Improving Empirical Estimation of Demographic Drivers:

Fertility, Child Mortality & Malaria Ecology*

Gordon C. McCord[†]

Dalton Conley[‡]

Jeffrey D. Sachs[§]

March 4, 2014

Abstract

Much of Africa has yet to go through a demographic transition; this Malthusian crisis of high mortality, high fertility, rapid population growth and chronic extreme poverty has been attributed to factors including the status of women, pro-natalist policies, and poverty itself. Large uncertainty exists among demographers as to the relative importance of these factors, mostly since econometric estimation is complicated by the endogeneity of fertility to other variables of interest. We attempt to improve estimation of the effect of the child mortality variable on fertility by deploying exogenous variation in the ecology of malaria transmission. Results show that child mortality is a powerfully robust driver of fertility behavior. Meeting the Millennium Development Goal of reducing 1990 child mortality rates by 66% in sub-Saharan Africa would translate into a reduction of total fertility rates from around 6.3 in 1990 to 4.5 (holding other fertility drivers constant), around halfway towards achieving replacement fertility levels of 2.1.

Keywords: fertility, mortality, malaria, demography, human ecology
JEL Codes: J13, I10, O15

[†]gmccord@ucsd.edu, School of International Relations & Pacific Studies, University of California, San Diego; 9500 Gilman Drive #0519, La Jolla, CA 92093; (858) 534-0590; Fax: (858) 534-3939

[‡]conley@nyu.edu, New York University

[§]sachs@columbia.edu, Columbia University and NBER

1 Introduction

The broad categories of determinants of fertility are generally thought to be reasonably well identified by demographers, sociologists and economists, though the detailed quantitative determinants of fertility levels and changes are much less understood. The relationships between fertility, on the one hand, and economic development, the status of women, access to family planning, pro-natalist or pro-planning policies of government, and mortality (both adult and child), on the other hand, have been elegantly theorized and extensively studied. Likewise, many researchers have gone before us in empirically modeling these relationships in both cross-country and within-country analyses, some of which explicitly tackle endogeneity problems in estimation using instrumental variables.[1]

In quantitative terms, however, we still lack a good understanding of why some countries have experienced significant reductions of fertility rates, while those in Africa remain with very high fertility rates—on average difference of almost three births between Sub-Saharan Africa and the rest of the developing world. (See Figure 1, below, for the distribution of total fertility rates in the developing world.) To what extent are Africa's high fertility rates the result of illiteracy, poverty, high child mortality, or lack of access of the poor to contraception? If the region experiences a mortality decline, what would the effect on fertility be? This paper seeks to contribute towards a quantitative assessment of these questions. We acknowledge from the start, however, that many of the potential explanatory variables are only imperfect proxies for the household and community-level drivers of fertility and fertility change. Bongaarts et al. (1984) posit that fertility is regulated directly by proximate determinants (e.g., contraception, age at marriage, abortion), while socioeconomic variables (income, education, mortality) affect fertility only indirectly by modifying the proximate determinants. Thus, we can aim only to get a rough quantitative assessment of the role of key categories of determinants, rather than precise point estimates of how specific policy changes would affect fertility rates.

[FIGURE 1: TOTAL FERTILITY RATES IN 2005]

Most theories of the demographic transition have put great stress, and we believe rightly so, on the causal link from high child mortality to high desired fertility. Simply put, when parents do not know whether their children will survive, they respond by having large families. In a high mortality context, cultural patterns – age of marriage, social norms in childrearing, community support structures – also favor high natality. The original model of the demographic transition, indeed, was driven almost solely by child mortality rates. Exogenous changes to child mortality (e.g., the advent of public health, safe drinking water, immunizations,

---

[1] The most relevant to this paper are attempts to instrument child mortality: Schultz (1997), Benefo and Schultz (1996), Drèze and Murthi (2001), and Kalemli-Ozcan (2006).

improved nutrition) were seen as the primary precursor to reduced fertility rates—albeit with a lag of one or more generations. This putative lag reflected two things according to the standard analysis: first, the lag in perception of households that mortality rates had indeed come down persistently and reliably; and second, the lag in cultural norms surrounding marriage age, birth spacing, family size, and so forth, all needed to promote the transition from high to low fertility. Many studies found, indeed, that the fertility transition is strongly conditioned by a preceding child mortality transition. That said, recent experience suggests that the lag may be waning and that demographic transitions are happening with increased rapidity once "triggered": whereas Western Europe's transitions took over a century (1800-1930 for Britain), more recent large declines in fertility have happened in as little as twenty or fewer years, such as in Bangladesh, Mauritius, and Iran (see Marandi et al. 2006 for a discussion of Iran). Most research attributes these declines to changes in access to family planning (Cleland et al. 1994 in the case of the Matlab experiment in Bangladesh or Aghajanian 1991 in the case of Iran), although some scholars emphasize female education and child mortality (Raftery et al. 1995 in the case of Iran). Others suggest that with the advent of mass media, cultural changes—such as those related to fertility behavior—spread more rapidly than they once did. These temporal (and other) challenges of identification leave open questions as to the relative magnitude of the role of child mortality reduction in the context of 20th century fertility declines.

One issue that continues to plague this basic line of research—and therefore which may be relevant to the African question—is the question of causal directionality between child mortality and fertility choice. As explained in Ronsmans (1996), there is a direct biological effect of high fertility on child survival: short birth intervals can prevent mothers' nutrient repletion in low-income settings, thus compromising the mother's ability to provide nutrients to the fetus during pregnancy. Moreover, several scholars have shown that reduced family size affects human capital investment (Conley and Glauber 2006; Joshi and Schultz 2005; Mogstad and Wiswall 2009) on the micro level as well as economic growth at the macro level (Hazan and Berdugo 2002; Moav 2005). Likewise, the argument can be made that at least some of the powerful correlation of child mortality and fertility represents increased child mortality due to higher fertility because of increased strain on household caloric resources and decreased parental care and supervision with the addition of more children. With these concerns in mind, in this paper we pursue a strategy of deploying exogenous variation in ecological conditions to derive estimates of the causal impact of child mortality on fertility. We then discuss the implications of these findings for the lagging demographic transition in Africa.

2 Mortality and Fertility

Chowdhury (1988) identifies three possible relationships between fertility and mortality: a lagged causal

relationship from mortality to fertility (the theory of demographic transition and choice theory) whether through child "hoarding" (as a precautionary insurance mechanism to guarantee surviving heirs) (Heer and Smith 1968) or direct replacement (for a discussion see, e.g., Cleland 2001); a causal relationship from fertility to mortality (the Ricardian theory); and an interdependent relationship between mortality and fertility (the modern economic theory of population). Note that causal pathway from high fertility to child mortality works through biological and human capital channels, as mentioned in the previous section. Perhaps the most influential recent model of fertility choice among economists is the economic theory of fertility offered by Becker and Barro (1988). Assuming stable wage rates and interest rates, falling child mortality lowers the costs average cost of raising surviving children since a greater proportion of the total investment in childrearing costs realizes a benefit (assuming little or no benefits from non-surviving children). Therefore, the authors argue that fertility rates will initially rise as child mortality declines (and cite evidence to this effect). However, if there is no accompanying change in the parents' interest or wage rates, they argue that there is no cumulative effect. Thus, in the Becker-Barro model, any reduction in fertility resulting from a decline in child mortality would have to work indirectly through wage or interest rates.

Many have attempted to elaborate on the Barro and Becker model theoretically as well as empirically. For example, Doepke (2005) seeks to understand whether "stochastic outcomes and fertility choice are quantitatively important." (p. 337) He differentiates between total fertility rate and net fertility rate, the latter being the average number of children per woman surviving to age five. Doepke concludes that child mortality is causally related to declining fertility rates, but that other factors are responsible for declines in net fertility. With replacement as the mechanism, each family has a target number of children. The death of one child induces the family to replace that child and, as a result, mortality directly affects total fertility rate. However, for mortality to affect the net fertility rate, the hoarding motive would have to take place. Parents would preemptively increase their fertility to protect against potential loss. If this mechanism is present, then a decline in mortality would result in a decline in net fertility. Doepke tests three models. He reports that "all three models are consistent with declining total fertility rates (i.e. number of births) in response to falling mortality. However, we are left without a clear-cut prediction for the relationship of child mortality to net fertility (i.e. the number of survivors)." (p. 344)

In his attempts to test for causality, Chowdhury (1988) finds no consistent results across his thirty-five country sample. Fourteen of his cases support the hypothesis that infant mortality causally impacts fertility, while only two cases support the opposite hypothesis. The remaining cases indicate feedback between the two variables, or the absence of a relationship between fertility and mortality. His results provide stronger support for the hypothesis that mortality effects fertility, but they are notably (and admittedly) inconclusive. Meanwhile, Zakir and Wunnava (1999) found that fertility rates impact mortality rates, and not vice-versa.

Their model employs GLS regression on cross-sectional data and fails to acknowledge the endogeneity of fertility and mortality, leading to a model that is notably mis-specified.

Seeking to better address the endogeneity of child mortality and fertility, Schultz (1997) instruments mortality using calorie availability. However, the validity of his instrument is open to question, as one can imagine a direct, negative causal impact of calorie availability on fertility. Moreover, the instrumented child mortality variable is significant only in cross section and not after including country dummies. In another attempt to deal with endogeneity concerns, Benefo and Schultz (1996) instrument child mortality using variables for community health services and environment, including distance to a market, distance to a clinic, amount of rainfall, and malaria/measles. However, the authors discover that child mortality is only statistically significant if treated as an exogenous variable, but when instrumented by community health services and environment, mortality is not a statistically significant determinant of fertility. Likewise, Drèze and Murthi (2001) instrument mortality using access to safe drinking water, claiming that the latter variable should be unrelated to fertility except through its effect on mortality—again a questionable estimation assumption since safe drinking water is correlated to economic development, which should have an independent effect on fertility. Finally, Kalemli-Ozcan (2006) examines the impact of AIDS mortality on halting the demographic transition in Sub-Saharan Africa by using circumcision prevalence as an instrument for AIDS risk. She finds that the AIDS mortality crisis has indeed led to high fertility rates and less human capital investment in offspring (the effects of HIV on fertility are debated, however, as Fortson 2009 has found very little effect of HIV prevalence on fertility).

