid cells, there are almost 24 clusters on average per grid cell. For each grid cell, we extract 6-hourly data on rainfall and temperatures from the ERA-40 archive, and aggregate these to the monthly frequency. Effectively, this gives us a large, balanced panel data set of rainfall and temperature, with 743 cross-sectional units and 540 (12×45) monthly observations for each unit. Summary statistics for infant mortality and weather outcomes, by clusters and various subgroups (to be defined below), are reported in Table 1.

Seasonal fluctuations in African weather The monthly frequency of observations in our data plays a crucial role in the analysis below because the most salient aspect of weather on the African continent is its strong seasonality. The seasonal differences in temperature interact in an important way with strong seasonal fluctuations in precipitation. Thus, while predictably rich throughout the year in tropical Africa, rainfall in many arid and semi-arid areas is much more erratic. The reason is that continent-wide rainfall patterns are largely governed by the so-called Inter Tropical Convergence Zone (ITCZ), in which the trade winds from the northeast and the southeast converge (Griffiths 1972). As a result of the low pressures along the ITCZ, convectional thunderstorms form daily and dump large amounts of precipitation in scattered afternoon rains. Over land, the ITCZ moves north and south with the seasons, following the hottest part of the continent, which

[13] Zhang, Körnich and Holmgren (Forthcoming) provide a recent discussion of the quality of re-analysis data – from different sources including ERA-40 – for Africa to the south of the equator.

causes large variations in rainfall between dry and wet periods in a typical year. This is illustrated in Figure 3, which shows the average amount of rainfall across Africa in four different months.

On top of this regular seasonal cycle, however, one finds considerable fluctuations across years in the precise timing and amount of temperature and rainfall. These are more marked in the arid and semi-arid areas that receive little rain, owing to the unpredictable movements of the ITCZ from one year to the next. Part of these movements are due to the chaotic weather dynamics over horizons beyond a couple of weeks. But another determinant is the poorly understood, and thus hard-to-predict, medium-term fluctuation in air pressure associated with the Southern Oscillation.[14] The warming phase (El Nino) is generally associated with wetter weather than normal in East Africa during March-May, but less rainfall than normal from December to February in parts of South and Central Africa, with the opposite patterns during the cooling phase (La Nina).

The fluctuations around the regular seasonal cycle play a crucial role in our analysis. In particular, the malaria season, as well as the growing season for 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 the local rate of infant mortality.

3 Malaria

In this section, we focus on malaria during pregnancy as the channel through which weather affects the survival of infants.[15] We start by a brief and selective overview of the epidemiology, immunology and pathology literatures on malaria and infant death. This suggests that large risks are associated with malaria infected mothers when the child is in utero, and that these risks may differ by malaria prevalence and type of baby. Next, we describe the index that we use to measure the weather conditions suitable for malaria transmission and infection, and how this index can be used to classify our different DHS clusters into different zones of malaria risk. Then, we present

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

[15] 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.

and discuss our econometric methodology and some results for the full sample and different malaria zones within a simple model where the number of malaria months enter linearly. These basic results clearly indicate that site-specific shocks to malarious conditions only have a significant effect on infant death in African areas with epidemic malaria, a result which is robust to a variety of statistical pitfalls. Thus, we look closer at subsets of these epidemic areas, allowing for a non-linear effect in the number of malaria months and in the time of the year when the child is born. Finally, we ask whether the risk of infant mortality varies systematically with household and mother characteristics.

Malaria and infant mortality Malaria is one of the major causes of death for children in Africa. Estimates provided by Black et al. (2010) suggest that malaria caused about 16 % of deaths of children under the age of five in Africa. It is estimated that about 75 % of the estimated malaria death toll of nearly one million people in sub-Saharan Africa in 1995 is made up of children less than five years old (Snow et al. 1999). However, infants are known to have a reduced sensitivity to malaria during the first few months of life, and fatal infections are believed to be more likely in the latter half of the first year of life and the first few years of childhood (Maegraith 1984, p. 262).

Malaria in pregnancy[16] is known to raise the likelihood of infant death via 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 birth weight induced by maternal malaria.[17] The exact mechanism for the association between malaria in pregnancy and low birthweight 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. For areas where malaria is endemic, 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.

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

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

(2007) review studies conducted in low malaria transmission areas of Africa and report a median prevalence of placental infection amounting to 6.7 %.

On top of a higher likelihood of low birth weight, 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) and may become susceptible to measles earlier than other babies due to reduced placental transfer of maternal antibody (Owens et al., 2006).[18]

Given the above-mentioned 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.[19] 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 during the first year of life on mortality.

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 malaria on infant mortality to be larger in areas where malaria transmission is low.

Firstborn babies are believed to face a higher risk of death due to malaria in pregnancy than those of higher birth order, although this heterogeneity appears to be absent in low malaria transmission areas (McGregor 1984). Rogerson et al. (2000) and Walker-Abbey et al. (2005) find that teenage women are more likely to be infected with malaria during pregnancy in Malawi and in Cameroon, respectively. Infants born to mothers infected with HIV as well as malaria face higher risks of low birth weight (ter Kuile et al. 2004). In general, firstborn babies, female babies, and babies born by

[18] 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).

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

stunted mothers, face a particular risk of low birthweight (Kramer 1987), which makes it plausible that such babies might be particularly at risk in the wake of malaria shocks. We investigate whether these individual-level characteristics result in heterogeneous impacts of malaria-prone weather conditions.

How to measure 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. Based on clinical measurements of malaria prevalence, researchers have tried to combine such information on 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 for which we have infant mortality data.

A necessary condition for malaria to spread is the growth and survival of parasites causing the disease and vectors (a certain species of mosquitoes) transmitting the parasites. The rates of growth and survival are known to be heavily dependent on temperature and rainfall, and we want to capture these conditions in a parametric way.

To this end, we follow Tanser, Sharpe, and le Seuer (2003), who propose a relatively parsimonious weather-based index for malarious conditions for Africa in their study of malaria and climate change. This index builds on the comparison of mean long-term (1920-80) monthly rainfall and temperature with monthly profiles of malaria transmission intensity in 15 different locations with differing malaria prevalence rates as well as biological ranges affecting both vector and parasite development. The resulting monthly predictions of malaria transmission are empirically validated against the malaria occurrence data from about 3800 parasite surveys in different African locations. The index correctly predicts 63 % of malaria transmission incidents and 96 % of the absence of malaria transmission.[20]

Following the approach of Tanser et al. (2003), we adopt the following:

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

[20] 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.

(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* $\tau)$ *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 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.[21] 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.[22]

Malaria zones in Africa Climatological conditions play a major role for prevalence of malaria. In some areas, malaria is *endemic*, meaning that a high risk of malaria permanently, or at least a good part of every year. In other *epidemic* areas, malaria spells are more short lived. This can be either because the transmission is seasonal, i.e., it recurs in a particular season due to stable variations in rainfall and temperature, or because it is unstable,

[21] The vector obtains a parasite by biting a malaria-infected human being. However, it takes a while for the parasite to become infectious and thus for the vector to transmit malaria by biting another human being. Higher temperature shortens the time required for the parasite to become infectious and helps the vector survive long enough.

[22] 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, Tanser et al (2003) treats it as malarious if it is sandwiched by two malarious months. A priori such a sandwich condition may make sense in a cross-sectional context as theirs, but makes less sense in a time-series context as ours. When we implement the index in exactly the same way as Tanser et al (2003), the results are weaker presumably due to less variation over time. By dropping separately each of the four conditions, we found conditions (a) and (d) to be the most relevant ones to predict infant death.

i.e., it is present in some years but not in others. Given the malaria index discussed above and the seasonality of African weather discussed in Section 2, the distinction will largely reflect the seasonality of rainfall with epidemic areas falling into the continent's arid and semiarid areas. 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 rates and more severe malaria infections in such regions. 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, as defined by the malaria index $Z_{g,\tau}$, over the entire sample period; epidemic areas have strictly positive malaria exposure between 0 and 4 months on average; while endemic areas have higher exposure rates. (We have also set the epidemic-endemic split at 6 months with similar results.)

Our classification is illustrated in Figure 4. Non-malarious areas, the green circles on the map, entail about 20% of the births in our sample and are found in the very North and South of Africa, and in 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 between endemic and epidemic areas. The epidemic areas, colored in yellow, are mainly found in the Sahel, in higher terrain in East Africa, and in dry areas of the South. Endemic areas, in red, are mainly found in the tropical parts of Africa with stable warm and humid conditions throughout the year.

The geographical distribution of these three zones, based on weather conditions alone, corresponds 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).

Malaria exposure for individual pregnancies Focusing on the malaria conditions during pregnancy, we create a measure of maternal malaria exposure of the 12 months up to the birth month for each birth in our sample. 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 gauge during how many months in the year before birth the child's mother was exposed to malarious weather conditions. This measure varies substantially across areas and time. In endemic areas, mothers 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 $z_{g,t}$).

Basic econometric specifications In our most basic econometric specifications, we estimate 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 a binary infant mortality indicator. It indicates death at the age of 12 months or less, for child i, who is born in cluster c, within grid cell g in country x, and in running month t, which is calendar month s of year y.[23] 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 is β, which measures how many more children per 1,000 die in the first year of life due to one additional month suitable for malaria transmission in the 12 months before birth. Further, $\alpha_{c,s}$ is a *fixed effect* for cluster c and calendar month $s = 1, .., 12$. That is, when we run this regression 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 the parameter β from the deviation within each cluster from its site-specific monthly mean. To allow for a non-parametrically declining trend of infant mortality throughout Africa, 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 infant mortality to have (non-parametric) trends in national health systems, policies, or economic conditions, which could conceivably be related to local weather realizations.[24] Finally, $\varepsilon_{i,c,x,t}$ is

[23] 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.

[24] For example, Kudamatsu (forthcoming) finds democratization has reduced infant mor-

an error term. We compute Huber-White robust standard errors, allowing for clustering at the grid level (encompassing 743 grid cells in the full sample).

Basic results The results we obtain when running versions of (2) in the full sample 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, thus allowing for different cluster means and a non-parametric time trend for all of Africa. Column (2) replaces the cluster fixed effects with cluster-by-month fixed effects, whereas Column (3) estimates equation (2) by replacing year fixed effects with country-by-year fixed effects so that non-parametric trends for each country are allowed.

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 from neighboring areas in the same country.

However, this basic specification assumes a treatment effect of malaria shocks that is homogenous across all of Africa – a very strong assumption. To test our prior of a larger effect in epidemic areas, we split the sample into its endemic and epidemic part, dropping non-malarious areas which, by definition, have no variation in the malaria index. The corresponding estimates for endemic areas are shown in columns (4)-(6) of panel A. The point estimates for temporary malaria shocks show a similar pattern as those in the full sample, but they are never statistically significant.[25] 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. Year-to-year variation in seasonal malaria transmission for endemic areas is not very large (see Table 1 Panel B), and the bulk of malaria-induced infant deaths are likely absorbed by the

tality in sub-Saharan Africa while Bruckner and Ciccone (forthcoming) find negative rainfall shocks led to democratization in Africa.

[25]The result is similar if the boundary between epidemic and endemic is instead drawn at 6 months average exposure.

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 who face three months higher malaria exposure than normal have a raised infant mortality risk in the average epidemic cluster by just below 3 per thousand (close to the total infant mortality rate for Sweden).

In the three remaining specifications in Panel B, we show the results of some robustness analysis for epidemic areas. Clustering of the 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. While such local serial correlation certainly exists, weather and the survival of babies are likely to be correlated across grid cells and also between a particular cell in a certain month and its neighboring cells in the following months. To allow for simultaneous spatial and temporal correlations, we use an alternative clustering scheme by 5 year-period and average malaria exposure (specifically, we split epidemic areas in those above and below 2 months of exposure north and south of the equator, respectively), giving a total of 36 clusters. As Column (4) shows, this yields standard errors for β, which are slightly lower than those with grid-level clustering.

While the specification looks at the linear effect of different number of malaria months, the definition of a malaria month is highly non-linear in temperature and precipitation. Perhaps this specification really picks up some other non-linear effect of weather on infant mortality. To check for this possibility, in Column (5) we include cubic polynomials in rainfall and in temperature during the 12 months preceding each specific birth.[26] This is like adding a control function to the specification in equation (2). 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 mor-

[26]Specifically, we include the following terms to the right hand side of equation (2):

$$\rho_3^T T_{g,t}^3 + \rho_2^T T_{g,t}^2 + \rho_1^T T_{g,t} + \rho_3^P P_{g,t}^3 + \rho_2^P P_{g,t}^2 + \rho_1^P P_{g,t}$$

where $T_{g,t}$ and $P_{g,t}$ are the average temperature and the total rainfall, respectively, in grid cell g over the months $t-11$ to t. In subsequent analysis, we always refer to these terms as cubic polynomials in rainfall and in temperature.

tality at the level of each country, it is conceivable that some confounding variation is more local. Thus, column (6) adds (linear) trends also 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 in epidemic areas? All specifications in Table 2 assume that the impact of malaria shocks is linear in months of malaria exposure. But infant mortality is an extreme outcome, so 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 exposure per year. This further classification is illustrated in Figure 5. Based on an immunity argument, one could presume that weather shocks increasing the susceptibility to malaria may have their 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 (2) is estimated on the two separate epidemic subsamples. As the estimates show, however, the linear model does not produce very different estimates in the two samples.

To get further, we allow for a more general non-linear response within each subsample, by allowing for five bins of malaria exposure, setting the omitted default bin at average exposure. In Columns (3)-(5), we look at the 0-2 month exposure sample, the cream-colored regions in Figure 5. The distribution of malaria exposure over time in those places is highly skewed to the left. Over 60 percent of the observations have no malaria exposure at all. But about 1 percent of the 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 the 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, exposing a set of mothers to a potential more-than-six-months epidemic raises infant mortality by about 38 per 1000, compared to a control group of pregnancies with no or little exposure at all. This is a huge effect, given an average infant mortality rate of about 100 per 1000 in the sample, which is consistent with mothers in these epidemic areas having little or not immunity at all. Columns (4) and (5) show that the results is basically robust

to including cubic polynomials in rainfall and in temperature during each pregnancy and linear ERA-40 cell-specific linear trends. As shown by the F-test, the polynomial terms are not statistically significant.