It is worth noting that all of these studies find a relatively small elasticity of child mortality to fertility. If we convert their coefficients to percentages using sample means, Benefo and Schultz (1996) find an elasticity of 0.02-0.03, and admit that, "...we estimate that four to fifteen fewer child deaths are associated with a reduction of only one birth. We have no good explanation for the small size of this estimate of the fertility response to child mortality." (p.152) Similarly, Kalemli-Ozcan (2006) finds an elasticity of 0.02 (after converting to percentages), while Drèze and Murthi (2001) find an elasticity of 0.11 in the specification using IV, although the instrument's validity might be open to question. Finally, Schultz (1997) finds an elasticity of .64 (converted to percentages) when instrumenting child mortality with caloric intake, but only achieves significance in cross-section and is thus open to more omitted variable bias.

3 Malaria Ecology

To preview our empirical approach, we argue that the strength of malaria transmission as a function of ecological factors is exogenous to fertility, and use it as an instrument for child mortality. Over our study

period, malaria is the fourth leading cause of death (after neonatal disorders, diarrhea, and pneumonia) for children under five in low income countries (see Black et al. 2003) and is responsible for at least one in every five child deaths in sub-Saharan Africa.[2] Estimates of malaria mortality in Africa range from one million to three million deaths per year. Malaria mortality, in turn, is highly sensitive to ecological conditions, as explained below. Employing malaria ecology as an instrument does not imply, of course, that malaria is the only disease affecting child mortality. However, its strong link to ecology allows us to exploit it in order to remove the endogenous part of child mortality rates and isolate its causal effect on fertility.

Specifically, we deploy an ecological index of malaria transmission (used elsewhere as well; see, e.g., Sachs 2003; Carstensen and Gundlach 2006 for the use of a time-static version, and McCord 2012 for the time-varying version). The index combines ecological factors—rainfall and temperature—with biological ones such as the human biting preference of the mosquito species that serves as the vector for the transmission of malaria (see the Appendix for more details on the construction of the index). It is worth highlighting that the index does not include any information on human population nor on mosquito abundance; both variables affect actual malaria outcomes but are endogenous to fertility and public health efforts and are excluded from the index construction. The 1960-2005 average distribution of this malaria ecology index is mapped in Figure 2, below. While the underlying factors determining malaria transmission may be endogenous to human population movements over the course of thousands of years (through co-evolution with mosquito species), we assert that from the point of view of the current demographic transition in recent decades the biophysical ecology of malaria transmission is exogenous. Moreover, we expand over Conley et al. (2007) by employing the time-varying version of malaria ecology: this allows us to pursue a longitudinal strategy within countries over time, which, in turn allows for a fixed-effects approach to factor out time-invariant country-specific determinants of fertility. The index varies month to month only with changes in temperature and changes in the dominant anopheles vector due to seasonality or varying precipitation. Each anopheles vector has a different human biting preference, which affects the index. More detailed information on the index construction is available in the appendix and in McCord (2012). Note that many parts of the world have stable vectors (breed on brackish or other waters and their life cycles are less sensitive to precipitation) and unstable vectors (depend on a minimum level of precipitation for breeding). This means that there is year-to-year variation in the dominant vector of the grid cell as precipitation varies, and employing country-level fixed effects does not absorb the human biting preference portion of the malaria ecology index in many parts of the world.

---

[2] Since infection with malaria leaves an individual more vulnerable to morbidity and mortality from other infections, malaria is an indirect as well as a direct killer. It is very likely implicated in more than one fifth of all deaths. In some malaria control trials, the reduction of malaria has reduced all-cause under-5 mortality by as much as 40 percent.

[FIGURE 2: 1960-2005 AVERAGE MALARIA ECOLOGY INDEX]

As an added motivation for the adequacy of the malaria ecology instrument, we tested its relationship to national level malaria incidence and mortality data from 1990-2005 as reported by the World Health Organization (Aregawi et al. 2008). The evolution of malaria ecology and malaria incidence (in cases per thousand people) is shown in Figure 3 below for Colombia and Sao Tome and Principe. Note that the lack of monotonicity in the index and lack of country-level trends assuage concerns that later results are due to common trends in fertility, malaria ecology, and an unobserved determinant of fertility. We run country-level regressions of the annual incidence (cases per thousand people) on the malaria ecology for that year, using country dummies, country-specific time trends, and weighing observations by population. The results are in Table 1 below, and show that higher values for the index are associated with higher malaria mortality after including country and period dummies and country-specific time trends.

[FIGURE 3 HERE: MALARIA INCIDENCE & ECOLOGY IN COLOMBIA & SAO TOME & PRINCIPE]

[TABLE 1 HERE]

It is important to note that the malaria ecology index does not include the number of malaria cases or deaths, but only the potential strength of transmission, which we argue should be exogenous to fertility. However, in order for malaria ecology to be a valid instrument, it is important that the instrument affect fertility only through child mortality. A review of the medical literature shows that malaria may have a direct effect on fertility through malaria-related severe anemia, as well as through increased incidence of hypertensive diseases of pregnancy, and spontaneous abortion (Etard et al. 2003, Maubert et al. 1999, Sartelet et al. 1996, Guyatt and Snow 2001).[3] Indeed, Lucas (2013) finds that malaria elimination in Sri Lanka caused women to have their first live birth at a younger age. Since these effects of malaria prevent a live birth, and since TFR counts only live births, then this effect should work in the opposite direction of our putative causal model, thereby biasing any net effect of child mortality on fertility toward zero. In addition, it is worth noting that the reduced-form result in Lucas (2013) leads to a different conclusion from ours; however, it might be the case that earlier first births are consistent with overall total fertility rate reductions if simultaneously women are less likely to have a fifth or sixth birth (Sri Lanka's total fertility in 1960 was 5.5, and probably higher when Lucas' study period begins in 1938). Lucas finds that the effect of malaria elimination was smaller on second births (and not robust to specification checks) than on first births, and does not report effects on higher parities. This leaves open the possibility that the direct effect of malaria on earlier first live birth is not inconsistent with an overall TFR reduction; moreover the direct effect would

---

[3]See also http://www.rbm.who.int/cmc_upload/0/000/015/369/RBMInfosheet_4.htm for overview information on malaria in pregnancy.

7

bias our estimates towards zero. It is also important to caution that malaria in Sri Lanka is seldom fatal (the Plasmodium species is vivax as opposed to falciparum), perhaps explaining part of the difference between our findings and those of Lucas (2013).

Maternal malaria during pregnancy is also associated with low birth weight and increased neonatal and infant mortality—which is in line with our models. That said, there is some evidence that malaria may reduce lactation period (Bates et al. 2004), which might increase fertility through decreased child spacing. Malaria might also increase maternal mortality, however this does not affect the calculation of total fertility rates; TFR is calculated as the average number of children a hypothetical woman would have at the end of her reproductive period if she were subject during her life to the fertility rates of a given period. Finally, Lorentzen et al. (2008) have argued that adult mortality should have an independent effect on fertility (and child human capital investment) by changing the discount rate of mothers and fathers. However, we note that although malaria can kill adults, most malaria mortality occurs among children.

One may be also concerned that temperature and precipitation are components of the index and since they could affect fertility through other channels (such as agricultural production as in Jones and Olken (2010), and thus the return to children's labor on the field). However, that the climatic variables enter the index in a specific nonlinear form emerging from the epidemiological dynamics and so unlikely to be strongly correlated to agriculture or other non-epidemiological systems. Figure 4 below plots the malaria ecology index against temperature and precipitation to illustrate the nonlinearity. Since the human biting rate of the local dominant anopheles vector also goes into the index, the figure shows the value of the function at all levels of precipitation and temperature for a given HBI of 0.5, as well as the actual value of several countries on an average month (these countries are not on the surface because they have HBI different from 0.5). Correlation between the index and agricultural yields is only -.31, moreover the analysis below will demonstrate that the estimation of the child mortality effect is robust to including temperature and precipitation as controls, as well as agricultural yield.

[FIGURE 4: MALARIA ECOLOGY, TEMPERATURE AND PRECIPITATION FOR HBI = 0.5]

4   Data

We compiled a national-level, cross-country dataset covering the period 1960-2005. The demographic data (fertility, infant mortality and child mortality) come from the United Nations Population Division (2006). All other data sources are described below. A note on the time series: the time series data we use in this dataset is divided into quinquennia, beginning in 1960 and ending in 2005. This is determined largely by the fact that the U.N. Population Division, our main source for demographic data, uses five year averages for several

8

of the key demographic variables considered in our analysis, such as TFR and child mortality. Whenever we have used yearly time series data, we compute five year averages for the appropriate quinquennia.[4]

The variables used in the analysis are the following:

- TFR: Total fertility rate, or number of children per woman of reproductive age. Data from the U.N. Population Division reported as 5 year averages.

- Under-5 Mortality Rate: Data from the U.N. Population Division on the mortality rate in children under the age of 5, collected in averages over 5 year periods. Note that we use the natural logarithm of both total fertility and child mortality; the natural limits to a childbearing during a woman's reproductive lifetime suggest a nonlinear relationship which calls for a log-log approach. Figure 5 shows the bivariate relationship between fertility and child mortality with and without logarithms; evidently the log-log form is more appropriate in the linear regression context.

[FIGURES 5A & 5B: TOTAL FERTILITY RATES AND CHILD MORTALITY RATES]

- Index of malaria ecology. Malaria Ecology is an ecologically-based spatial index of the stability of malaria transmission based on the interaction of climate with the dominant properties of anopheline vectors of malaria that determine vectorial capacity (Kiszewski et al. 2004). The index is constructed on a 0.5 degree spatial grid to derive the climatic characteristics of individual months, and then averaged over a 12-month period for every year (McCord 2012). For a complete description of the ME variable see the appendix. Note that the index does not rely on disease incidence or human mortality or morbidity, but instead only on biophysical factors, and therefore provides a variable that is ideally exogenous to human intervention.

- Temperature and Precipitation. The Malaria Ecology variable is constructed using temperature and precipitation data from the University of Delaware described in Matsuura and Willmott (2012). The data is available monthly at the 0.5 degree grid cell level from 1900-2008; we use data from 1960-2005.

- Log of GDP per capita, measure at purchasing-power-parity, in 2000 international dollars. Yearly data are taken from the World Development Indicators database of the World Bank (2012). We calculate our own averages for each 5 year period.

- Average years of females' secondary schooling: Data are from Barro and Lee (2012). We calculate our own averages for each five-year period. We chose this age band to measure educational attainment since it was presumed that post-puberty adolescence and young adulthood were the critical time periods

---

[4]The quinquennia are the following: 1960 through 1964; 1965 through 1969; 1970 through 1974; 1975 through 1979; 1980 through 1984; 1985 through 1989; 1990 through 1994; 1994 through 1999; 2000 through 2004.

9

during which "fertility engines" would ignite (Wu and Martin 2002) and thus the correct population to measure.

- Region: We use the World Bank regional classifications, with a dummy variable for Sub-Saharan African countries. Please see the World Development Indicators (World Bank 2012) for more information regarding how specific countries are classified.

- Population: Total population by country. Five year averages calculated from U.N. Population Division yearly data. For robustness, we re-run regressions using weighted least squares and weigh observations by country population in expectation that variance is likely higher in smaller countries. (Results are unchanged).

- Cereal Yields per Hectare: Kilograms per hectare of harvested land of wheat, rice, maize, barley, oats, rye, millet, sorghum, buckwheat, and mixed grains (World Bank 2012).

Means and standard deviations for the sample of country-years are presented in Table 2, below. We present un-weighted values, as well as values obtained using weights by population size. Weighting by population size diminishes mean TFR in our sample by less than 1 child per woman (4.78 to 3.84), and reduces the mean child mortality rate by around 19 deaths per 1,000 live births.

[TABLE 2 HERE]

5 Methods and Findings

In order to evaluate the effects of child mortality on fertility we employ several statistical models on our panel. We first use reduced form OLS, then OLS with fixed effects, and then test which of those two models is preferred. We then move to an instrumental variable framework (instrumenting for child mortality) using malaria ecology. We test for the endogeneity of the child mortality variable and the strength of the malaria ecology instrument in the first stage. The preferred IV specification includes country and year dummies, as well as a vector X of controls:

$$ln(TotalFertilityRate_{it}) = \beta_0 + \beta_1 ChildMortality_{it} + \Gamma X_{it} + \delta_i + \delta_t + e_{it}$$

$$ChildMortality_{it} = \alpha_0 + \alpha_1 MalariaEcology_{it} + \xi X_{it} + \gamma_i + \gamma_t + \nu_{it}$$

[TABLE 3 HERE]

10

As mentioned above, the vector of controls include average years of secondary school attainment among women; income per capita; temperature, precipitation, and cereal yields per hectare. We note that there are many other variables affecting fertility decisions and outcomes, nevertheless to the extent that we are interested in measuring the causal effect of child mortality we would only worry about omitted variables if they are correlated to each country's (exogenous) interannual variation in the ecology of malaria transmission. We begin the analysis looking simply at regional deviations from a baseline time trend (regression (i) in Table 3). Since we are interested primarily in sub-Saharan Africa, we collapse the other World Bank regions into the suppressed category in order to interpret the difference between sub-Saharan Africa and the rest of the developing world. Note that high-income countries are excluded, since they have completed the demographic transition and intertemporal variation in fertility is likely to be following very different dynamics. From regression (i) we see that sub-Saharan Africa has a TFR that is roughly 67% higher ($e^{.51}$) than the rest of the developing world. Regression (ii) then looks at child mortality, the variable of interest. The regression surely suffers from endogeneity (we have discussed above how high fertility can impact child mortality, and how previous studies have found child mortality to be endogenous after formal tests), so while it is not a perfect model and does not allow us to deduce causality or interpret coefficients, it documents the strength of the correlations between these variables and fertility. Regression (iii) adds other variables which we will use as controls in the analysis (income and women's education). In this simplified framework, high child mortality is associated with higher fertility, increases in women's education are associated with lower fertility rates, and higher incomes are not associated with fertility after controlling for child mortality and female education. Note that there are several observations for every country in the sample, so we estimate Huber-White standard errors robust to clustering within countries in regressions (i) − (iii). Regression (iv) is estimated using fixed-effects. All variables maintain their significance and sign. Note that the sub-Saharan Africa dummy with the controls in these "naïve" models comes down from 0.51 in every case, and as low as 0.08 when child mortality is included. Although this equation is mis-specified, the result motivates a careful examination of child mortality in explaining Africa's high fertility rates. A Hausman test to compare specifications (iii) and (iv) rejects the null that the OLS model is valid, leaving us with the fixed effects specifications as preferred. We tested the robustness of the FE regression (iv) by omitting the income variable, since the purported endogeneity of income might be biasing other coefficients. Both the child mortality and female education variables are unchanged.

[TABLE 4 HERE]

We then move to the instrumental variables framework, where the time-varying malaria ecology variable instruments for child mortality. All regressions include country dummies to absorb time-invariant omitted

variables and all include global period dummies to flexibly control for global trends. Note that following Bertrand et al. (2004), the serial correlation in our dependent variable (fertility) calls for clustering by country in all regressions. Table 4 lists the countries in the sample, and Table 5 presents the results. Regression (i) is the first stage of the basic regression, showing that the exogenous malaria ecology is leading to increases in child mortality. The second stage is (ii), where the instrumented child mortality variable has a coefficient of 0.60. Regression (iv) and its first stage (iii) add the covariates from Table 3 (female education and income); the first stage is qualitatively the same, and the second stage coefficient on child mortality comes down to 0.39. Just as in Table 3, the second stage in (iv) finds female education to be significant and negatively associated to fertility, while income is not. In columns (v) and (vi) we add temperature, precipitation and cereal yields per hectare to show that the malaria ecology instrument and the estimate for child mortality are robust to those controls (the first stage coefficient hardly changes, and the coefficient on child mortality increases to 0.66). Including temperature and precipitation controls strengthens the case that malaria ecology is capturing the nonlinear relationship between climate and disease transmission; moreover, including cereal yields further assuages concerns that the malaria ecology instrument might be affecting fertility through a channel other than child mortality. Columns (vii) and (viii) include all controls at the same time, resulting in a sample of 95 countries. Columns (ix) and (x) then repeat the regressions with no covariates in (i) and (ii) but limiting the sample to the same 95 countries so that results can be compared directly to (vii) and (vii). The coefficients on malaria ecology in the first stage and child mortality in the second stage are remarkably consistent. Finally, regression (xi) shows the reduced form, finding that malaria ecology is directly correlated to fertility, even conditioning on all covariates. Across all these specifications, the point estimates for malaria ecology on child mortality in the first stage and for child mortality in the second stage are quite consistent. Evaluating the lag structure of child mortality's effect on fertility is nontrivial in an instrumental variables framework. We ran regression (ii) and its first stage using the contemporary and one of 5-year, 10-year, 15-year, and 20-year lagged values of child mortality instrumented by the lagged malaria ecology (these results are not reported in the table). In all specifications, the lagged values of child mortality were not significant, and the regression overall became very inefficient (the coefficient on contemporaneous child mortality remained positive though slightly higher than the ones in Table 4, though never significant). As a test for reverse causation from fertility to mortality, we included different leads for child mortality (instrumented) to the regression, and in all cases the lead was not significant.

[TABLE 5 HERE]

[FIGURE 6: FERTILITY & INSTRUMENTED CHILD MORTALITY]

12

Figure 6 above shows the main result graphically. We regress the endogenous variable (child mortality) on the instrument (malaria ecology) and on country and year fixed effects. Then both fertility and the fitted child mortality variable are independently regressed on country and year fixed effects, and finally the residuals are plotted against each other with a flexible polynomial and 95% confidence intervals. What the graph shows is that for the domain where most of the data occur (shown in the histogram), the relationship between fertility and the exogenous movement of child mortality is positive and with tight local error bounds. The graph on the right repeats the exercise including all the covariates in Table 4 (female education, income, temperature, precipitation and cereal yields).

Table 6 limits the sample to low-income countries. The results are qualitatively the same as using both middle- and low-income countries, with low-income countries having the same range of coefficients for child mortality in the second stage (0.44 when including covariates, compared with 0.43 in column (viii) of Table 5). We limit discussion below to results using the entire sample because sub-Saharan Africa's demographic transition will likely go hand-in-hand with rising incomes, and looking at the average estimated elasticity across low and middle income countries seems warranted.

[TABLE 6: LOW-INCOME COUNTRY SAMPLE]

We proceed to run some tests on the malaria ecology instrument. First we test the endogeneity of the child mortality variable (the null of exogeneity can be rejected at a one percent alpha level) and for the strength of the instrument (the F-tests for the instrument in the first stage are reported in Table 5; all are above the Staiger and Stock (1997) rule of thumb value of F=10, so we can conclude that the instrument is strong). To further dispel concerns of a weak instrument, we follow Stock and Yogo (2002) and compute the Cragg-Donald statistic, which tests underidentification by examining the smaller eigenvalue of the F-statistic matrix of the first stage regression. In all cases, the statistic exceeds the critical value of 16.38, suggesting that bias and significance test distortions due to a weak instrument are not a concern here. Next, we run all specifications with a limited information maximum likelihood estimator (Fuller 1977) with a modification parameter of $\alpha = 1$, which is robust than 2SLS with a weak instrument (Hahn, Hausman and Kuersteiner 2004, Hausman et al. 2005). In all cases, the coefficient child mortality remained significant and of the same magnitude (within 0.01 of the 2SLS estimates).