In Columns (6)-(8), we show analogous estimates for DHS clusters with 2-4 months average exposure, the orange regions in Figure 5. In this subsample, nearly 10 percent of the pregnancies have no exposure to malaria while 1 percent of them 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. Since the polynomial terms are significant in this case, the point estimates in columns (7) and (8) are the most appropriate. We see that eliminating malaria in a given year, saves about 10 infants in 1000, i.e., it brings down infant mortality by one percentage point. A rise in malaria exposure from the average to above 6 months, on the other hand, increases infant mortality by about 20 in a 1000, i.e., 2 percentage point.[27]

Figure A2 in the Appendix shows which areas in the sample contribute to the estimates of these nonlinear effects. It shows all the ERA-40 grid cells belonging to the epidemic area, which had at least one incidence of malaria epidemic (5 or more malaria months in the 0-2 month area; 7 or more in the 2-4 month area) for months in which we observe births in the sample. The figure shows that these events did occur 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 the results here to estimate historical death tolls due to malaria, and discuss the implications of climate change.

Heterogeneity by individual characteristics Following the medical literature reviewed at the beginning of the section, we have also investigated if the impact of maternal malaria exposure is heterogeneous across different types of babies, mothers, or households. In particular, we have estimated extensions of our basic econometric specification in equation (2), where all

[27] 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.

right-hand side variables are interacted with indicators for female babies, firstborn babies, young mothers (under 18), stunted mothers (2 standard deviations below the median stature of the WHO Child Growth Standard by WHO Multicenter Growth Reference Study Group, 2006), and households living in regions with high HIV prevalence rate (10 % or higher according to the DHS HIV test results conducted in the 2000s). We have also investigated the heterogeneous impact by the education level of the household (whether both the baby's mother and her husband went to school for more than 8 years) and by affluence of the household (owning a majority of the consumer durables listed in the survey questionnaire). In these regressions, we always split the sample between endemic and epidemic areas. However, we find no significant patterns of heterogeneity in the data, while we always continue to find a significant effect of malaria shocks in epidemic areas but no such effects in endemic areas. This lack of heterogeneity across mothers and households is a bit surprising given the clinical evidence cited above.[28]

Malaria shocks in the first year of life For each child, we have focused on malaria shocks during the year before birth and we have seen that these shocks in utero have a significant effect on the likelihood of survival. Do malaria shocks after birth affect the probability that a child dies before age one, directly or indirectly through the health of the mother? To analyze this question, we have run regressions where malaria exposure during the first year of life – either month by month or the cumulated number of months with a positive malaria index – is added as its own term and as its interaction term with $z_{g,t}$ to the right hand side of equation (2).[29] We find no significant effects on infant mortality of in-life shocks neither in epidemic areas, nor in endemic areas. On the other hand, in-utero malaria exposure continues to exercise a significant effect on infant death in epidemic areas of similar magnitude as

[28] Our failure to find heterogeneous impacts of malaria in pregnancy across parities in endemic areas, however, is consistent with Guyatt and Snow (2001), who report malaria in pregnancy doubles the risk of low birthweight across all parities as well as for first pregnancies. Mutabingwa et al. (2005) also find that infants born to women with malaria-infected placenta are susceptible to malaria infection even if they are of higher birth order.

[29] Malaria exposure in the n-th month after birth does not affect the survival of babies who died before turning n months. Therefore, including the 12-month exposure to malaria infection during the first year of life as a regressor to the whole sample will bias the estimated effects towards zero. To deal with this problem, for each n from 1 to 12, we restrict the sample to babies who survive at least the first n months after birth and use how many months are malarious during the n months after birth as a regressor.

in our earlier estimates.

Summary To summarize, we find that weather shocks which raise exposure to malaria, as measured by the Tanser et al (2003) malaria index, significantly raise the incidence of infant death. The largest increases arise from exposure for more than six months in areas where malaria is otherwise rare. The largest decreases are found when weather in a particular year does not permit malaria at all in areas where malaria is around from time to time.

4 Malnutrition

In this section, we continue to explore how past weather events impact on infant death. But here, we focus on the prospective mechanism through malnutrition. Following a brief literature review, we discuss how to measure weather-induced crop-yield fluctuations in agricultural societies highly dependent on rainfall during a limited growing season, and how to partition the African continent into different climate zones. Next, we describe our measure of weather conditions conducive to more or less malnutrition during a child's period in utero and validate it against the data on crop prices in a subset of the countries in the infant mortality sample. Our econometric estimates show that a simple measure of rainfall during the growing season(s) tied to each childbirth are significantly related to infant mortality. In addition, we find a large effect of extreme events in the form of droughts (but not of floods), in Africa's arid climate zone. When we allow for heterogenous effects for different types of households, we uncover a significant linear effect of rainfall among agricultural households in tropical and temperate climate zones; we also find drought effects in the arid areas to be weaker in households dependent on agriculture and in well-educated households. Finally, the data suggest that babies born in the hungry season – the time just after the start of the rains – are particularly sensitive to malnutrition in utero.

Infant mortality and malnutrition Maternal and child malnutrition poses a major risk for child health, particularly in poor countries – for a recent review see Black et al. (2008). Because maternal intake is the sole source for fetal energy requirements, a lack of food during pregnancy diminish 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). The medical literature finds that low weight gain during pregnancy increases the risk of low birth weight (Kramer 1987 for a review). This effect is found to be stronger for women whose nutritional status is already poor before pregnancy (review by Krasovec and Anderson 1991) and during the second and third trimesters (Strauss and Dietz 1999).

Most African children are breast-fed during the period after birth, which is known to lower mortality risk compared to children 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)[30] Consequently – and in analogy to the previous section on malaria – 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.

Crop yield and growing seasons Most African countries are agricultural economies – in 2004, some 55% of people on the continent are employed in agriculture (Frenken 2005, Table 2), and many more depend on agriculture in other ways. In addition, transportation infrastructure in Africa is poorly developed.[31] Most people are thus largely dependent on the local yields of subsistence crops for nutritional intake (or of cash crops for earning income to buy foods). Moreover, irrigation of land plays a minor role in crop production, especially in Sub-Saharan Africa – only 6.4% of cultivated land was irrigated in 2004 (Frenken 2005, Table 12). These stylized facts about Africa suggest that maternal nutritional intake largely depends on local rainfall.

Thus, given the facts in Section 2 about the seasonality of African rainfall, we expect the largest effects on malnutrition to come from shocks that bring the amount of rainfall away from its seasonal average in the arid and semi-arid parts of the continent. In particular, 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. We therefore use the total amount of rainfall during the growing season as a proxy for the amount

[30] 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).

[31] 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.

of nutrition available for pregnant women in the analysis below. We have also experimented with various measures of temperature during the growing season, but with little success.[32]

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 in 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.

How to measure the growing season? The growing season in a particular location depends on many factors other than the extent of rainfall, including soil qualities, crop types and the use of fertilizers. While some gridded information on these other factors exists, we take a convenient short cut to determine the relevant growing season for each of our DHS clusters, through measures of photosynthetic activity by remote sensing.

Photosynthesis 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. Therefore, ecologists often use data collected by satellites to measure plant growth through ongoing photosynthesis. 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. In the ecology and biology literature, the integral of NDVI values over the growing season is often used as a proxy for crop yields (e.g. Rasmussen, 1992 for millet yields in Burkina Faso, Rasmussen, 1997 and Rasmussen, 1998 for millet yields in Senegal).

[32] We are certainly not the first to use growing season rainfall as a proxy for crop yields. 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.

The map in Figure 6 shows the distribution of the average annual integrated NDVI across Africa, with bluer areas denoting areas with a low value – little photosynthetic activity over the year – and redder areas a high value. The two graphs in Figure 6 plot observed NDVI values as the jagged thin curves over two years, 1982 and 1983, in two locations: one in Burkina Faso just at the boundary to Niger, and one in Tanzania just south of the Victoria Lake. In these graphs, the horizontal axis shows time measured in two-week periods; the vertical axis shows the NDVI value (multiplied by 10,000). Clearly, the peaks are much lower (note the different scales) for the Burkina Faso location than the Tanzania location, reflecting a lower amount of rainfall.

To obtain the growing season from this time-series NDVI data, we use the TIMESAT program (Jonsson and Eklundh, 2004).[33] The two graphs in Figure 6 demonstrate how this program works. The TIMESAT program first produces smoothed (filtered) values of NDVI (shown as the thick curve in the graphs), where the smoothing is meant to eliminate temporary random fluctuations, for example, due to variations in cloud cover. Following the common practice among ecologists (e.g. Heumann et al. 2007), the program then produces the times for the start and the end of the growing season defined as the time period in between 20% above one trough to 20 % above the next, as shown by the points on the smooth curves in the figure. Notice that the duration of the growing season is much shorter in Burkina Faso than in Tanzania. Finally, 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.[34]

Climate zones Because the seasonality of weather and agriculture differs so much, crop types, cultivating practices, and lifestyles have most likely adapted to the local conditions in different parts of Africa. We would therefore like to allow the effects of weather on nutrition and health to depend on the prevailing climate. A straightforward way of making such conditioning operational is to follow the approach originating with German climatologist

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

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

Wladimir Köppen, who was the first to classify different areas on Earth into different climate zones.

The well-known Köppen climate classification system distinguishes between different climate types based on annual and monthly temperature and precipitation, as well as the seasonality of precipitation (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, which include rainforest, monsoon, savannah and temperate climates, and *arid* areas, which include steppe and desert climates. The resulting classification of our DHS clusters is shown in Figure 7. Notice that the arid climate zones largely overlap with the epidemic malaria zones, as shown in Figure 4.

Malnutrition exposure for individual pregnancies We want to determine how weather affects each mother's nutritional intake for the 12 months before her child is born. When doing so, we focus on effects through local crop yields driven by variations in rainfall during the relevant growing seasons, as summarized by a simple index.

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, one month after the last harvest in this location (August 2000). In the last year before giving birth, the mother has consumed food for one month from that harvest and for eleven 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 had the ability to consume from each harvest. In the example, our rainfall exposure index weights rainfall during the growing seasons of 2000 and 1999 by the weights 1/12 and 11/12, respectively.

To be more precise, we define a simple *rainfall exposure index*, proxying for the nutritional dependence 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) 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 proxy the nutritional dependence on weather during the 12-month period up*

to the birth date t in location g by the rainfall exposure index, defined 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, the index assumes that all crop yield in location g becomes available at the final month of the growing season, and this harvest month, $h^{g,t}$, is the same calendar month every year. Second, it assumes that 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, it assumes that the marginal effect of weather variation on nutritional intake is constant across the year of exposure.

Below we will 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. As discussed above, we will also relax the third assumption and investigate whether the marginal impact of harvested crop yields on children's health is larger during the hungry season.

The mean rainfall exposure index in the sample is 70.0 centimeters (cm) of rainfall, while the average grid-level standard deviation is 19.2 cm. The corresponding statistics for the rainy climate zone sample are 122.7 and 28.5 while they are 17.3 and 5.9 for the arid climate zone. As mentioned above, 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 for summary statistics.)

Validation of the rainfall exposure index How can we make sure that this index based on growing season rainfall that we have just defined is a relevant measure of the scarcity of local crop yield? One way is to relate the measure of growing season rainfall to observed crop prices. In doing so, 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 compiled from the data in the USAID Famine Early Warning Systems Network (FEWS NET).

We then relate these local crop prices to local rainfall and drought incidence (to be defined below), exploiting only the within-market monthly

deviations from the local seasonal mean, relying on an empirical strategy that is fully analogous to our infant-mortality analysis. See the 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 percent with a one standard deviation increase in rainfall during the previous completed growing season, i.e., $r_1^{g,t}$ in equation (3). A drought incident significantly increases the crop price by 6.7 percent. 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 give support to our assumption that local rainfall affects the availability of food due to the lack of irrigation and transportation infrastructure.

Basic results Table 5 reports the estimates from running panel regressions like (2) 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, following from the first two assumptions underlying Definition 2, 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 subsamples of babies born in rainy and arid climate zones, respectively. In both areas, the point estimates have the same negative sign as in the full sample, but are too noisy to be statistically significant.

Non-linear effects: droughts and floods 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. The linear specifications estimated in Table 5 do not allow for disproportional effects of extreme events, however.

We use a drought index based on extreme growing season rainfall outcomes, defined 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 by*

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

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.[35]

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) – in 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 arid-area sample, 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.

[35] 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.

Column (4) shows that these estimates for arid areas are robust to clustering the standard errors at climate zones by 5-year periods.[36] 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).

Figure A3 in the Appendix shows all the ERA-40 cells which experience at least one incidence of drought for months in which we observe births in the sample, analogous to Figure A2 mentioned in Section 3. 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 of our sample.

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 climate change.

Heterogeneity by household characteristics? The specifications in Tables 5 and 6 do not allow for any heterogeneous effects across households, mothers and babies (beyond a difference across climate zones). We now turn to these issues. It is natural to believe that the vulnerability of the offspring to maternal malnutrition might differ with mother or household characteristics, such as occupation, income, or education. Table 7 presents some estimates relevant to this hypothesis. 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,

[36] 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.

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 baby's household as *well-educated* if both the baby's 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.

We then run the following regression:

$$\begin{aligned} m_{i,c,x,t} = \beta r_{g,t} + \beta^f r_{g,t} \cdot f_i + \gamma d_{g,t} + \gamma^f d_{g,t} \cdot f_i \\ + \alpha_{c,s} + \alpha^f_{c,s} \cdot f_i + \alpha_{x,y} + \alpha^f_{x,y} \cdot f_i + \varepsilon_{i,c,x,t} \ , \end{aligned} \tag{5}$$

where f_i is an indicator of baby i's household type (agricultural or well-educated). Note that this specification allows cluster-by-month and country-by-year fixed effects to differ across different household types. Table 7 reports the estimated coefficients: β, β^f, γ, and γ^f.

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 if we look at agricultural households. The sum of the two coefficients on rainfall ($\beta + \beta^f$) – the total linear effect of rainfall for agricultural households – is statistically significant at the 5 percent level. The non-interacted coefficient (β) 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 γ and γ^f is not significantly different from zero).

A reasonable interpretation of these results is that in rainy areas a normal downward variation in rainfall tends to make agricultural households worse off nutritionally. On the other hand, in arid areas such households have better access to whatever little crop yield there may be 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 – as might be expected – the well-educated appear to be protected from the high infant mortality effects of a drought shock in arid areas, perhaps as a result of higher purchasing power or better opportunities.

As in the malaria section, we have also experimented with conditioning on various baby and mother characteristics (gender, birth order of child, age or stature of mother, etc.), on the notion that some types of babies or mothers may be more vulnerable to malnutrition shocks than others. But this has produced no robust results.

Heterogeneity by timing of birth As mentioned after Definition 2, our simple rainfall and drought exposure indexes implicitly assume that the marginal effects on infant mortality are constant across time. We now relax this assumption by conditioning the effects of shocks on the time of birth relative to the beginning of the growing season. Given the earlier results, we focus on the drought effects in arid areas and run the regression:

$$m_{i,c,x,t} = \sum_{k=0}^{3} \beta^k r_{g,t} \cdot q_{g,s}^k + \sum_{k=0}^{3} \gamma^k d_{g,t} \cdot q_{g,s}^k + \alpha_{c,s} + \alpha_{x,y} + \varepsilon_{i,c,x,t} \ , \qquad (6)$$

where $q_{g,s}^k$ is a dummy that equals one if calendar month s of date t falls within the k^{th} quarter since the beginning of the growing season in grid g ($k = 0$ for the quarter immediately before the growing season starts).

Figure 8 plots estimated coefficients of the γ^ks and their 95% confidence intervals. The vertical line in the figure indicates the beginning of the growing season – recall Figure 6 and our definition of this as the time when the NDVI value is 20% above its last trough. The babies born in the quarters around the beginning of the growing season, marked 0 and 1 in Figure 8, seem to 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 these babies – on the order of 60 per 1000 births – is a stunning number indeed.

These results are related to the notion of a "hungry season", in the literature on food availability and poverty. The average length of the growing season in arid areas is about 6 months in our sample, and the actual harvest may start before the end of the fixed growing season we use in the analysis.

Therefore, food is the least available in the period around (in particular, after) the beginning of the growing season. On top of that, the beginning of the growing season 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.

A study on pregnant women in a Gambian village shows that pregnancy, even in the last month before giving birth, does not reduce the time women spend at their farms (Roberts et al. 1982). Studying the same rural area in Gambia, Rayco-Solon et al. (2005) find that the incidence of premature birth (a major cause of low birth weight) significantly increases during the first few months of the rainy season, which suggests a possible causation from increased amount of workload for pregnant women to low birth weight.

Moreover, we can compare the estimated effects of random variations in rainfall with the results from randomized controlled trials which vary nutritional intakes in other ways. Thus, in one study, a treatment group of randomly selected pregnant women in Gambia were given dietary supplements (Ceesay et al., 1997). According to that study, the effect of these supplements on the incidence of low birth weight 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.

Malaria and malnutrition 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 the shock measures emanate from the same weather data. It is thus legitimate to ask if the results hold up when we allow both types of shocks to occur simultaneously. 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.

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.

Summary Let us briefly summarize the message of this section. Extreme negative rainfall shocks (droughts) have a powerful effect on infant death in areas with steppe and desert climates. Rainfall above the site-specific seasonal mean in the relevant growing season diminishes infant mortality only for babies born in agricultural households in the rainy parts of Africa. Drought shocks impinge especially hard on babies to parents that do not work in agriculture, are not well educated, and on babies born around the start of the rains.

5 Aggregate Effects and Methodology Issues

What can our results say about aggregate effects of weather on infant mortality? In this section, we first estimate how many infant deaths during (part of) our sample period were caused by extreme malaria episodes in epidemic areas and by drought episodes in arid areas. Second, we estimate how many infants deaths in all of Africa are caused by malaria in epidemic regions and by droughts. We compare the resulting figures to existing estimates and discuss methodological differences. Finally, we try to pinpoint areas at risk for high infant mortality at the end of this century, given a distribution of future weather implied by simulations of general circulation models with a changing climate due to continued emissions of carbon dioxide.

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 expected number of births for each year and ERA-40 cell in the epidemic regions of Africa, during the subperiod for we which we have reliable gridded population data, namely 1981-2000.[37] We then multiply the estimated number of births

[37]We start with the Gridded Population of the World version 3 (CIESIN and CIAT 2005) to obtain the population count in each 2.5 by 2.5 arc minute cells in 1990, 1995 and 2000. Next, we aggregate this number to the intersections of the ERA-40 cells and country boundaries. We then linearly interpolate the population at the intersection level for years in the 1990s. For years before 1990, we use the country-level population growth from the World Development Indicators (WDI; World Bank 2012) to extrapolate population counts. Finally, we use the country-level crude birth rate from the WDI to obtain the number of

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 (3) and (7) of Table 3. This yields the excess infant mortality caused by each such malaria episodes. Finally, we sum the excess infant mortality across all these malarious episodes.

If we restrict attention to the ERA-40 cells in our sample, then the total excess deaths caused by these malarious episodes during 1981-2000 is 84,000 out of 139 million births. Extrapolating to all malaria epidemic areas of Africa – which are shown in Figure A4 of the Appendix – 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 percent of infant mortality.

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 – shown in Figure A5 – we estimate 11,000 extra infant deaths, relative to 206 million births. This is only 0.05 children in 1000, accounting for 0.05 percent of infant mortality. The reason that these additional death tolls are relatively small is of course that the underlying extreme events are infrequent.

Total number of infant deaths from malaria We can also estimate the total number of infant deaths caused by malaria in the epidemic regions. Our estimate is based on the fall in infant mortality when there are 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 for this reason non-malarious) with infants of mothers not given treatment (see below). 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. Note that we cannot similarly estimate the effect of a long-term reduction of malaria from an average 2-4 months to an average of 1-2 malarious months based on the estimates in Table 3. The reason is that adaptation (for example, partial immunity to malaria) would change. We can, however, estimate that change by comparing the effects of a drop to

births in each year and then aggregate it to the ERA-40 cell level.

zero malaria in the two regions.

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 when there are zero malarious months in the year before birth relative to the average year. Since the average infant mortality rate in these areas is 108.9 per 1000, malaria is estimated to account for 7.4% of infant mortality (95% confidence interval is 4-11%). We multiply this number by the total number of births in a year to 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.

Existing studies of infant deaths from maternal malaria How do these estimates of infant death in malaria compare with existing medical studies? Mortality estimates 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 use two types of alternative data sources. The first is epidemiological surveys, including randomized control trials, on the impact of medical treatment of malaria infection.[38] The second is 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. Both types of data, however, come from a limited number of selected populations in Africa.

We now compare our methodology with that used in studies using each type of data sources.

Epidemiological surveys The existing study based on epidemiological surveys with a scope most comparable to our own is probably Steketee et al (2001). These authors review studies on malaria infection in pregnancy and estimate that 3-8% of infant mortality in endemic areas is due to malaria

[38]There also exist some randomized control trials on the prevention of malaria. Insecticide-treated bed nets are, for example, estimated to reduce infant mortality by an average 5.5 deaths per 1000 births, 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.

in pregnancy. Of 34 reviewed studies, only two attempt to estimate the contribution of malaria to infant mortality, namely Greenwood et al. (1992) and Steketee et al. (1996). Both studies are unable to directly estimate the effect because sample sizes are not large enough to estimate effects on such rare events as infant deaths. Instead, both studies first estimate the effect of malaria medicine on birth weight by using a randomized control trial, and then combine this estimate with the correlation coefficient between low birth weight and infant mortality based on observational data. In the words of Steketee et al. (2001), "No study has made the direct observation because the required sample size to make the observation is prohibitively large." This issue is of course even more pronounced in non-endemic areas, which explains the paucity of epidemiological studies there.

Specifically, Greenwood et al. (1992) study villages near the town of Farafenni on the north bank of the Gambia river and finds that malaria medication could reduce infant mortality by 6% (from 50 to 48 per 1000). Stekeete et al. (1996) study women enrolled in four antenatal clinics in a highly endemic malaria area of rural Malawi and estimate the effect of malaria to 3-5% of infant mortality, at an average infant mortality rate of 157 per 1000. Steketee et al. (2001) summarize these estimates and adjust the summary estimate to an upper limit of 8%, to account for effects through anemia. Scaling the effects by regional infant mortality rates, the paper finds that globally, 75,000 to 200,000 infant deaths in each year might be attributable to malaria infection during pregnancy.

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

In comparison, our estimates 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 sample size of our data set (nearly a million births), we can estimate the effect on infant mortality directly and do not have make assumptions such as (E1)

and (E2) to draw conclusions. Our sample is also a lot more representative of all of Africa during the 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.

Verbal autopsy reports We now turn to studies based on verbal autopsy reports. The estimates of malaria's contribution to mortality of children aged below 5 in the WHO's World Malaria Report are based on results from meta-regression studies. For example, Morris et al. (2003) use data from 38 verbal autopsy studies (14 in Africa) to establish the cause of death. The share of deaths in each study attributed to each of five causes (relative to the share attributed to pneumonia), is regressed on a number of characteristics. The ratio of malaria to pneumonia deaths, for example, is regressed on the estimated population at risk of malaria based on a model similar to ours (the MARA model), the proportion of births attended by a qualified professional, access to safe drinking water, and a South Asia dummy variable. The regression coefficients are then used to predict the distributions of under-5 deaths by cause for the entire region of Africa. Using this methodology, the study concludes that 24 percent of child deaths in sub-Saharan Africa are caused by malaria. Later studies such as Johnson et al. (2010) and Black et al. (2010) reach similar numbers based on a larger number of verbal autopsy studies. For Africa as a whole, these authors attribute 16% of under-5 deaths to malaria.

While such cross-sectional regressions may predict the relative importance of mortality causes, there are concerns about their ability to identify causes of deaths. Three obvious ones are: (V1) cross-sectional correlations may reflect confounding factors, (V2) errors in the verbal autopsy reports correlate with the regressors, (V3) children may die of multiple illnesses, and deaths due to malnutrition are assigned to other causes. Regarding (V1), many omitted factors, like agricultural production, may correlate with the malaria index in the cross-section. With respect to (V2), verbal autopsy may poorly differentiate malaria from other diseases that cause fever symptoms (e.g., septicaemia, viral encephalitis or pneumonia), and thus fever may more likely be seen as a sign of malaria in endemic areas. This alone would create a correlation between malaria assigned as a cause of death and the population at risk of malaria, biasing the estimated coefficient on this factor.

Concerning (V3), extrapolating to the effects of eliminating malaria is not

straightforward. The verbal-autopsy methodology is designed to predict the relative importance of mortality causes, not the absolute number of deaths, which is taken as exogenous. On one hand, as children die of multiple illnesses, eliminating malaria may reduce vulnerability to other illnesses. On the other hand, weak and vulnerable children, whose deaths are attributed to malaria, may increasingly die of other causes when malaria is eliminated.

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 hence we capture the effect on total infant mortality directly, avoiding concerns (V2) and (V3).

Existing studies of malnutrition and infant mortality Our results on how weather influences infant deaths are supported by the fact that, in arid areas, the same weather pattern (droughts) that influences crop prices (and thus likely nutrition) also affect infant mortality. How do they compare with existing research on the effects of malnutrition on infant mortality? Let us compare our aggregate estimate of 11,000 infant deaths due to droughts in 1981-2000 with numbers from the EM-DAT data base of the Centre for Research on the Epidemiology of Disasters. This data records 195 droughts in Africa 1981-2000, involving a total of 554,756 killed. Assuming that infants make up 3% of the population (from the WDI crude birth rate), and that all ages are hit uniformly, this would imply 16,600 excess infant deaths due to drought over the twenty-year period. However, a uniform distribution of mortality risk across ages may not be realistic. De Waal (1989) found that infants were less affected by Sudan drought in 1984-1986 than average, because of late weaning.[39] Similarly, Ezra and Kiros (2000) find that infants were less affected by the Ethiopia drought in 1984-1985.

But the vast majority (99%) of the deaths recorded by EM-DAT are

[39]De Waal conducted a survey of 1182 households in Sudan in 1986, investigating the second deadliest drought in our sample period. He found that infant mortality was not higher during the drought 1984-1986 than typical, although infant deaths may have been under-enumerated. He argues that a reason that infants were protected from the drought was habits of late weaning. The infants were less vulnerable to many of the diseases associated with unclean water supplies or inferior foods, and had the protection against infection provided by their mothers' milk.

associated with a mere three droughts in the mid 1980s (Ethiopia, Sudan and Mozambique). Our mortality toll is much more dispersed in time and space. It captures 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.

More generally, estimates of the relation between malnutrition and infant mortality in existing studies are not directly comparable to ours. 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 in the 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. When such studies are used to predict infant mortality levels, they may not capture the causal effect of malnutrition on child mortality. In particular, 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.

Another approach is to rely on randomized experiments to assess the effect of nutrition on child 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. Ceesay et al. (1997), mentioned above, find large effects on fetal growth and mortality. However, this particular study provided much higher energy supplements than typical to under-nourished women from Gambia. 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, and the average response of infant mortality to these changes.

Areas at risk and their evolution over time We have identified the African areas most vulnerable (in terms of infant survival) to weather shocks, namely epidemic malaria zones and arid climate zones. What can we say

about areas at risk when the climate changes? As a lead-in to this question, we can ask if the location of vulnerable areas has changed during the past three decades, possibly due to ongoing climate change. For this purpose, we split the 28-year period from September 1974 to August 2002 in half and classify ERA-40 cells into different malaria zones and climate zones in each subperiod.

Appendix Figure A6 shows where malaria zones shifted (and the average number of malaria months per year changed by one or more) from the first subperiod (before 1988) to the second (after 1988). Some of the areas just south of the Sahel region became less malarious, while some areas in eastern and southern Africa became more malarious.[40] Similarly, Appendix Figure A7 shows where the climate zone shifted between arid and rainy areas.