All regressions were re-estimated with population weights; results were qualitatively unchanged, though the coefficient on mortality was larger (0.82 and significant when including all covariates; as compared to 0.43 in Table 5). Another robustness test was to add a dummy for China after 1980, since its fertility rate was to some extent artificially lowered by the one-child policy put in place in 1979 (this would only be econometrically relevant in the population-weighted regressions, given China's large weight). Adding the

dummy did not affect results in either the weighted or unweighted regression. Finally, we dispense with clustering and analytical weights in order to estimate the basic model in (i) and (ii) using first differences instead of fixed effects. The results are qualitatively similar: the child mortality coefficient drops from 0.60 under FE to 0.49 under FD when no covariates are used, and from 0.43 to 0.2 when covariates are included. The coefficient on the serial correlation of errors in FD is 0.46, which does not strongly warrant one model above the other. We opt to report FE results throughout so that clustering of standard errors can be employed (as well as population weights in the robustness check).

The effect of child mortality on fertility is overwhelmingly robust across specifications. The income and education variables are less robust: after child mortality is instrumented, they are only significant in the low-income sample of Table 6. We do not focus on interpreting their coefficients due to the potential endogeneity problems (fertility might affect these control variables), but they serve to give a range of plausible estimates on the child mortality variable given potential omitted variable bias. The coefficients on child mortality have ranged between 0.18 in the non-IV fixed-effects specification to 0.66 in the IV fixed-effects specification (0.44 for low-income countries).

The preferred specifications are the IV regressions with country dummies and with all covariates, which produces a coefficeint of 0.43 (0.44 for low-income countries). This estimate corresponds to having a decrease of TFR of 28% (or a decrease of 1.8 births from the 1990 level of 6.3) if child mortality were to decrease by 66% from its 1990 value in sub-Saharan Africa (the Millennium Development Goal for child mortality). This points to the fact that child mortality still plays a powerful role in fertility choice in the 20th century, an order of magnitude larger than the effect identified in some other studies mentioned previously.

Before concluding, it is worth pausing to discuss the generalizability of the child mortality coefficient estimated through IV. Some authors have expressed concern with interpreting coefficients from IV too generally: the coefficient from IV only is the local average treatment effect for the sub-population for which the instrument is well-correlated to the endogenous variable. In order to make sure that we can draw policy implications for sub-Saharan Africa, we check the first stage regression of under-5 mortality on malaria ecology using only countries that have nonzero malaria cases over the period. We find that the coefficient on malaria ecology is 0.39 and significant, indicating this subgroup is a "complier" in the sense that the sub-sample exhibits a strong first stage. In the second stage, the estimated coefficient on child mortality is 0.39 with a standard error of 0.8, which means that the estimate above of 0.43 is quite close. Given that malarious countries tend to be poorer and have higher fertility rates, we prefer to measure the average elasticity of child mortality across all developing countries and not just the poorest countries that are in the earlier stages of the demographic transition.

A second concern is that our child mortality coefficient might only be valid for children dying due to malaria. However, there is nothing in the demography literature or theory that identifies a differentiated fertility response to a child death depending on the cause of death (though one might imagine different fertility responses between health-related deaths and conflict-related deaths, for example). Nonetheless, whether there is a differentiated fertility response depending on cause of child death is beyond the scope of this paper, but in the absence of such evidence we maintain that the child mortality coefficient that we have estimated is a step forward in the quantitative study of fertility dynamics.

5.1 Model Limitations

In this paper, we have deployed a novel instrument that generates exogenous variation in child mortality to study its effect on fertility rates. The results strongly suggest that child mortality remains a quantitatively important driver of fertility rates; nevertheless two limitations of this study should be pointed out. The first, which is mentioned above, is that variation in malaria ecology might affect adult mortality and morbidity, and that these in turn may affect fertility rates, thus violating the exclusion restriction. We argue above that adult mortality from malaria is far less than child mortality, and that adult mortality would change parental discount rates and might lead them to reduce fertility. This would bias our estimate towards zero, but any other effects would be confounding our estimate. Meanwhile, malaria's effects on adult morbidity might raise or lower fertility (on the one hand, morbidity might lower fertility due to miscarriages, but on the other hand desired fertility might rise as the value of child labor on the farm rises when parents can't work).

Another concern is that the variables being studied (fertility, child mortality, and malaria ecology) might have underlying time trends at the country level generating spurious correlations even after country and year fixed effects. Given the slow-moving nature of the TFR variable (it is a national variable smoothed into 5-year quinquennia), and that the panel has only 8 time periods, country-specific time trends demand too much of these data. While the results we present include country and year fixed effects, our results are not robust to including country-specific linear trends (adding country-specific linear trrends to Table 5 column (viii) gives a coefficient on the second stage of 0.24). This demonstrates some degree of consistency with our result of 0.44, but the coefficient not statistically significant when including country-specific linear trends.

6 Conclusions

We are only the latest in a long research tradition to test the link between child mortality and fertility. We add to this literature through the deployment of a novel instrument: malaria ecology. Instrumenting

child mortality in this way yields a result that appears robust across a number of models that include or exclude various control variables, time and country fixed effects and with or without population weights. In all our models, we find that child survival is a quantitatively important and robust driver of fertility. This is where the theory of the demographic transition started: save children and families will choose to have fewer children. The finding is consistent with the fact that the demographic transition has proceeded in the widest range of social settings: rural and urban, male-dominated and gender equal, impoverished and middle-class (Cleland 2001). Since the child mortality transition has also proceeded in a wide range of settings, it is plausible that fertility has been driven largely by changes child mortality.

This is not to say, however, that child mortality is the only important driver of a demographic transition. As explained in Bongaarts et al. (1984), family planning through contraception, sexual behavior and abortion, are proximate determinants of fertility which are affected by socioeconomic variables such as income, education, and health. If socioeconomic and policy variables are to successfully trigger a transition, then the change in desired fertility must be allowed to translate into a change of actual fertility through the proximate variables (especially contraception use). Though we have focused on better quantifying the role of reducing child mortality, the importance of other variables affecting fertility such as female education or access to family planning should not be understated.

If this conclusion is correct, it heralds the possibility of a rapid fertility transition in Africa. Child survival can be dramatically improved in a short period of time (United Nations Millennium Project 2005). It is indeed possible, even in a very low income setting and within a five-year period, to reduce the child mortality rate from 150 per 1,000 or above, to well below 100 per 1,000. Similarly, other determinants of fertility, such as increases in agricultural yields and public policies in support of family planning, may change in just a few years, in contrast with many socioeconomic variables. Sub-Saharan Africa's TFR in 1990 was 6.3, and in 2011 it was 5.2 (World Bank 2012). If the Millennium Development Goals are achieved and child mortality is decreased by two-thirds compared to 1990, our results would predict a decrease in TFR of around 2 babies, almost half of the reduction necessary to reach replacement fertility of 2.1. If in addition female education is increased and access to family planning is improved, the resulting fertility reductions would correspond to a new TFR in sub-Saharan Africa of around 2.1. While these predictions are very crude, nevertheless the prospect for a rapid, policy-supported transition to lower fertility in Africa looks reasonable. The results also point to the continued importance of child mortality in fertility declines: while other variables emphasized in research and policy (such as family planning or female education) are important contributors to decline in fertility, child mortality declines seem to account for around half of the fertility decline and its importance in demographic dynamics should not be ignored.

More research is needed to continue exploring the causes of rapid fertility transitions seen in some countries

in the last few decades. In particular, our models do not attempt to solve the endogeneity issues with the control variables, nor attempt to understand the lag structure in changes to the independent variables (though, as mentioned above, we tested for lagged effects of changes in child mortality on fertility). Future work could look intensively at case studies of rapid fertility declines and attempt to better model lagged effects of the determinants of TFR. In the meantime, however, this paper provides a more reliable range of estimates for how changes in child mortality causally translate into changes in fertility behavior. To our knowledge, no paper has employed a time-varying exogenous instrument to solve the endogeneity problem between fertility and child mortality. The results are also encouraging: whereas previous estimates of the elasticity of fertility to child mortality using IV have failed to find statistical significance or have found perplexingly small magnitudes, our result finds that child mortality is likely to account for almost half of the fertility reduction during the demographic transition.

References

Aghajanian, Akbar, "Population Change In Iran, 1966-86: a stalled demographic transition?," *Population and Development Review*, 1991, pp. 703–715.

Aregawi, Maru, Richard Cibulskis, Mac Otten, Ryan Williams, Christopher Dye et al., *World Malaria Report, 2008.*, World Health Organization, 2008.

Barro, Robert J and Jong-Wha Lee, "A new data set of educational attainment in the world, 1950–2010," *Journal of Development Economics*, 2012.

Bates, Imelda, Caroline Fenton, Janet Gruber, David Lalloo, Antonieta Medina Lara, S Bertel Squire, Sally Theobald, Rachael Thomson, and Rachel Tolhurst, "Vulnerability to malaria, tuberculosis, and HIV/AIDS infection and disease. Part 1: determinants operating at individual and household level," *The Lancet infectious diseases*, 2004, *4* (5), 267–277.

Becker, Gary S and Robert J Barro, "A reformulation of the economic theory of fertility," *The Quarterly Journal of Economics*, 1988, *103* (1), 1–25.

Benefo, Kofi and T Paul Schultz, "Fertility and child mortality in Côte d'Ivoire and Ghana," *The World Bank Economic Review*, 1996, *10* (1), 123–158.

Bertrand, Marianne, Esther Duflo, and Sendhil Mullainathan, "How much should we trust differences-in-differences estimates?," *The Quarterly Journal of Economics*, 2004, *119* (1), 249–275.