Now we turn our attention to the implications of our findings on the possible impacts of climate change in the future. Projections of future climate indicate that Africa will get significantly warmer. Areas hitherto non-malarious due to cold temperature – mountainous regions in Ethiopia, Kenya, Madagascar, and Zimbabwe close to the yellow dots in Figure 5 – are thus likely to become new epidemic areas, where mothers will have little immunity and infants will be very vulnerable to temporary malaria epidemics. Moreover, if future climate change leads to more dramatic variations in the annual fluctuations of rainfall in the drier parts of Africa, this too will shift the distribution of malaria exposure – epidemics may become more frequent in low-prevalence areas, with potentially large effects on infant mortality. [**We are currently obtaining climate predication data to be more precise about these future patterns. Specifically we will create maps similar to Figures A6 and A7 of the areas that may become more or less malarious, and arid or rainy, in the last three decades of the 21st century compared to our sample period.**]

To further illustrate these possibilities, Figure A8 shows whether the epidemic episodes we identify in sample are mainly caused by higher temperature or larger precipitation.[41] Epidemic episodes in Ethiopia and Kenya are

[40] This observation is consistent with papers that point to a resurgence of malaria in the East African Highlands from the mid 1980s, typically citing evidence that derives from a small number of clinics (see e.g., Hay et al. 2002) or verbal autoposies.

[41] For each ERA-40 cell and month where the number of malaria months in the past 12 months contain an epidemic, we check whether the past 12-month average monthly temperature and precipitation are higher than the 1957-2002 average. If only temperature (precipitation) accounts for at least 90% of the epidemic months in a given cell, we say that

mostly caused by larger-than-usual precipitation. This is consistent with the notion that El Nino episodes are associated with wetter condition in East Africa (recall Section 2). That the epidemics are mainly driven by rainfall illustrates how larger fluctuations of rainfall in a future climate may lead to more frequent malaria epidemics. On the other hand, the epidemic episodes in Zambia are mostly caused by higher temperature than usual. This illustrates the possible epidemics caused by a higher frequency of warm enough weather as the climate gets generally warmer.

To make this discussion more precise, we use the data on predicted monthly temperature and precipitation for the period 2071-2100.[42] We use these data to identify the areas of Africa that will become more vulnerable to weather shocks in terms of infant survival by becoming an epidemic area or an arid area (or by a higher frequency of malaria epidemics and droughts in areas that are already epidemic or arid). We then use our estimates in the paper to estimate how many infants may be expected to die due to certain types of weather shocks at the end of the 21st century.

[Results and discussions to be inserted here.]

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 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 sea-

the episodes are predominantly caused by temperature (precipitation). This is indicated by orange (blue) in the figure. If both temperature and rainfall, or neither of the two, explain at least 90% of the epidemic episodes, we regard epidemics caused by both, indicated by yellow in the figure.

[42] We use the climate prediction data derived from the EC-EARTH model, the climate model closest to the one used in the ERA-40 reanalysis, and based on two emission scenarios under the CMIP5 (Climate Model Intercomparison Project phase 5) project: a high emissions scenario (RCP8.5) and a midrange mitigation emissions scenario (RCP4.5). See Taylor et al. (2012) for the detail on the CMIP5.

son. The malaria epidemic and drought effects we estimate are statistically robust and quite large.

The continental scale in our study leads to a bleak picture for certain parts of Africa, especially in some areas with scant rainfall that also suffer from bursts of epidemic malaria, such as the mountainous areas in East Africa. Due to the erratic movements of the Intertropical Convergence Zone, these areas face large variations in annual rainfall. 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. We have also shown how to use our results to estimate the total number of infant deaths due to maternal malaria in epidemic areas.

In terms of methodology, we have outlined a possible research design for 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 this methodology allows us to draw reasonable conclusions about the aggregate impacts for all of Africa in the past few decades.

We have also indicated how some of our results could be used to identify areas at risk for large future health impacts in the wake of future climate change. **[Say more in light of the, now missing, results]** On this front, much more work is needed to come up with more robust conclusions.

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 child mortality, child health, or fertility. Perhaps one may also study more complex issues, such as generational spillovers, whereby girls with negative weather shocks in early life become physically or cognitively impaired adults and thus face a larger risk of bad outcomes when they give birth themselves.

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

These and other tasks are left for future research.

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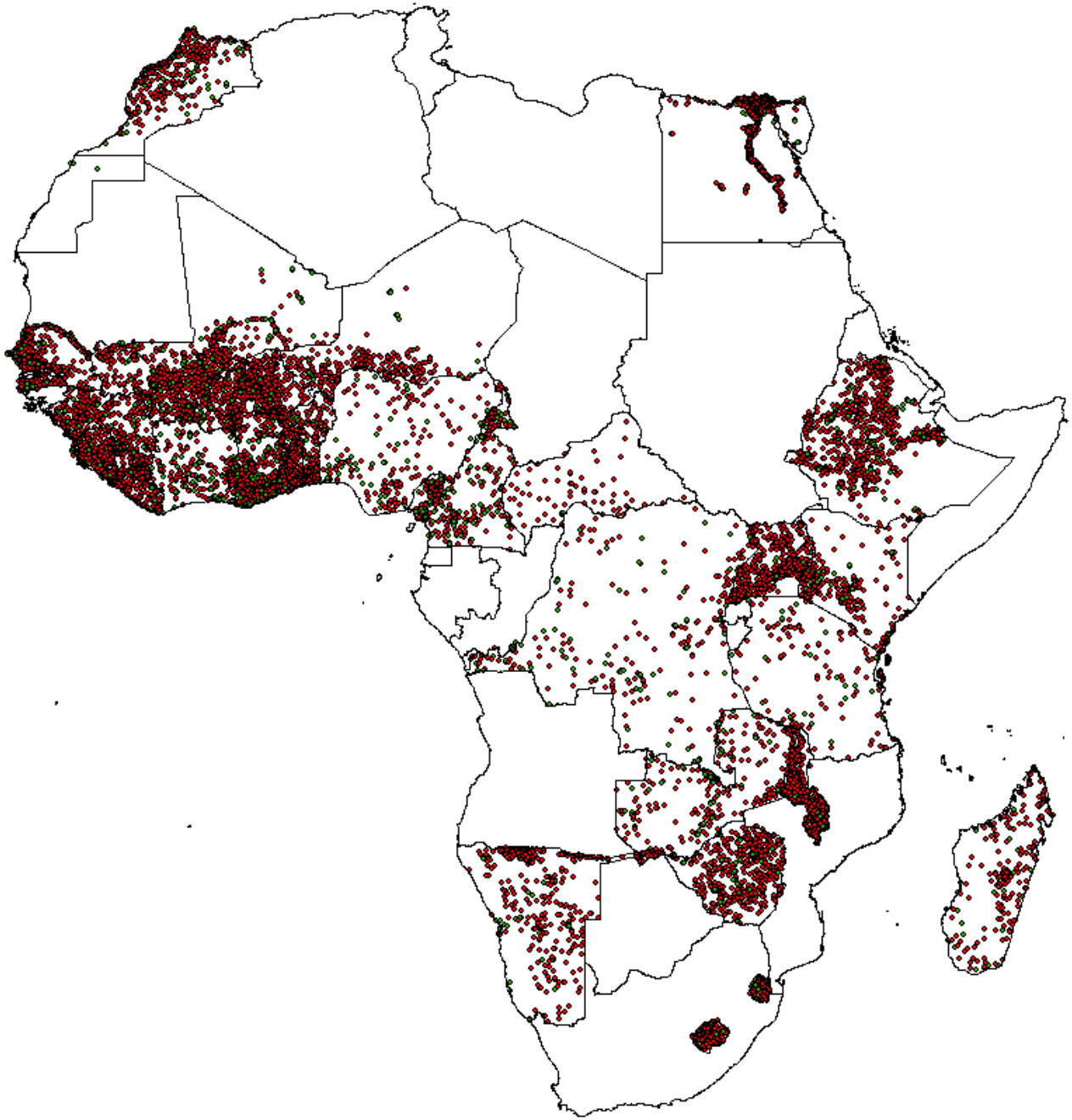

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Figure 1 – DHS Clusters in the Sample

Notes: Green circles indicate urban clusters; red ones indicate rural clusters

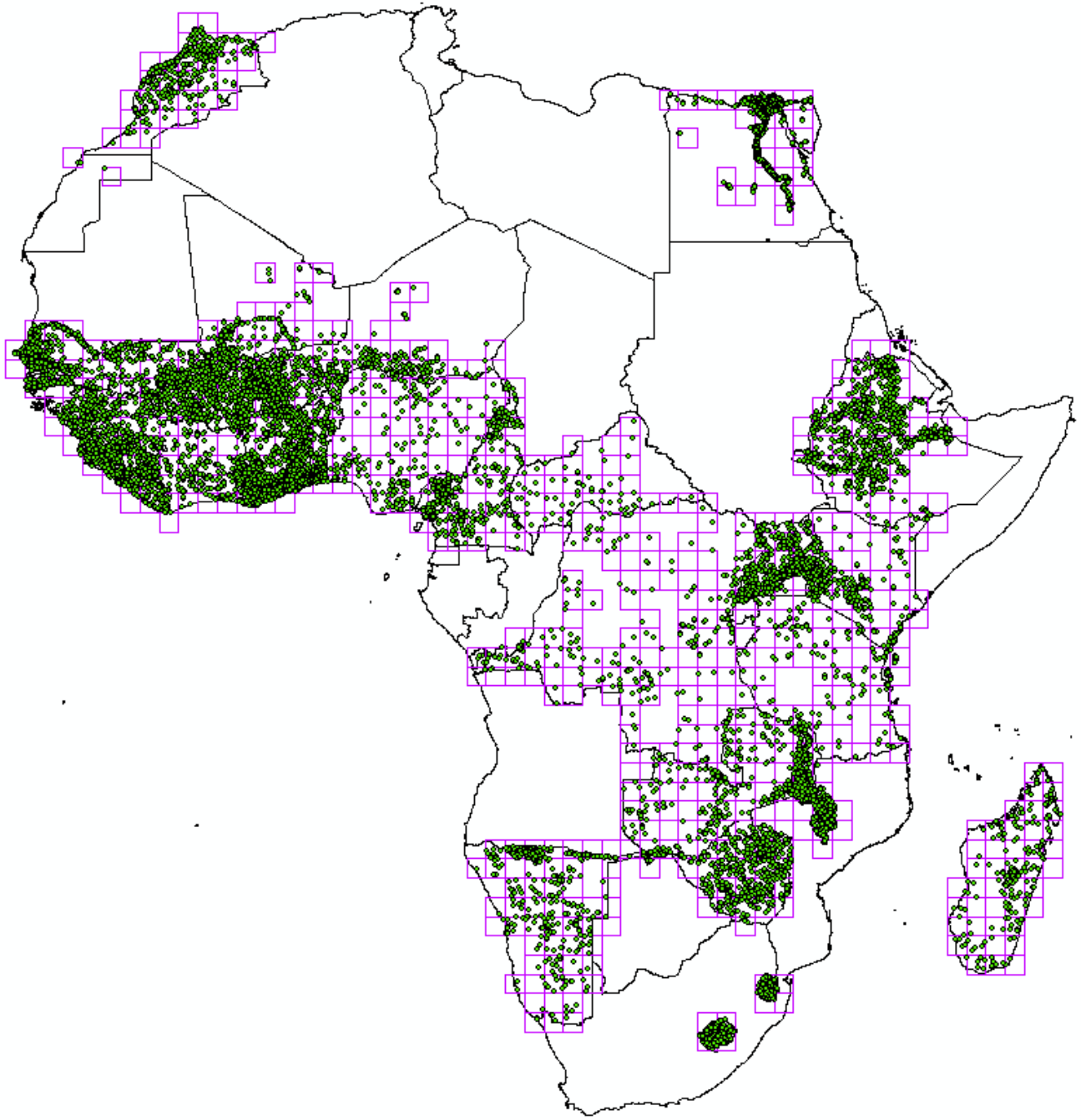


Figure 2 – ERA-40 Grid and DHS Clusters in the Sample

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

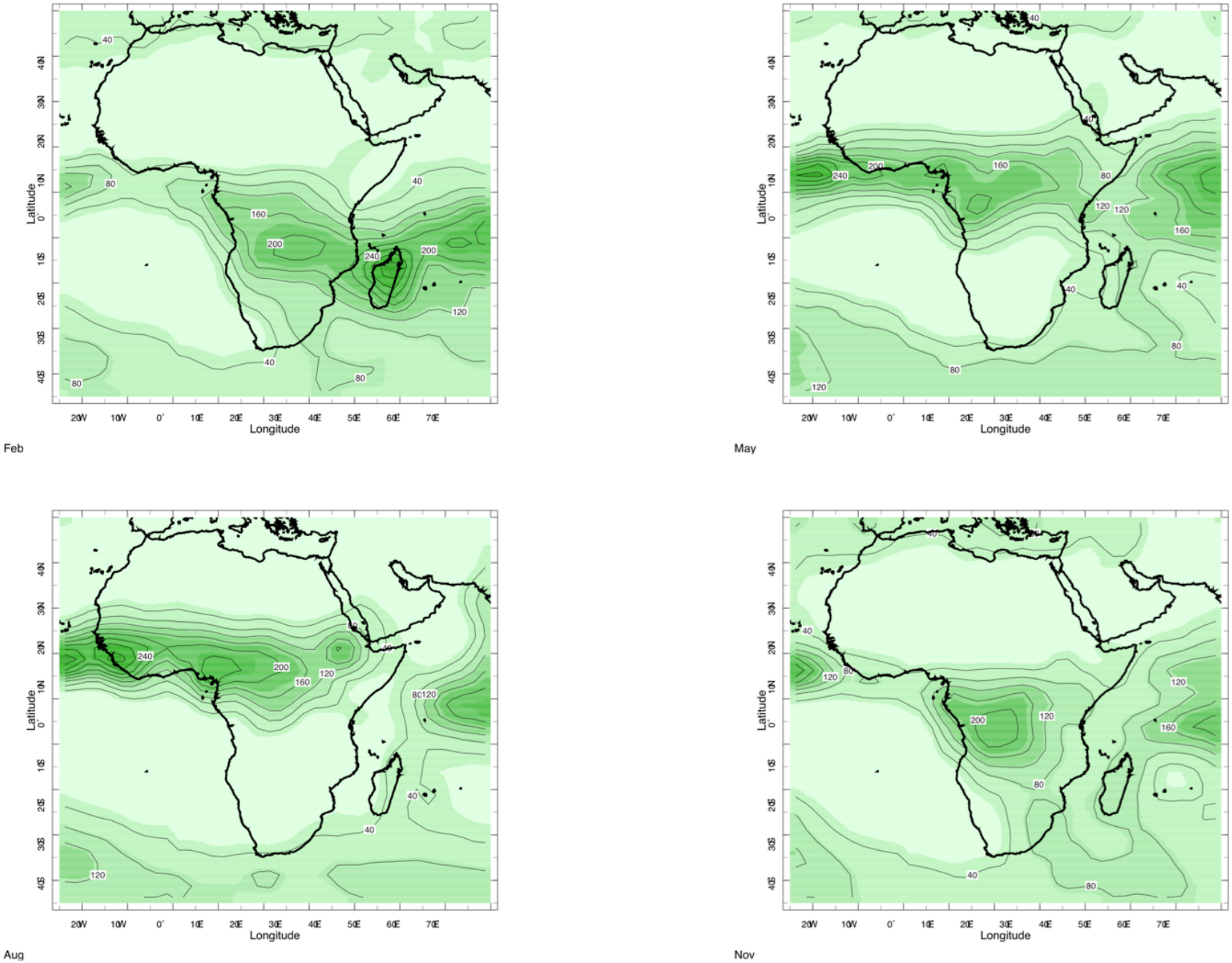


Figure 3 – Total monthly rainfall (in mm) in Africa for February, May, August, and November

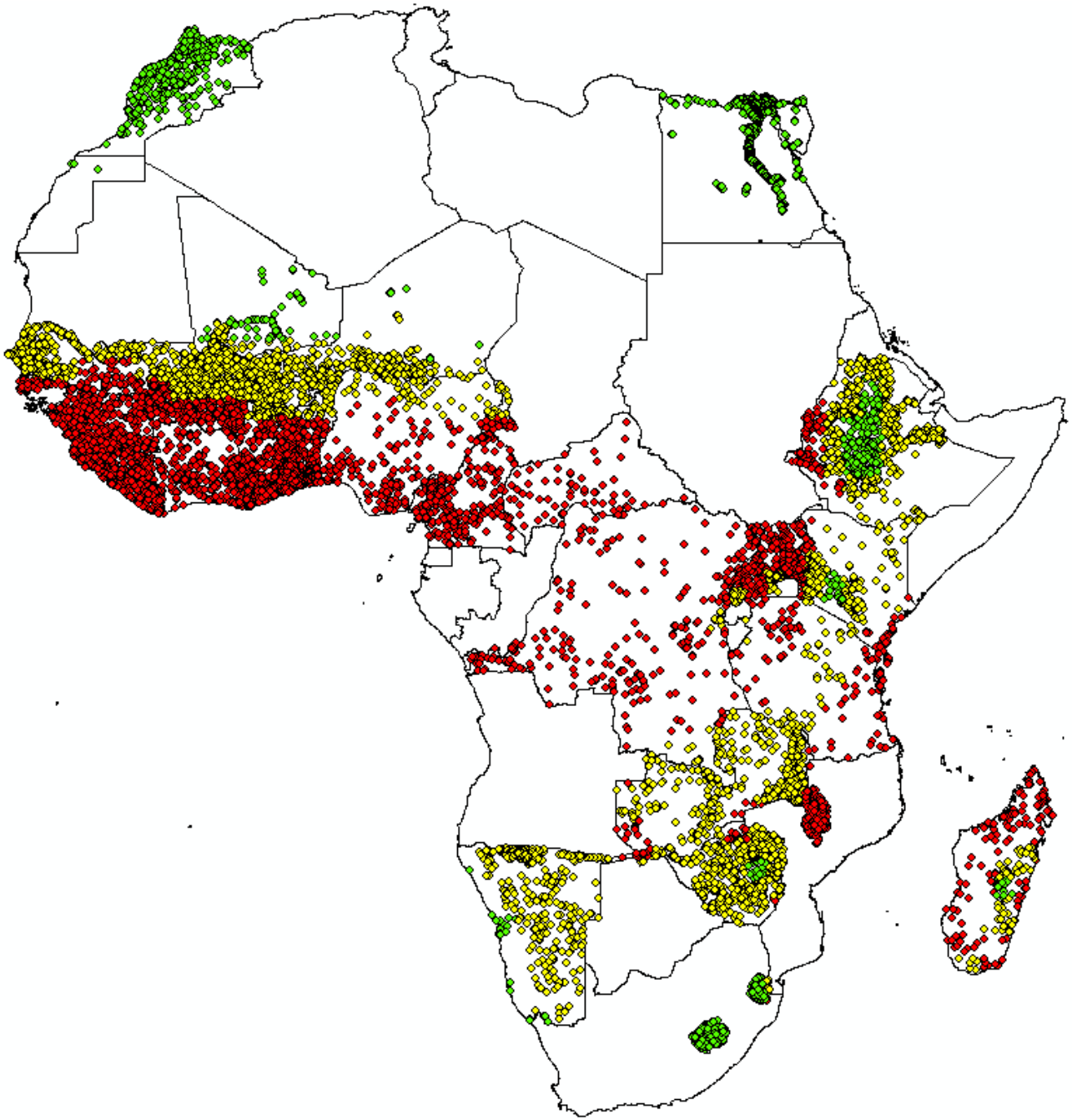


Figure 4 – Malaria Exposure Zones in Africa

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

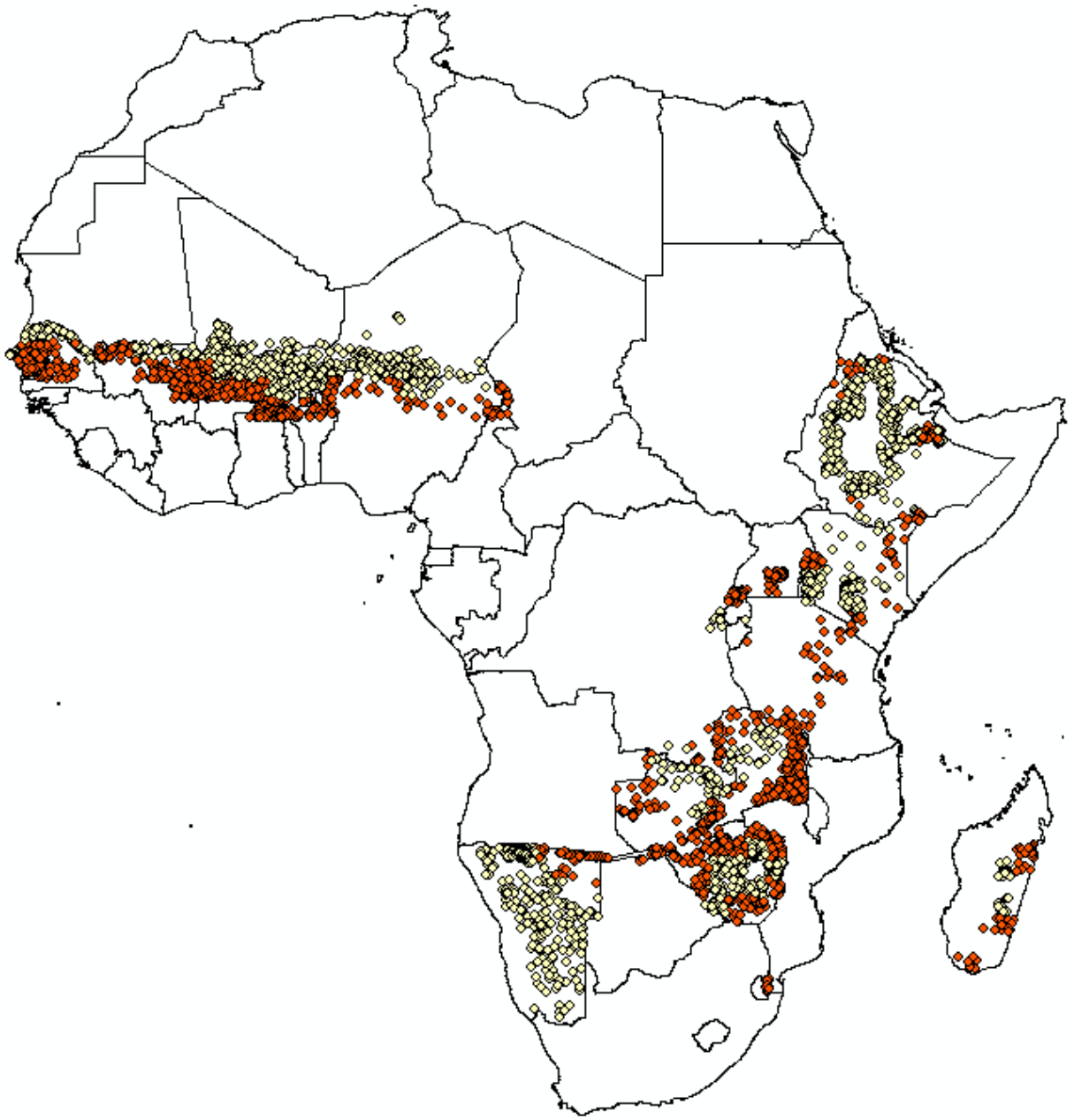


Figure 5 – Low and High Epidemic Malaria Exposure

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

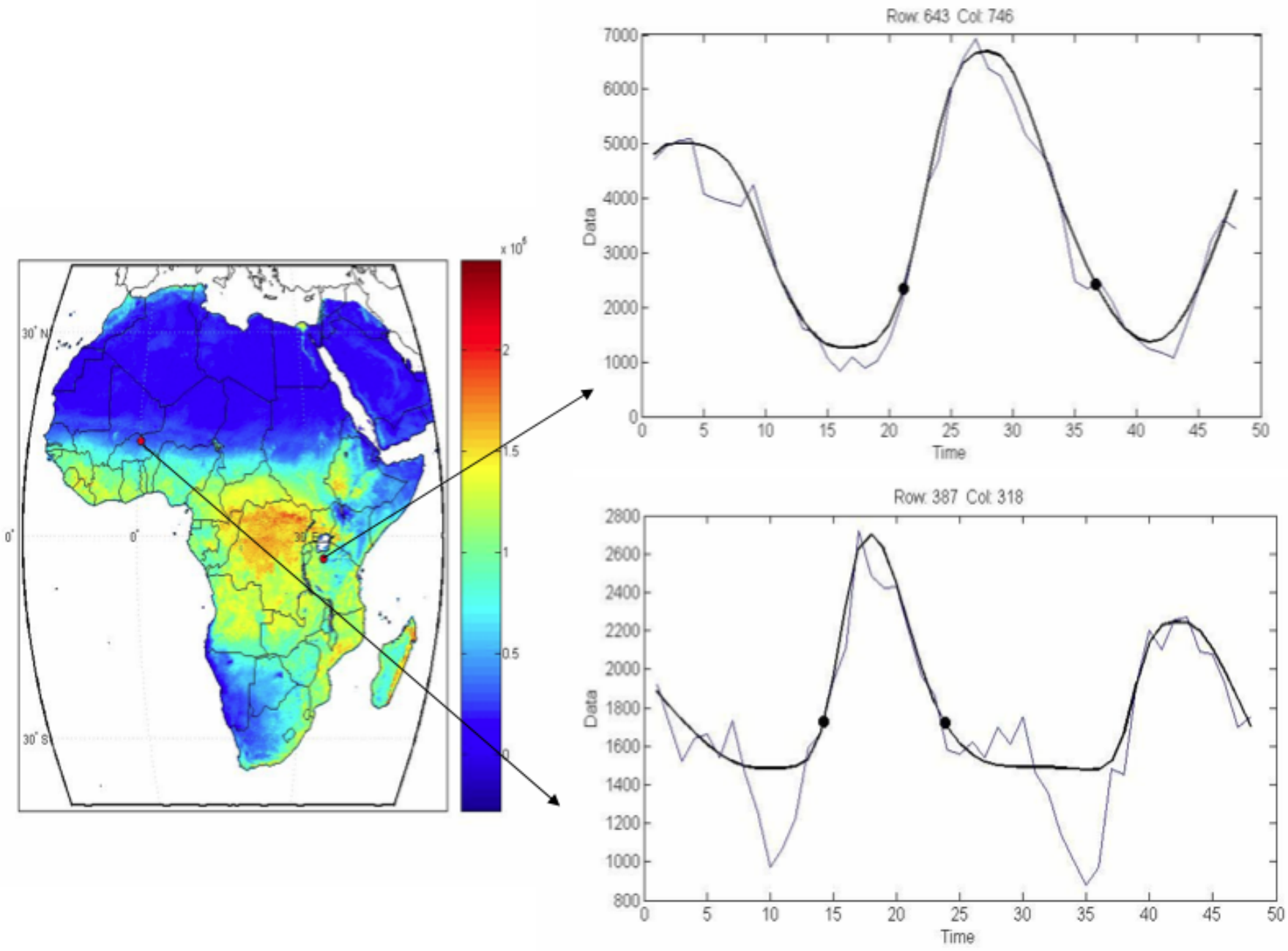


Figure 6 – Actual and fitted NDVI in Burkina Faso and Tanzania

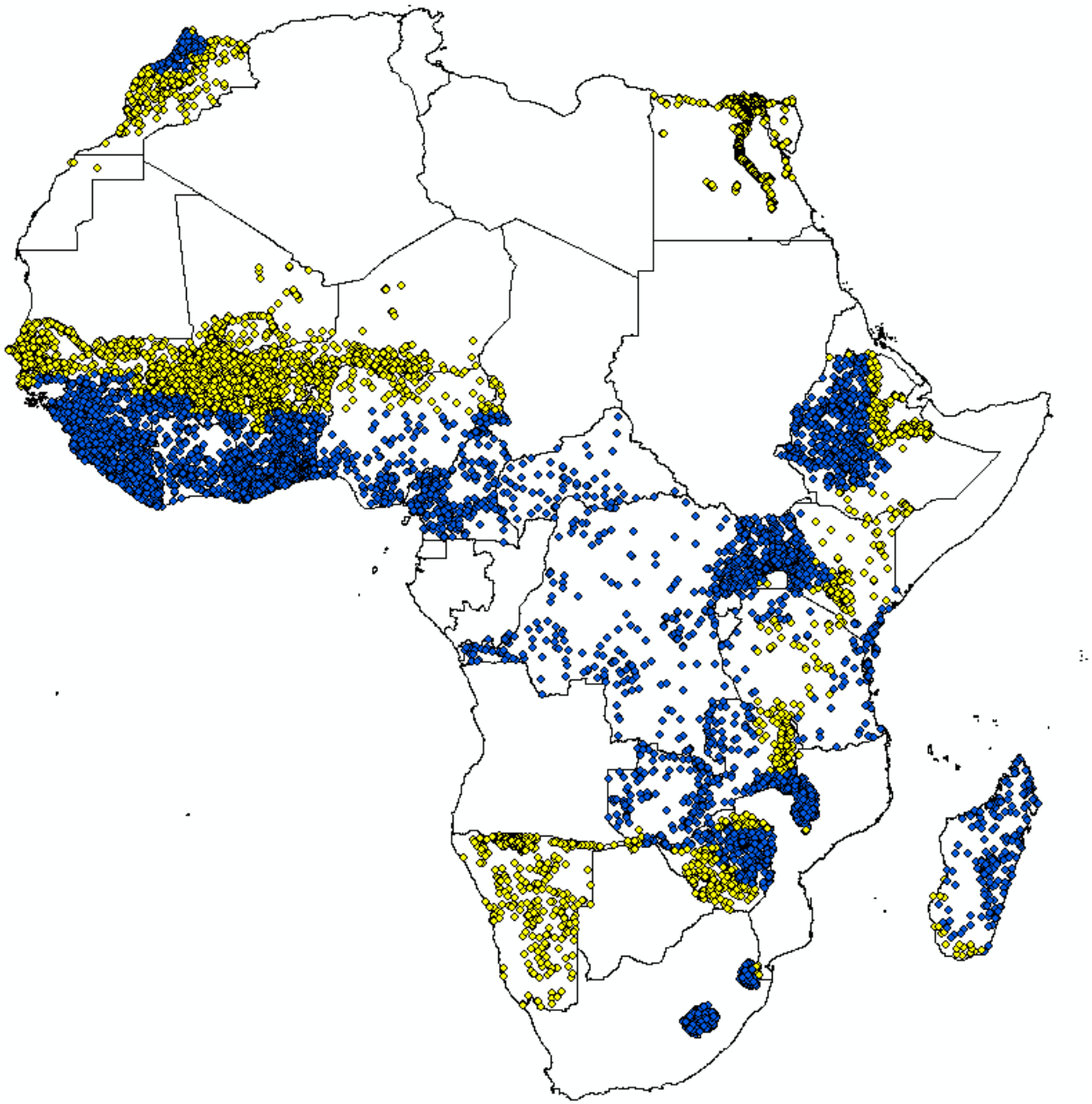


Figure 7 – Arid and Rainy Climate Zones in Africa

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.

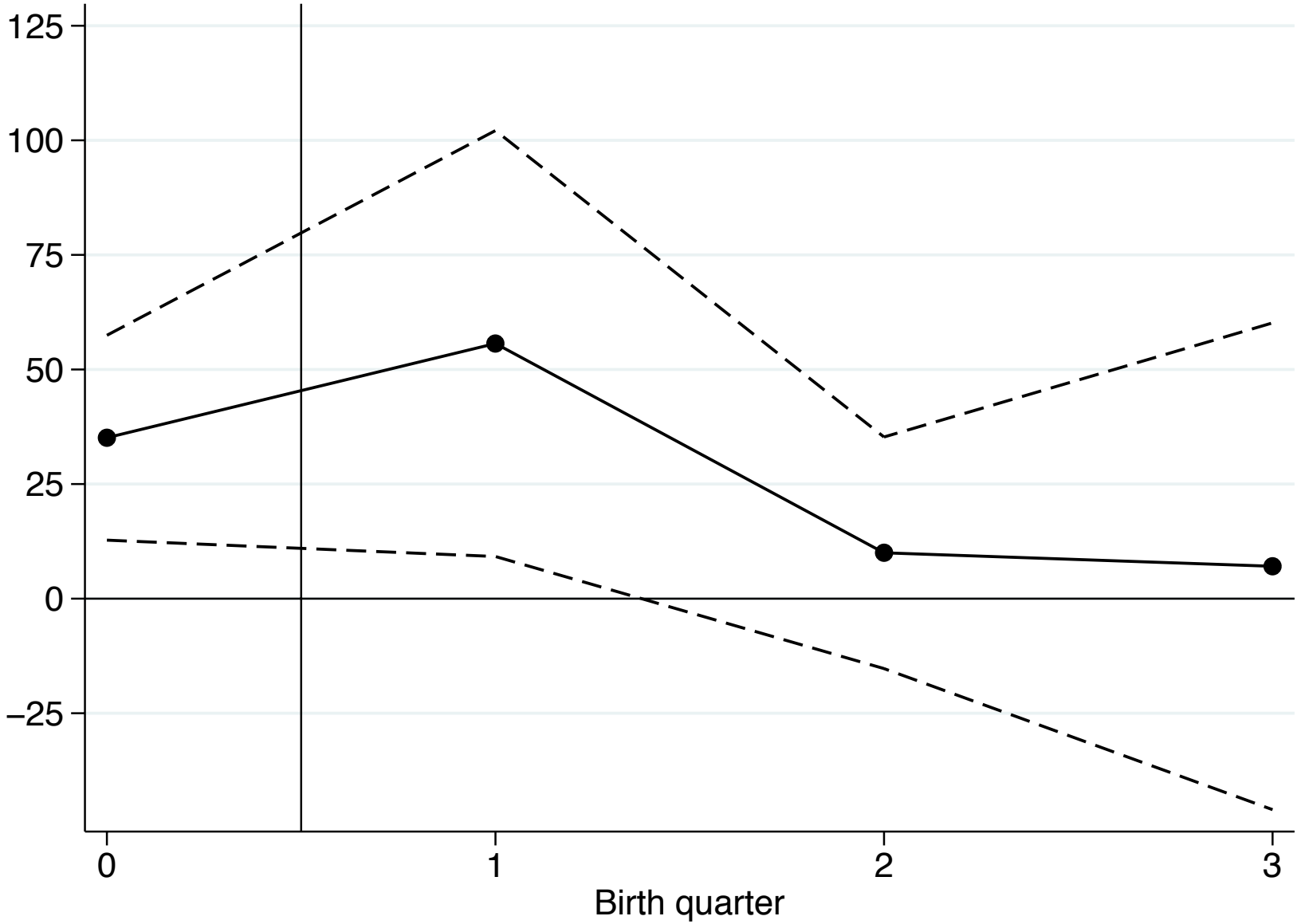


Figure 8 – Infant Death and Drought by Birth Quarter relative to the Beginning of the Growing Season

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.

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.

Appendix

Data We use crop-price data compiled by the USAID Famine Early Warning Systems Network (FEWS NET).[43] Specifically, we sample six major crops in Africa (maize, rice, wheat, cassava, millet, and sorghum) and markets in eight countries, for which we also have data for infant mortality (Burkina Faso, Ethiopia, Kenya, Malawi, Mali, Tanzania, Uganda, and Zambia).[44] Price data is aggregated to the monthly frequency if the original data is daily or weekly. The geographic coordinate of each market in the data is obtained by searching the name of the market in the National Geospatial-Intelligence Agency's Geonames Search.[45] The ERA-40 weather data used in our infant-mortality analysis is then matched spatially with each market in ArcGIS 9.3 (the Spatial Join tool). Figure A1 shows the locations of 424 markets in the sample with the color indicating which climate zone (rainy or arid) the market belongs to. The sample period is from 1970 to 2002.