Black, Robert E, Saul S Morris, and Jennifer Bryce, "Where and why are 10 million children dying every year?," *The Lancet*, 2003, *361* (9376), 2226–2234.

Bongaarts, John, Odile Frank, and Ron Lesthaeghe, "The proximate determinants of fertility in sub-Saharan Africa," *Population and Development Review*, 1984, pp. 511–537.

Carstensen, Kai and Erich Gundlach, "The primacy of institutions reconsidered: Direct income effects of malaria prevalence," *The World Bank Economic Review*, 2006, *20* (3), 309–339.

Chowdhury, Abdur R, "The infant mortality-fertility debate: some international evidence," *Southern Economic Journal*, 1988, pp. 666–674.

Cleland, John, "The effects of improved survival on fertility: A reassessment," *Population and Development Review*, 2001, *27*, 60–92.

_ , **James F Phillips, Sajeda Amin, Golam M Kamal et al.**, *The determinants of reproductive change in Bangladesh: success in a challenging environment.*, World Bank, 1994.

Conley, Dalton and Rebecca Glauber, "Parental educational investment and children′s academic risk estimates of the impact of sibship size and birth order from exogenous variation in fertility," *Journal of Human Resources*, 2006, *41* (4), 722–737.

_ , **Gordon C McCord, and Jeffrey D Sachs**, "Africa's lagging demographic transition: Evidence from exogenous impacts of malaria ecology and agricultural technology," Technical Report, National Bureau of Economic Research 2007.

Doepke, Matthias, "Child mortality and fertility decline: Does the Barro-Becker model fit the facts?," *Journal of population Economics*, 2005, *18* (2), 337–366.

Drèze, Jean and Mamta Murthi, "Fertility, education, and development: evidence from India," *Population and Development Review*, 2001, *27* (1), 33–63.

Etard, Jean-François, Belco Kodio, and Carine Ronsmans, "Seasonal variation in direct obstetric mortality in rural Senegal: role of malaria?," *The American journal of tropical medicine and hygiene*, 2003, *68* (4), 503–504.

Fortson, Jane G, "HIV/AIDS and fertility," *American Economic Journal: Applied Economics*, 2009, pp. 170–194.

Fuller, Wayne A, "Some properties of a modification of the limited information estimator," *Econometrica: Journal of the Econometric Society*, 1977, pp. 939–953.

Guyatt, Helen L and Robert W Snow, "Malaria in pregnancy as an indirect cause of infant mortality in sub-Saharan Africa," *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 2001, *95* (6), 569–576.

Hahn, Jinyong, Jerry Hausman, and Guido Kuersteiner, "Estimation with weak instruments: Accuracy of higher-order bias and MSE approximations," *The Econometrics Journal*, 2004, *7* (1), 272–306.

Hausman, Jerry, James H Stock, and Motohiro Yogo, "Asymptotic properties of the Hahn–Hausman test for weak-instruments," *Economics Letters*, 2005, *89* (3), 333–342.

Hazan, Moshe and Binyamin Berdugo, "Child Labour, Fertility, and Economic Growth*," *The Economic Journal*, 2002, *112* (482), 810–828.

Heer, David M and Dean O Smith, "Mortality level, desired family size, and population increase," *Demography*, 1968, *5* (1), 104–121.

Jones, Benjamin F and Benjamin A Olken, "Climate Shocks and Exports," *The American Economic Review: Papers and Proceedings*, 2010, *100*, 454–459.

Joshi, Shareen and T Paul Schultz, "Family planning as an investment in female human capital: Evaluating the long term consequences in Matlab, Bangladesh," *Yale University. New Haven, CT. Processed*, 2005.

Kalemli-Ozcan, Sebnem, "AIDS," Reversal" of the Demographic Transition and Economic Development: Evidence from Africa," Technical Report, National Bureau of Economic Research 2006.

Kiszewski, Anthony, Andrew Mellinger, Andrew Spielman, Pia Malaney, Sonia Ehrlich Sachs, and Jeffrey Sachs, "A global index representing the stability of malaria transmission," *American Journal of Tropical Medicine and Hygiene*, 2004, *70* (5), 486–498.

Lorentzen, Peter, John McMillan, and Romain Wacziarg, "Death and development," *Journal of Economic Growth*, 2008, *13* (2), 81–124.

Lucas, Adrienne M, "The Impact of Malaria Eradication on Fertility," *Economic Development and Cultural Change*, 2013, *65* (3), 607–631.

Marandi, SA, A Mehryar, MJ Abbasi-Shavazi, Z Majdfar, and MM Shamsian, "Iran. From family sizes of six to replacement levels in 15 years.," 2006.

Matsuura, Kenji and Cort J Willmott, "Terrestrial air temperature: 1900–2012 Gridded Monthly Time Series," *Center for Climatic Research, Dep. Of Geography, University of Delaware, Newark.*, 2012.

Maubert, Bertrand, Nadine Fievet, Germaine Tami, Michel Cot, Christian Boudin, and Philippe Deloron, "Development of antibodies against chondroitin sulfate A-adherent Plasmodium falciparum in pregnant women," *Infection and immunity*, 1999, *67* (10), 5367–5371.

McCord, Gordon Carlos, "Essays on Malaria, Environment and Society." PhD dissertation, Columbia University 2012.

Moav, Omer, "Cheap Children and the Persistence of Poverty," *The Economic Journal*, 2005, *115* (500), 88–110.

Mogstad, Magne and M Wiswall, "Family Size and Children's Education: How Linear Models Can Mask a Non-Linear Relationship," *Work in progress*, 2009.

Raftery, Adrian E, Steven M Lewis, and Akbar Aghajanian, "Demand or ideation? Evidence from the Iranian marital fertility decline," *Demography*, 1995, *32* (2), 159–182.

Ronsmans, Carine, "Birth spacing and child survival in rural Senegal," *International Journal of Epidemiology*, 1996, *25* (5), 989–997.

Sachs, Jeffrey D, "Institutions don't rule: direct effects of geography on per capita income," *National Bureau of Economic Research Working Paper*, 2003.

Sartelet, Hervé, Christophe Rogier, Isabelle Milko-Sartelet, Guy Angel, and Gabriel Michel, "Malaria associated pre-eclampsia in Senegal," *The Lancet*, 1996, *347* (9008), 1121.

Schultz, T Paul, "Demand for children in low income countries," *Handbook of population and family economics*, 1997, *1*, 349–430.

Staiger, Douglas and James H Stock, "Instrumental Variables with Weak Instruments," *Econometrica: Journal of the Econometric Society*, 1997, *65* (3), 557–586.

Stock, James H and Motohiro Yogo, "Testing for weak instruments in linear IV regression," 2002.

United Nations Millennium Project, *Investing in development: a practical plan to achieve the Millennium Development Goals*, Earthscan, 2005.

United Nations Population Division, *World Population Prospects: The 2004 Revision: Volume I: comprehensive tables*, United Nations Publications, 2006.

World Bank, *World Development Indicators 2012*, World Bank, 2012.

Wu, Lawrence L and Steven P Martin, *Is There an Engine of Nonmarital Fertility?*, Center for Demography and Ecology, University of Madison–Wisconsin, 2002.

Zakir, Mohammed and Phanindra V Wunnava, "Factors affecting infant mortality rates: evidence from cross–sectional data," *Applied Economics Letters*, 1999, *6* (5), 271–273.

Tables and Figures

Table 1: Malaria Ecology and Malaria Mortality

	(i) ln(Malaria deaths per 1,000 population)	(ii) ln(Malaria deaths per 1,000 population)
Dependent Variable:		
Independent Variables		
Malaria Ecology	0.55**	0.36**
	(3.13)	(3.94)
Country Fixed Effects	Y	Y
Year Fixed Effects	Y	Y
Country-Specific Linear Trend		Y
N =	654	654
Countries =	84	84
Years =	1990-2007	1990-2007
Within R-squared =	0.19	0.64

t-statistic in parentheses, ** indicates significant to 95% confidence.

Regressions include a constant (not reported)

Regressions report robust standard errors clustered by country

Regressions weigh observations by population

Table 2: Means and Standard Deviations of Variables in Sample

Variable	Mean		Standard Deviation	
	Un-weighted	Population weighted	Un-weighted	Population weighted
Total Fertility Rate	4.78	3.84	1.93	1.70
Under 5 mortality rate (per 1,000)	112.4	93.7	80.2	63.0
Malaria Ecology Index	2.80	1.42	3.89	2.92
Average Temperature (°C)	19.9	17.08	7.54	9.2
Average Precipitation (mm)	1159.4	1026.1	796.6	642.4
GDP per capita, PPP, constant $	4,064	2,942	3,741	2,984
Females' Years of Secondary Schooling	1.10	1.01	1.10	0.84
Average Population (millions)	30.3	475	116	472
Cereal Yields per Hectare (kg/ha)	1,694	2,459	1,057	1,293
Year	1986.2	1988.9	12.5	12.0

Table 3: OLS and Fixed-Effects Regression of Total Fertility Rates

	(i)	(ii)	(iii)	(iv)
Dependent Variable:	ln(TFR)	ln(TFR)	ln(TFR)	ln(TFR)
Independent Variables				
Sub-Saharan Africa dummy	0.51***	0.08**	0.08*	
	(10.39)	(2.44)	(1.66)	
ln(under-5 Mortality Rate)		0.43***	0.38***	0.18***
		(18.49)	(9.04)	(4.28)
Females' Years of Secondary Schooling			-0.08***	-0.10***
			(-3.00)	(-2.70)
ln(GDP per capita, in PPP prices & constant $)			0.02	-0.01
			(0.70)	(-0.37)
N =	1258	1145	536	536
Countries =	143	143	96	96
R-squared =	0.43	0.77	0.81	0.83

t-statistics in parentheses; *** indicates significant to 99% confidence, ** to 95%, and * to 90%.

All regressions include year dummies and a constant, which are not reported.