Empirical strategy We estimate the following regression equation:

$$\ln p_{m,t}^{p} = \alpha_{m,s}^{p} + \beta_{c,y}^{p} + \gamma r_{1}^{g,t} + \delta D_{g,t} + \varepsilon_{m,t}^{p}$$

where $p_{m,t}^{p}$ is the price (in domestic currency units) of crop p in market m (located within grid cell g and in country c) in running month t (which is month s of year y), $r_{g,t}^{1}$ the total amount of rainfall during the previous completed growing season (corresponding to the first term on the right-hand side of equation (3)) in grid cell g, and $D_{g,t}$ the indicator for $r_{g,t}^{1}$ being two standard deviations below the location-specific mean.[46] We control for crop-by-market-by-month fixed effects, $\alpha_{m,s}^{p}$, so that the impact of weather is

[43] We downloaded the data from `earlywarning.usgs.gov/adds` in October 2009. Since then, the FEWS NET has decided to discontinue the data distribution because, according to James Rowland at USGS, they are unable to keep the data up-to-date.

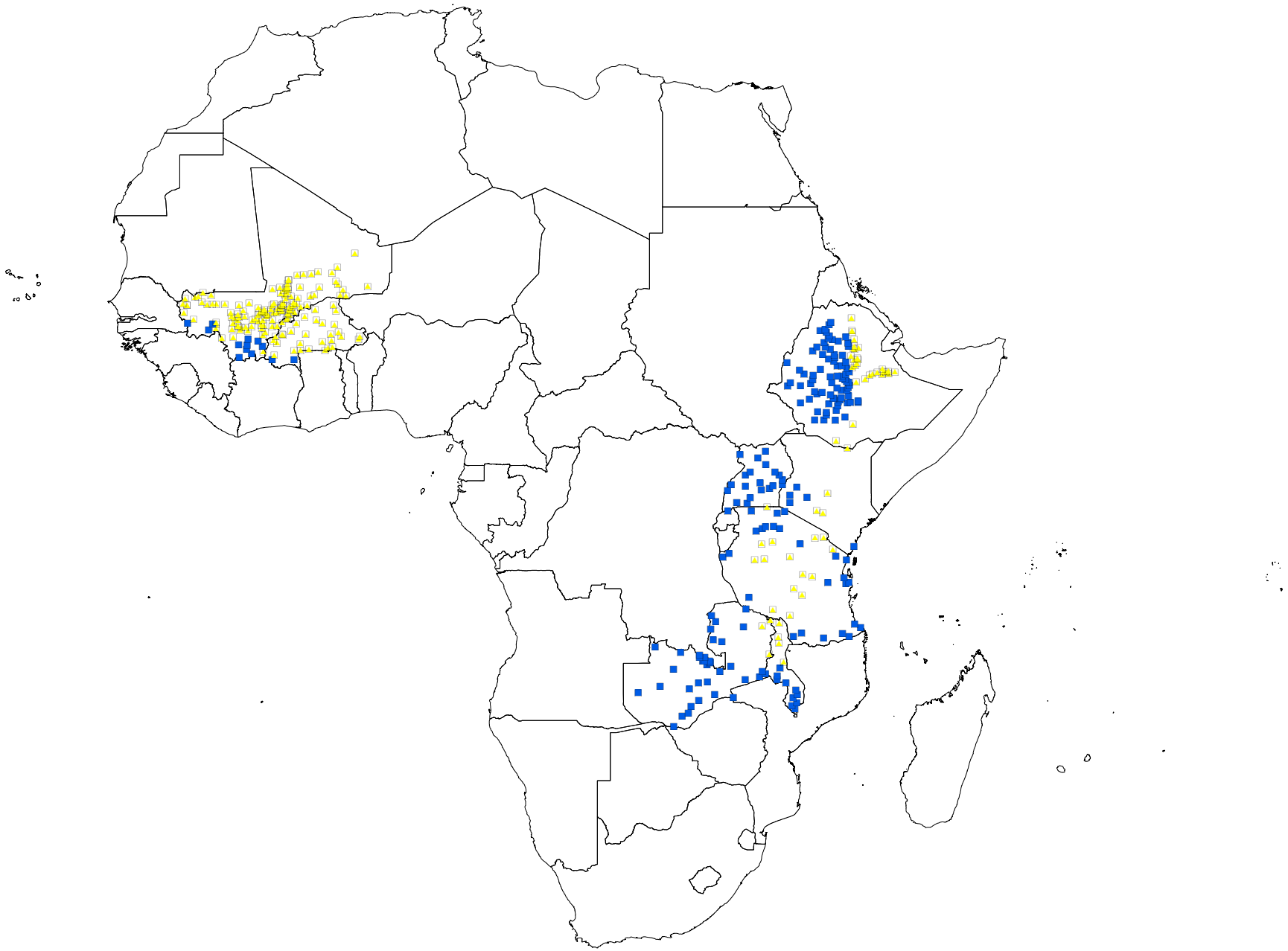
[44] These six major crops account for 57% of calorie availability in Africa in 2000 according to FAO's Food Balance Sheets. In the price data, each crop has subcategories (in flour, dried, fresh, etc.). We treat each subcategory as a single crop when we create fixed effects. Therefore, there are more than 6 crops in the sample.

[45] The address is `geonames.nga.mil`. If the name of the market cannot be found, we use Global Gazetteer Version 2.1 (`www.fallingrain.com/world`), Wikipedia and then the Google search as the final resort.

[46] Note that $D_{g,t}$ is different from $d_{g,t}$ defined in equation (4). It is defined over $r_{1}^{g,t}$, not over $r_{g,t}$.