All regressions cluster standard errors by country

Regression (iv) includes country dummies and reports the within R-squared

All regressions exclude high-income countries

Table 4: Sample of 133 Countries

Afghanistan	Gabon*	Nigeria
Albania*	The Gambia*^	Oman
Algeria*	Georgia	Pakistan*^
Angola	Ghana*^	Panama*
Argentina*	Guatemala*	Papua New Guinea*^
Armenia*	Guinea	Paraguay*
Azerbaijan	Guinea-Bissau	Peru*
Bangladesh*^	Guyana*	Philippines*
Belarus	Haiti*^	Poland*
Belize*	Honduras*	Russian Federation*
Benin*^	Hungary*	Rwanda*^
Bhutan	India*^	Sao Tome and Principe
Bolivia*	Indonesia*	Senegal*^
Bosnia and Herzegovina	Iran, Islamic Rep.*	Sierra Leone*^
Botswana*	Iraq	Slovak Republic*
Brazil*	Jamaica*	Solomon Islands
Bulgaria*	Jordan*	Somalia
Burkina Faso	Kazakhstan*	South Africa*
Burundi*^	Kenya*^	Sri Lanka*
Cambodia*^	Korea, Dem. Rep.	St. Vincent and the Grenadines
Cameroon*^	Kyrgyz Republic*^	Sudan*^
Cape Verde	Lao PDR*^	Suriname
Central African Republic*^	Latvia*	Swaziland*
Chad	Lebanon	Syrian Arab Republic*
Chile*	Lesotho*^	Tajikistan*^
China*	Liberia*^	Tanzania*^
Colombia*	Libya*	Thailand*
Comoros	Lithuania*	Togo*^
Congo, Dem. Rep.	Macedonia, FYR	Trinidad and Tobago*
Congo, Rep.*^	Madagascar	Tunisia*
Costa Rica*	Malawi*^	Turkey*
Cote d'Ivoire*^	Malaysia*	Turkmenistan
Croatia*	Mali*^	Uganda*^
Cuba	Mauritania*^	Ukraine*
Czech Republic*	Mauritius*	Uruguay*
Djibouti	Mexico*	Uzbekistan
Dominican Republic	Moldova	Vanuatu
Ecuador*	Mongolia*^	Venezuela, RB*
Egypt, Arab. Rep.*	Morocco*	Vietnam*^
El Salvador*	Mozambique*^	West Bank and Gaza
Equatorial Guinea	Myanmar*^	Yemen, Rep.*^
Eritrea	Namibia*	Zambia*^
Estonia*	Nepal*^	Zimbabwe
Ethiopia	Nicaragua*^	
Fiji*	Niger*^	

* Indicates country is in the 95-country sample in Table 5

^ Indicates country is in the 39-country Low Income sample in Table 6

25

Table 5: Instrumenting Child Mortality using Malaria Ecology

Dependent Variable: Independent Variables	(i) ln(u5MR)	(ii) ln(TFR)	(iii) ln(u5MR)	(iv) ln(TFR)	(v) ln(u5MR)	(vi) ln(TFR)	(vii) ln(u5MR)	(viii) ln(TFR)	(ix) ln(u5MR)	(x) ln(TFR)	(xi) ln(TFR)
Malaria Ecology	0.32*** (3.61)		0.39*** (5.91)		0.32*** (3.38)		0.41*** (5.92)		0.45*** (6.76)		0.17*** (5.50)
ln(under-5 Mortality Rate)		0.60*** (6.14)		0.39*** (4.80)		0.66*** (5.44)		0.43*** (4.80)		0.42*** (5.05)	
Females' Years of Secondary Schooling			-0.04 (-0.59)	-0.08* (-1.76)			-0.04 (-0.56)	-0.07 (-1.41)			-0.08* (-1.94)
ln(GDP per capita, in PPP prices & constant $)			-0.16** (1.96)	0.04 (0.68)			-0.11 (-1.14)	0.01 (0.27)			-0.03 (-0.85)
Average temperature over period					-0.11* (-1.92)	-0.02 (-0.56)	-0.07 (-1.31)	-0.05* (-1.87)			-0.08*** (-2.87)
Average precipitation over period					6.00E-06 (0.07)	0.00002 (0.36)	7.86E-06 (0.07)	0.00007 (1.10)			0.00007 (1.28)
ln(Cereal Yield per Hectare)					-0.33*** (-4.18)	0.07 (1.09)	-0.27*** (-3.06)	0.06 (0.88)			-0.05 (-0.91)
N =	1095		535		984		517		517		517
Countries =	133		95		132		95		95		95
F-test on instrument =	13.08		34.9		11.4		35.02		45.64		
Cragg-Donald Statistic =	107.23		80.22		93.51		80.79		109.94		
Within R-squared =	0.76	0.72	0.75	0.79	0.77	0.69	0.77	0.78	0.74	0.77	0.97

t-statistics in parentheses; *** indicates significant to 99% confidence, ** to 95%, and * to 90%.

All regressions include country dummies, time period dummies and a constant (not reported).

Regressions (i), (iii), (v), (vii), and (ix) are first stage for (ii), (iv), (vi), (viii), and (x) respectively. (xi) is reduced form.

All regressions exclude high-income countries.

All regressions cluster standard errors by country

26

Table 6: Low-Income Country Sample

	(i)	(ii)	(iii)	(iv)	(i)
Dependent Variable:	ln(u5MR)	ln(TFR)	ln(u5MR)	ln(TFR)	ln(TFR)
Independent Variables					
Malaria Ecology	0.25***		0.27***		0.12***
	(3.04)		(3.03)		(3.18)
ln(under-5 Mortality Rate)		0.56***		0.44***	
		(2.89)		(3.29)	
Females' Years of Secondary Schooling			-0.11	-0.18**	-0.23**
			(-0.95)	(-2.36)	(-2.52)
ln(GDP per capita, in PPP prices & constant $)			-0.03	-0.09**	-0.10**
			(-0.31)	(-2.16)	(-2.45)
Average temperature over period			-0.1	-0.02	-0.07*
			(-1.55)	(-0.66)	(-1.66)
Average precipitation over period			-8.00E-06	-0.00009	-0.00009
			(-0.04)	(-0.89)	(-0.88)
ln(Cereal Yield per Hectare)			-0.11	0.04	-0.007
			(-1.20)	(0.69)	(-0.12)
N =	217		217		217
Countries =	39		39		39
F-test on instrument =	9.26		9.16		
Cragg-Donald Statistic =	36.84		35.61		
Within R-squared =	0.63	0.72	0.66	0.79	-0.83

t-statistics in parentheses; *** indicates significant to 99% confidence, ** to 95%, and * to 90%.

All regressions include country dummies, time period dummies and a constant (not reported).

Regressions (i) and (iii) are first stage for (ii) and (iv), respectively. (v) is reduced form.

All regressions cluster standard errors by country

All regressions include only low-income countries
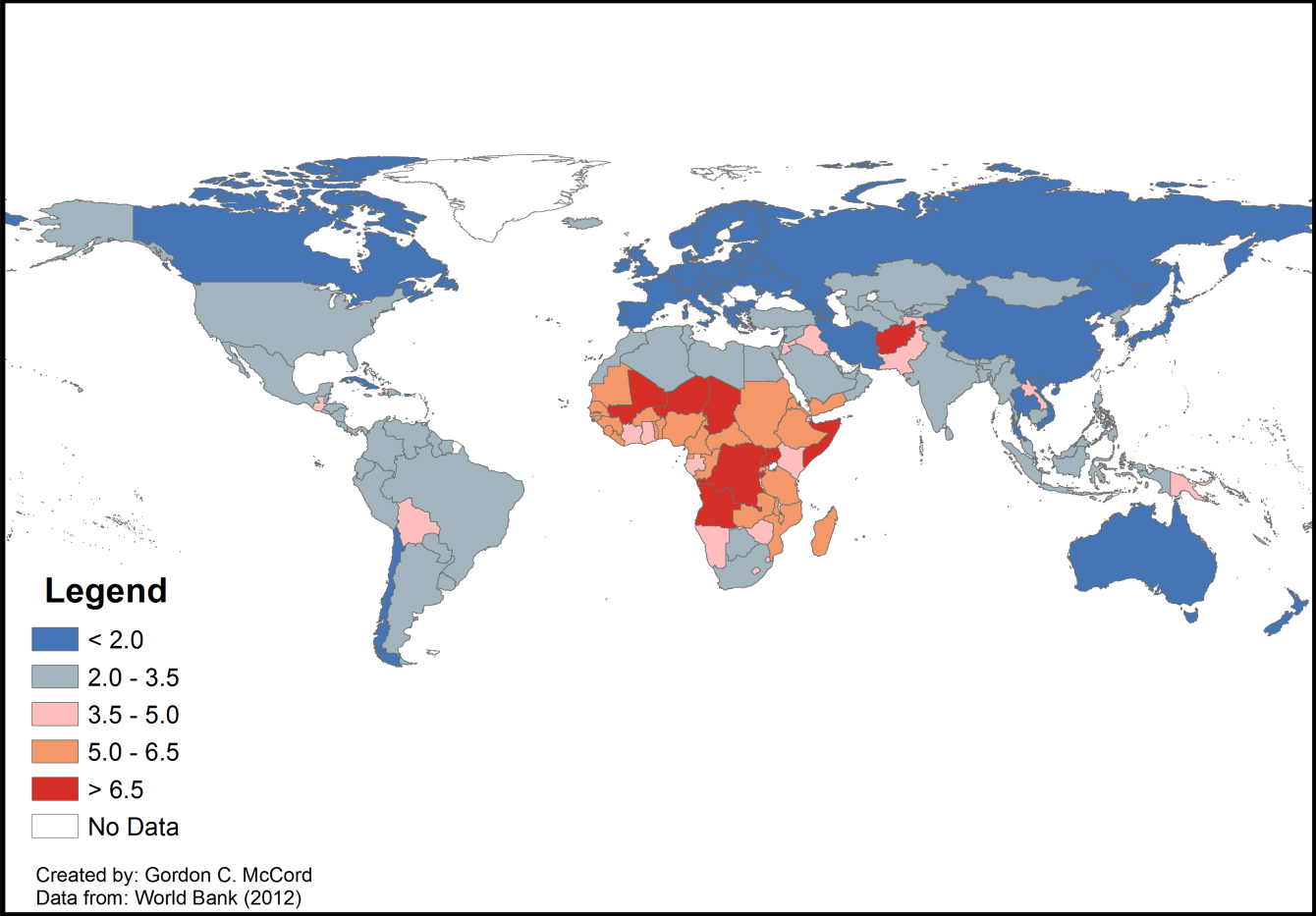
Figure 1: Total Fertility Rates in 2005