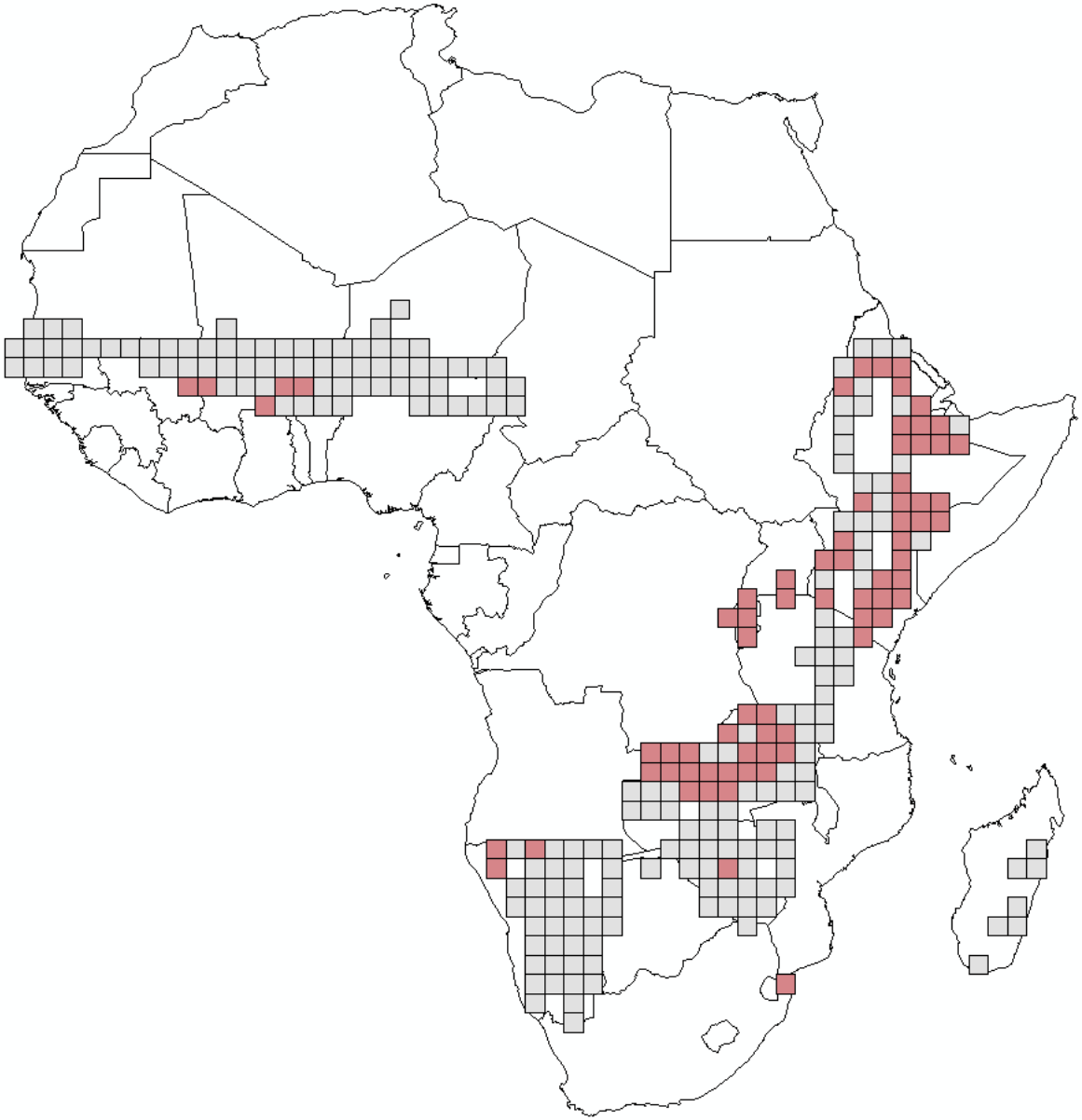
identified from year-to-year deviations from the average location-by-crop specific seasonal pattern. We also control for crop-by-country-by-year fixed effects, $\beta^p_{c,y}$, so as to take into account crop-by-country-specific non-parametric trends, as well as national price inflation and exchange-rate changes. The coefficients of interest, γ and δ, measure the percentage change in price due to a one-centimeter increase in growing-season rainfall and due to unusually low growing-season precipitation, respectively. We estimate this equation separately for rainy and arid areas with standard errors clustered at the ERA-40 grid-cell level.

The results are displayed in Table 4.



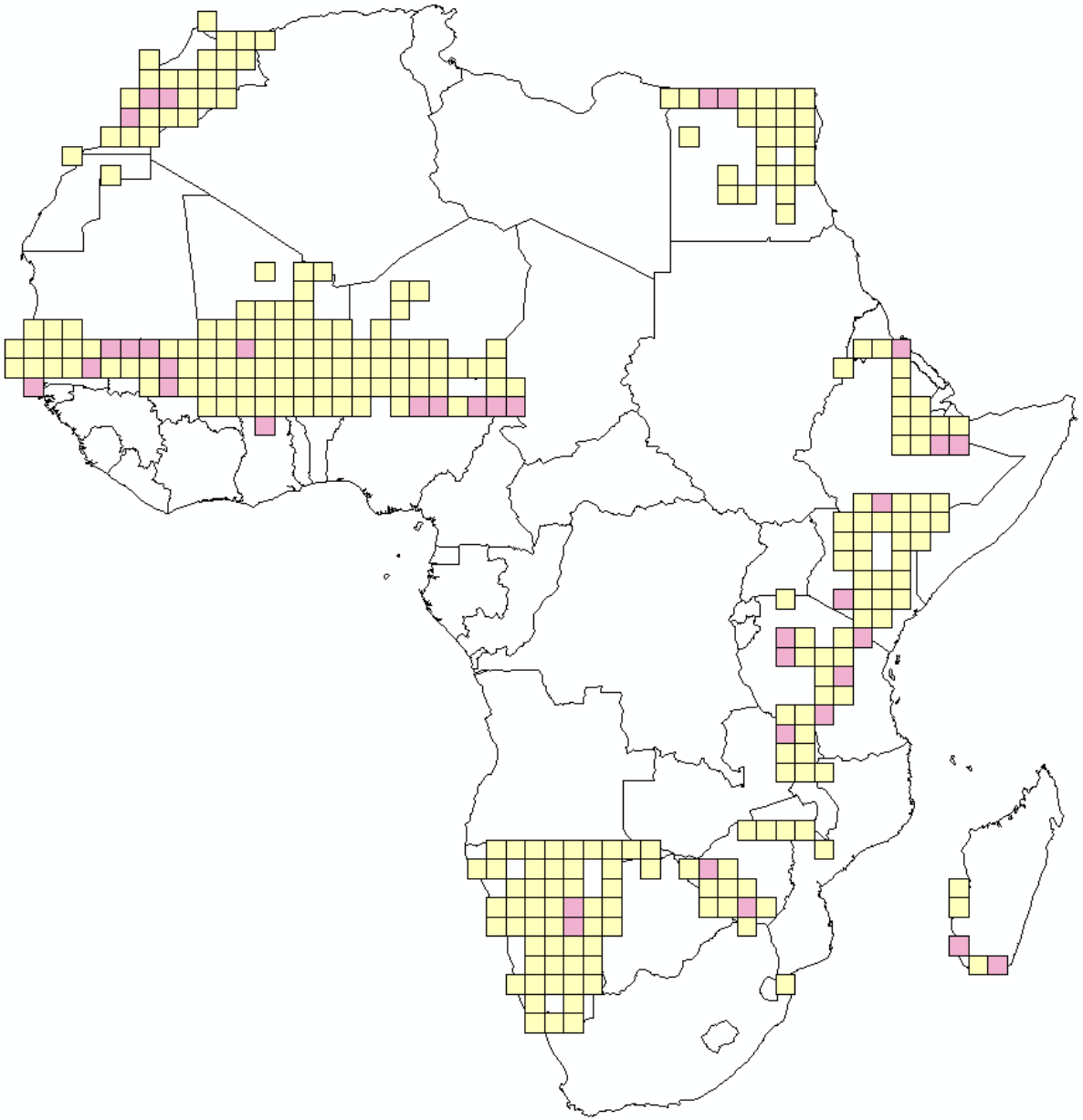
Appendix Figure A1 – Crop markets in the Sample

Notes: Blue and yellow squares indicate markets in rainy and arid areas, respectively.



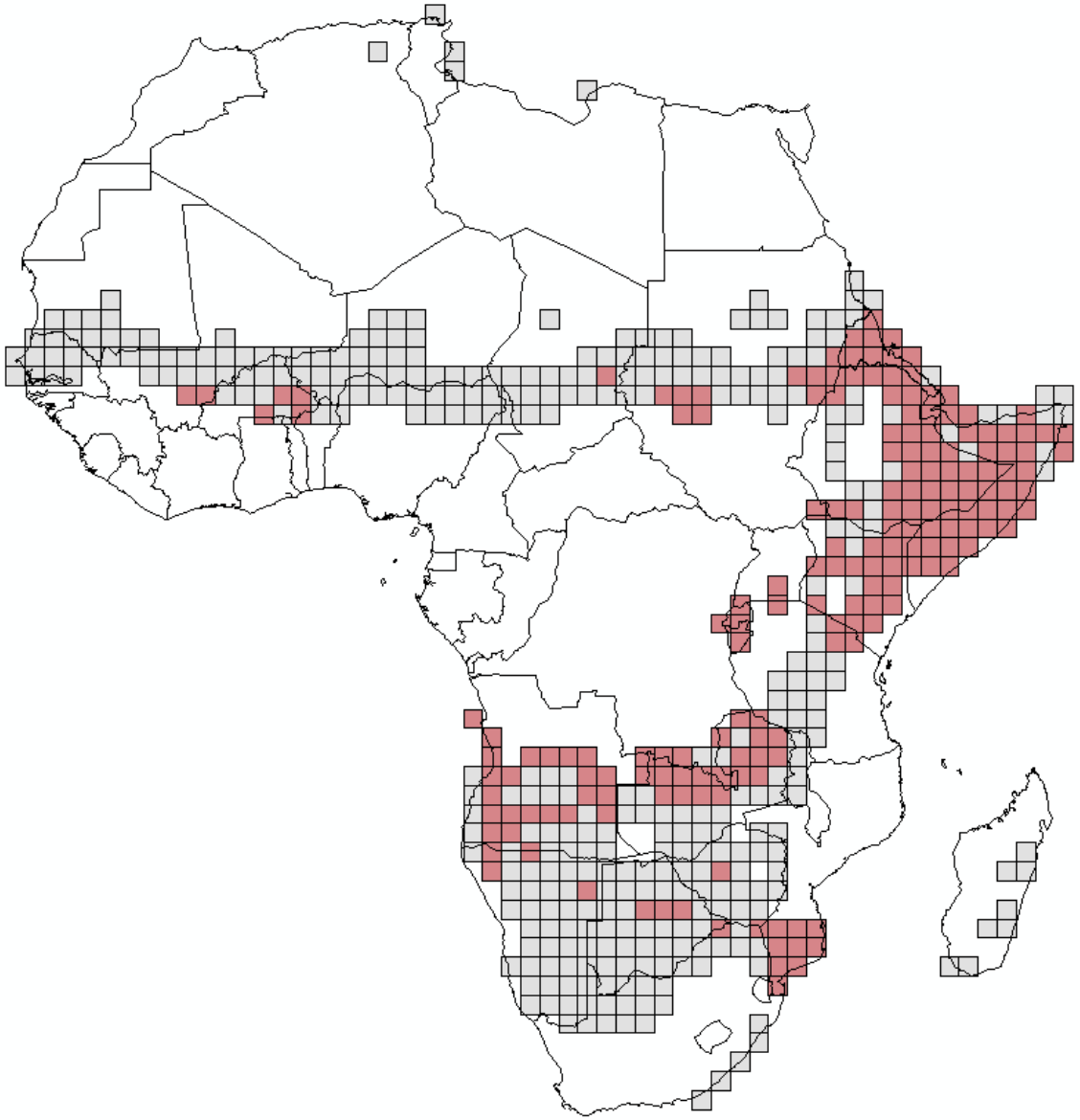
Appendix Figure A2 – Malaria Epidemics in the Sample

Notes: Colored squares indicate epidemic areas in our sample. Red squares indicate where there is at least one incidence of malaria epidemic (5 or more malaria months in the 0-2 month area; 7 or more in the 2-4 month area) for births observed in the sample.



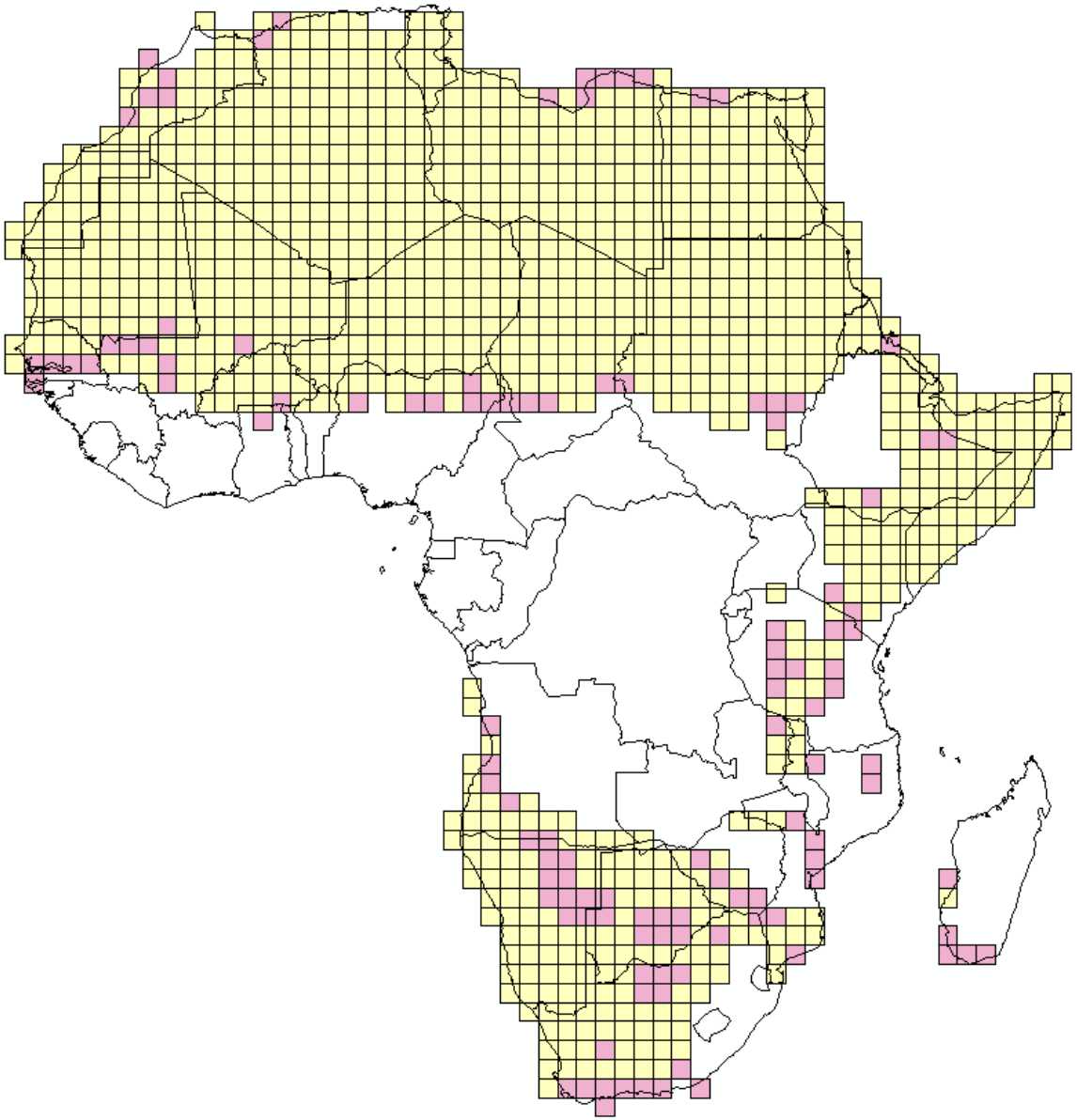
Appendix Figure A3 – Droughts in the Sample

Notes: Colored squares indicate arid areas in our sample. Red squares indicate where there is at least one incidence of drought for births observed in the sample.



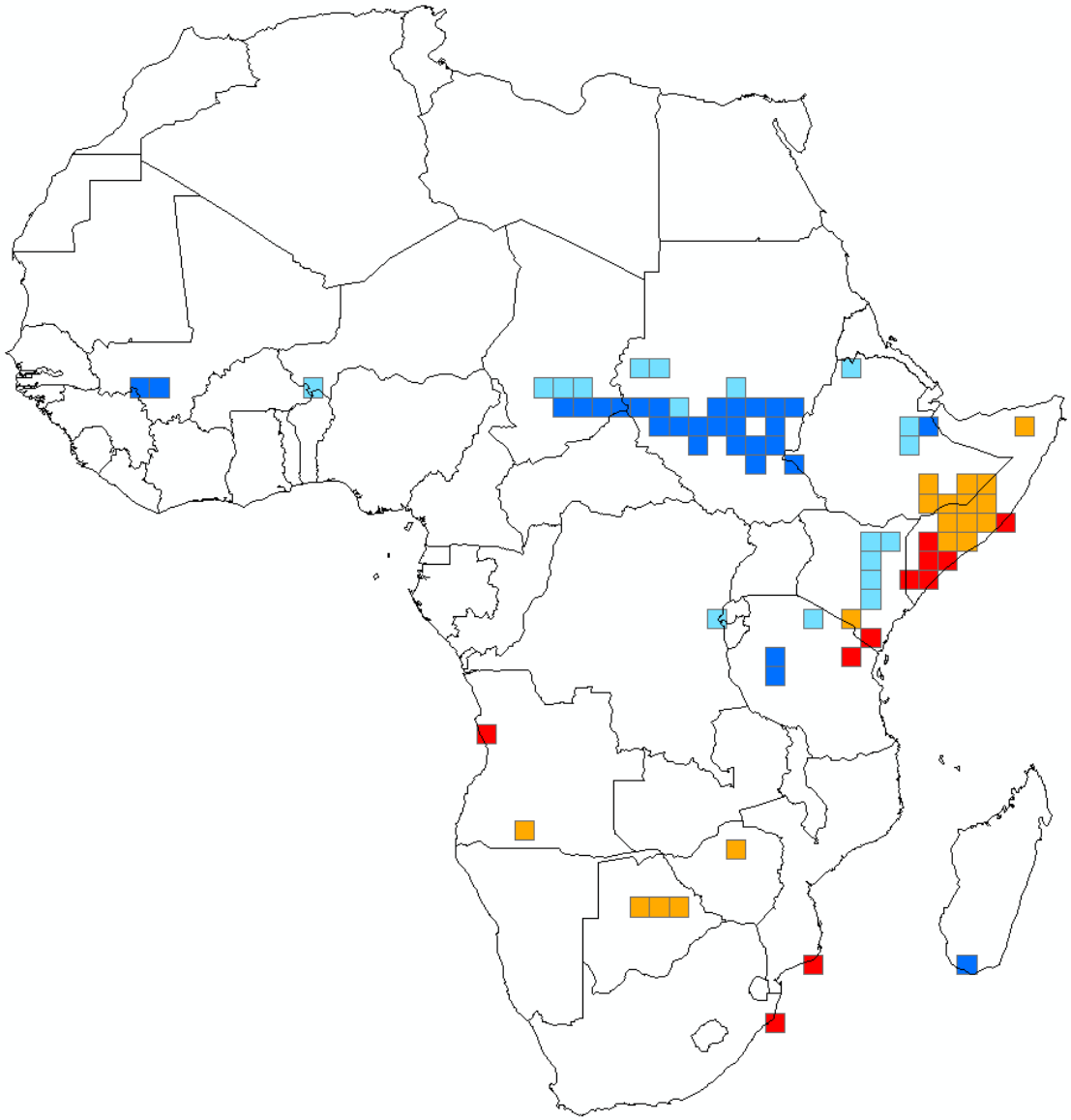
Appendix Figure A4 – Malaria epidemics for the whole Africa

Notes: Colored squares indicate all the epidemic areas in Africa according to the ERA-40 data. Red squares indicate where there is at least one incidence of malaria epidemic (5 or more malaria months in the 0-2 month area; 7 or more in the 2-4 month area) in the period 1958-2002.



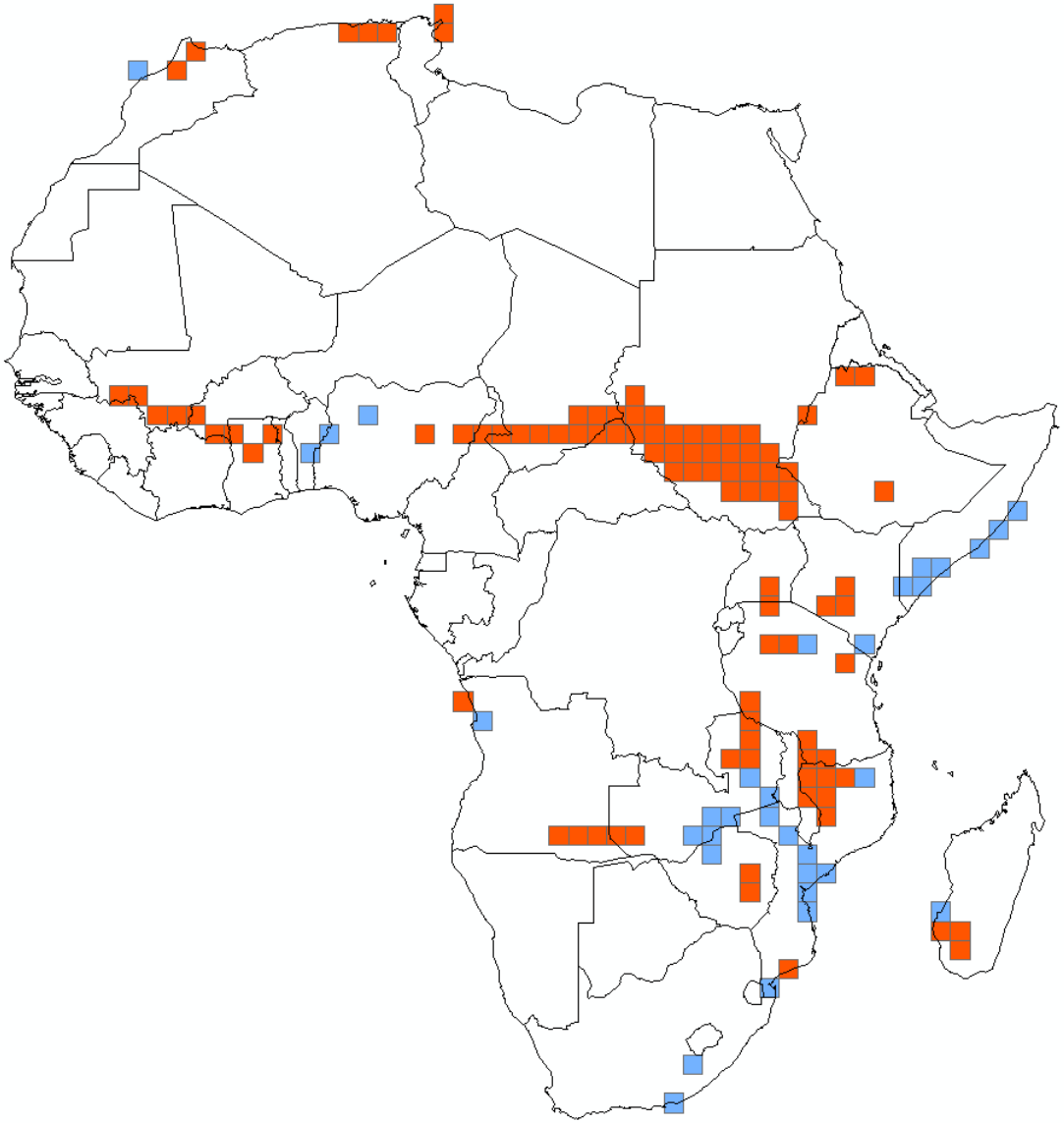
Appendix Figure A5 – Droughts for the whole Africa

Notes: Colored squares indicate all the arid areas in Africa according to the ERA-40 data. Red squares indicate where there is at least one incidence of drought in the period 1958-2002.



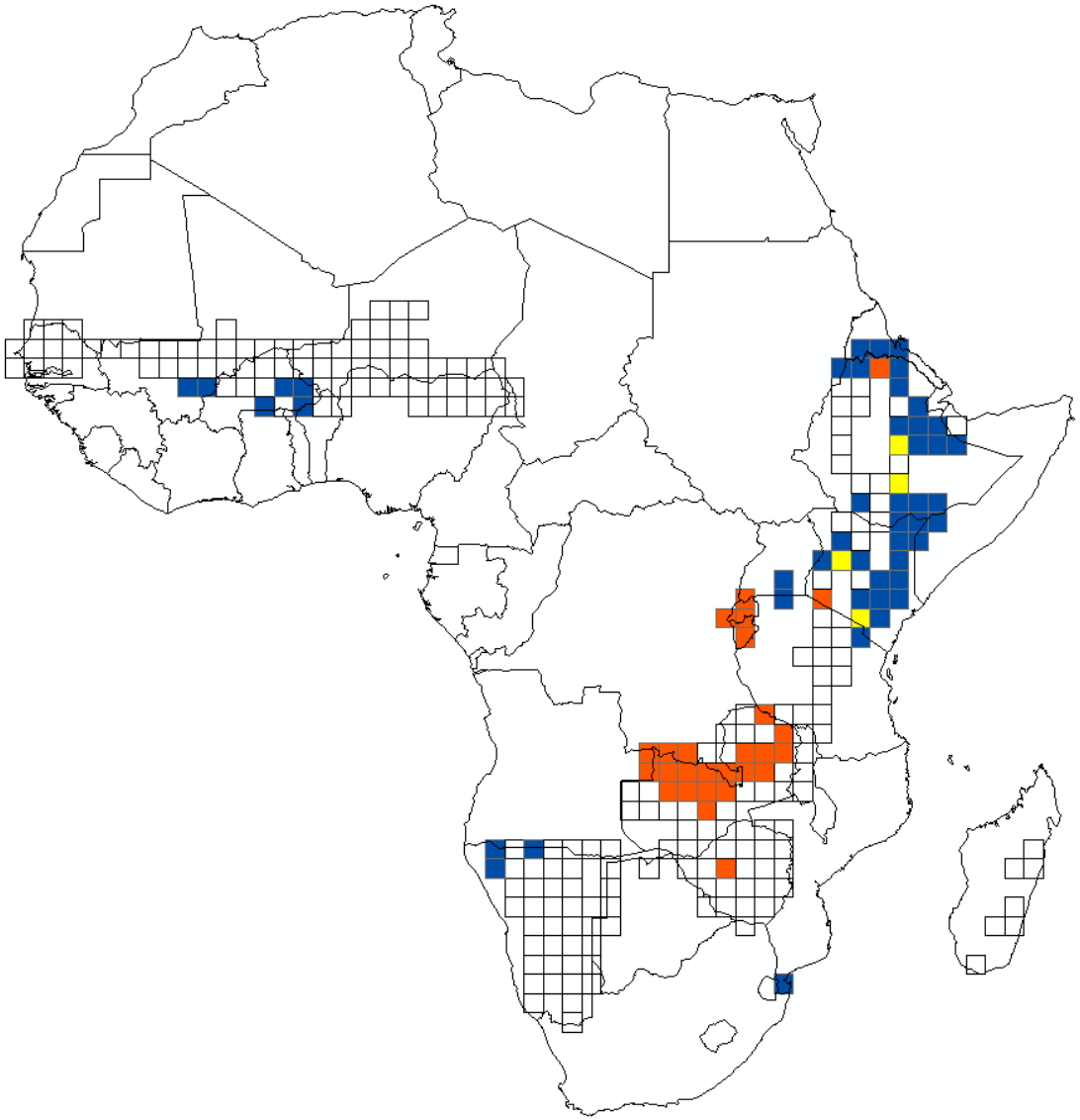
Appendix Figure A6 – Malaria zone shifts, 1974 to 2002

Notes: Colored squares indicate areas in which the malaria zone classification changed and the average number of malaria months per year changed by one month or more between the two 14-year periods before and after 1988, according to the ERA-40 data. Red squares moved from the 2-4 month area to the endemic area; orange from the 0-2 month area to the 2-4 month area; light blue from the 2-4 month area to the 0-2 month area; dark blue from the endemic area to the 2-4 month area.



Appendix Figure A7 – Climate zone shifts, 1974 to 2002

Notes: Colored squares indicate areas in which the climate zone classification changed from arid to rainy areas or vice versa between the two 14-year periods before and after 1988, according to the ERA-40 data. Orange squares moved from the rainy area to the arid area; light blue from the arid area to the rainy area.



Appendix Figure A8 – Causes of epidemic episodes

Notes: Squares indicate epidemic areas in the sample. Orange (blue) cells indicate the areas in which higher temperature (precipitation) in the past 12 months than the 1957-2002 average is predominantly associated with epidemic episodes; yellow cells indicate both higher temperature and precipitation in the past 12 months than the 1957-2002 average is associated with epidemic episodes. See footnote 39 for how exactly this classification is done.