Legend

- < 2.0
- 2.0 - 3.5
- 3.5 - 5.0
- 5.0 - 6.5
- \> 6.5
- No Data

Created by: Gordon C. McCord
Data from: World Bank (2012)

Figure 2: 1960-2005 Average Malaria Ecology Index

Legend

0	2.5 - 4
< 0.025	4 - 6
0.025 - 0.5	6 - 9
0.5 - 1	9 - 13
1 - 2.5	> 13

Created by: Gordon C. McCord
Source: McCord (2012)
Note: Index uses University of Delaware temperature &
precipitation data described in Matsuura and Willmott (2012)
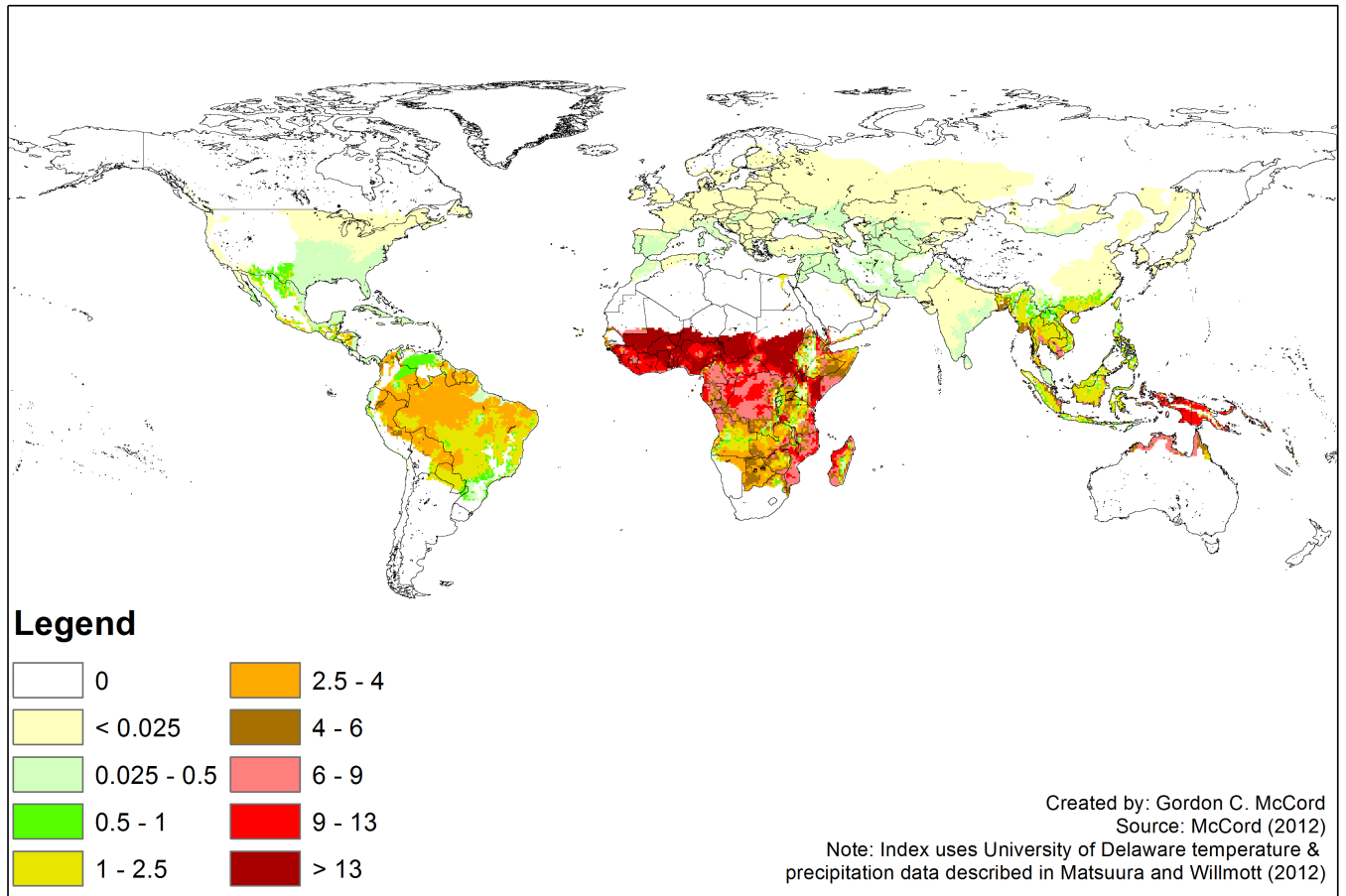
Figure 3: Malaria Incidence & Ecology in Colombia & Sao Tome & Principe
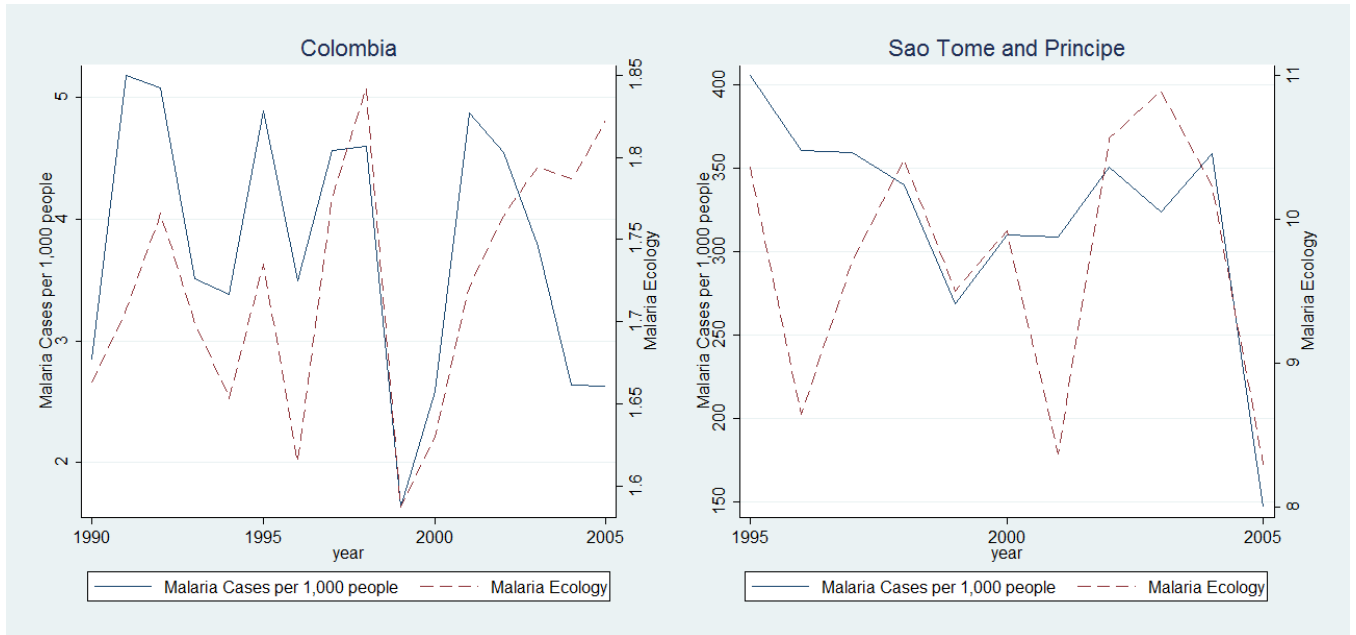
Figure 4: Malaria Ecology, Temperature and Precipitation for HBI = 0.5

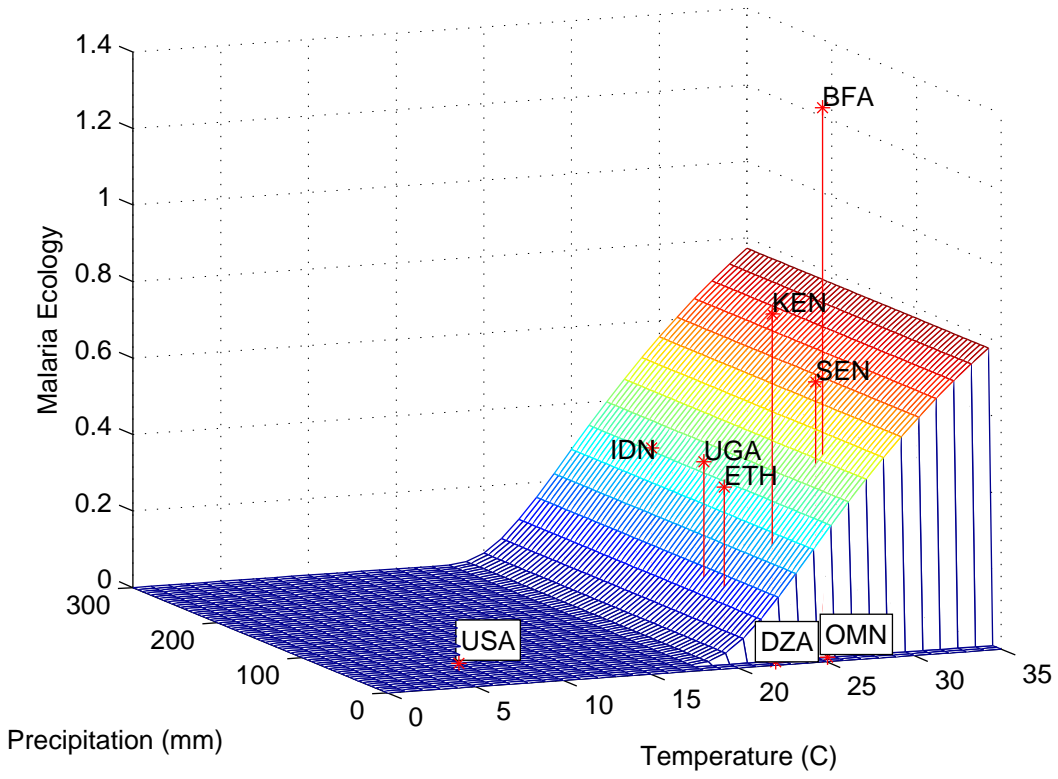**Figure 5: Total Fertility Rates and Child Mortality Rates**

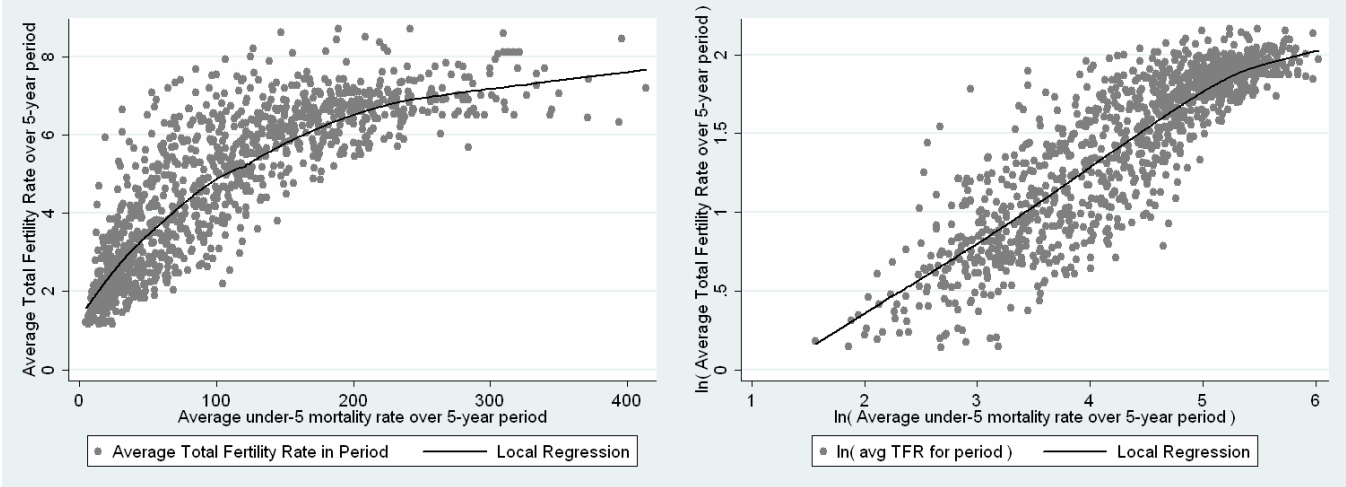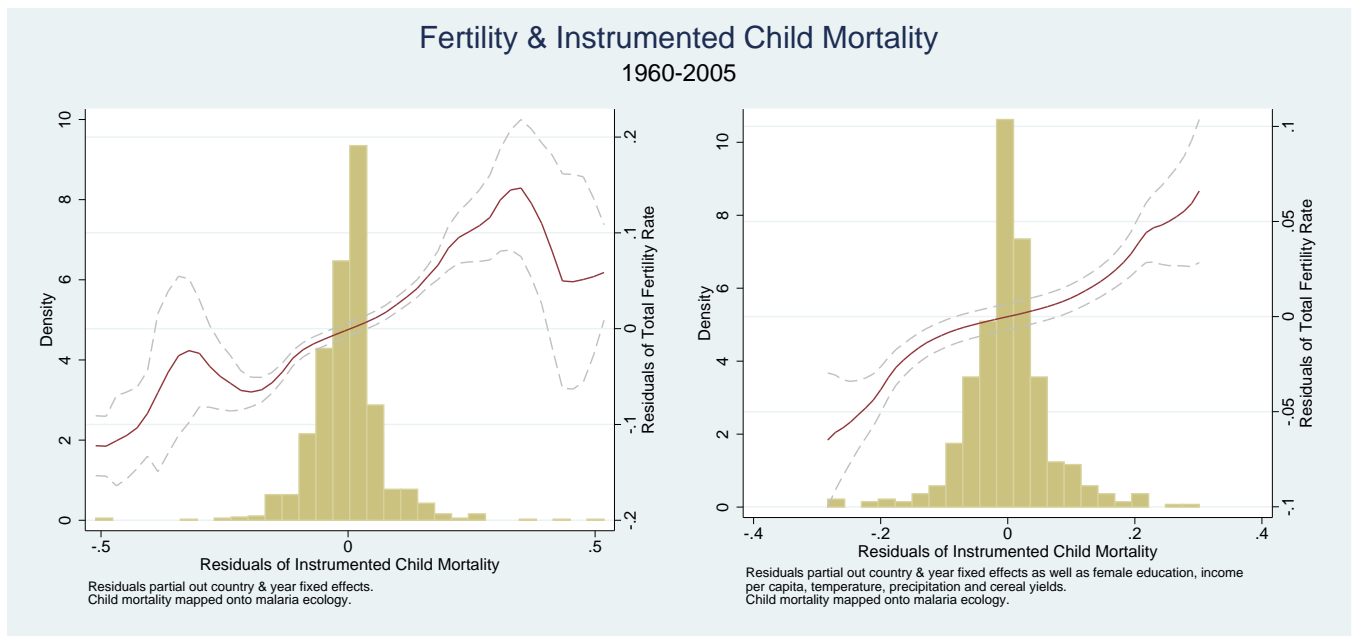**Figure 6**

Fertility & Instrumented Child Mortality
1960-2005

Appendix: Time-Varying Malaria Ecology Index

The time-varying malaria ecology index used in this paper was developed by McCord (2012) as an extension of the Malaria Ecology index developed by Kiszewski et al. (2004). Malaria Ecology is an ecologically-based spatial index of the stability of malaria transmission based on the interaction of climate with the dominant properties of anopheline vectors of malaria that determine vectorial capacity. Malaria is a disease of climate because a key part of the life cycle of the parasites (sporogony) depends on a high ambient temperature and because vectors require sufficient rainfall to provide breeding sites. Additionally, the intensity of malaria transmission depends on the specific mosquito species that are present and their relative attraction to humans versus animals. The Malaria Ecology variable published by Kiseweski et al. measures the effects of ambient temperature (using a monthly average from 1901-1990) on the force of transmission of malaria, as expressed through the length of the incubation period of the parasite in the mosquito's gut, and therefore the proportion of the vector population able to survive long enough to become infectious. The index is constructed on a 0.5 degree spatial grid to derive the climatic characteristics of individual months, and then averaged over a 12-month period. The first step is to identify the distribution of anopheline species across the world using observation records and satellite-based vegetation maps to identify likely habitats where observations have not been recorded.

A dominant species is identified for each spatial zone, and for each month (in cases where there is a seasonal pattern to the dominant species). An ecological screen was created for the presence or absence of a vector during particular months. (For those vectors that breed mainly in temporary water, a minimum precipitation threshold of 10mm per month, lagged one month, is used to judge when the vector would be present in the site during a given month. Vectors that mainly exploit permanent or semi-permanent bodies of water were considered to be independent of seasonal fluctuations in rainfall unless empiric evidence indicated otherwise. In temperate or altitudinous regions, temperature thresholds are used to determine whether parasites can develop in mosquito vectors in a particular month, assuming that malaria parasites cannot develop when the mean monthly temperature remains below 15°C). Note that the mosquito presence screen is ecology-based and not affected by human activity; indeed, it is worth keeping in mind that public health interventions against malaria serve to break the transmission cycle, but do not eliminate the presence of the vector itself (even until today, Anopheles mosquitoes capable of transmitting malaria can be found throughout the US and Europe, places where malaria has been largely eradicated).

The basic formula for Malaria Ecology combines climatic factors, the presence of different mosquito vector types and the human biting preference of the different mosquito vectors. The index expresses the factors that most powerfully and perennially influence the intensity of malaria transmission. It uses, therefore, a subset

of the vectorial capacity equation without terms for mosquito abundance, vector competence, or recovery rate for infected people. To calculate the duration of the extrinsic incubation period "E," the index was calculated for each month, and biting activity was designated based on the average monthly temperature and Moshkovsky's degree-day-based formulae:

$$MalariaEcology_{i,year} = \left(\sum \frac{a_{i,m}^2 * p_{i,m}^E}{-ln\left(p_{i,m}\right)} \right)_{year}$$

Where: m = month (1-12)

i = identity of dominant vector

a = proportion biting people (0-1)

p = daily survival rate (0-1)

E = length of extrinsic incubation period (in days) = $\frac{111}{(temperature-16)}$

Because it is built upon climatological and vector characteristics on a cell-by-cell basis, Malaria Ecology is exogenous to public health interventions and economic conditions. The Malaria Ecology index correlates strongly to malaria incidence, especially in the absence of public health interventions. Even a cursory comparison of the map of the geographic extent of malaria risk and Malaria Ecology show clear similarities: the high latitudes of the northern hemisphere are both where malaria was eliminated first, and also the malaria ecology index is lowest without being zero.

McCord (2012) re-calculates the index month-by-month using the same methodology using monthly data for temperature and precipitation. The index is aggregated to the country level without weighting by population to prevent potential endogeneity if humans can migrate internally to adapt to malaria prevalence.